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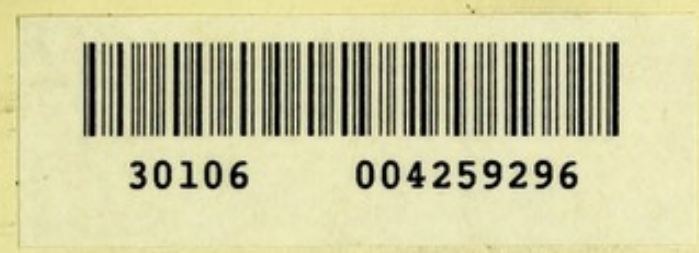
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LECTURES

ON

DISEASES OF CHILDREN

A HANDBOOK

FOR

PHYSICIANS AND STUDENTS

LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

BY

DR. EDWARD HENOCH,

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London:

SAMPSON LOW, MARSTON, SEARLE, AND RIVINGTON,

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PREFACE.

This work contains almost exclusively the personal experience which I have had the opportunity of collecting during a practice of thirty-seven years and an almost uninterrupted dispensary service in the field of diseases of children. The supervision of the Children's Wards in the Royal Charité, which was transferred to me in 1872, placed me in a position not only to raise the already very large number of my observations during all periods of childhood to an unusual height, but also to give them the positive anatomical basis which dispensary and private practice can never secure in themselves. It is with a reliance upon such enormous, carefully observed material, embracing all classes of a metropolitan population, that I could alone dare to assume the title of "Handbook for Physicians and Students" for this work, which is almost entirely the product of personal experience.

As a matter of course, the observations of any single physician will nevertheless remain imperfect, and the older and more experienced he becomes, the more he will be confronted by facts which are partly opposed to those observed at an earlier period; and for this very reason it should not be expected to find a description, or even mention, in this book of all the morbid processes occurring in childhood. Moreover, I do not consider it proper, in a work on diseases of children, to burden it with tiresome repetitions of matters which are treated of in detail in all works on general and special pathology and surgery, and a knowledge of which I may assume in my clinical audience, and even more so in my readers. The subject of this work is formed by those diseases of childhood alone which are distinguished from similar affections of adult life by a preponderating frequency, or by peculiarities in their symptomatology, and for that reason variola, which has become almost exceptional in children at the present time, has been excluded. I can only excuse my silence concerning vaccination for

the reason that I have nothing of import to add, from personal experience, to the innumerable treatises on the subject.

I need lose no time concerning my choice of the form of lectures, which has long been in vogue. Without disregarding its defects, I consider the advantages of this method, its informal character and more pleasant reading, as even greater than its disadvantages. To this may be added the fact that it considerably facilitates the introduction of reports of cases which serve, at the same time, as illustrations. I hope that the numerous reports of cases which I present to the reader will be of service to him, and I therefore beg that they be not overlooked. I have always endeavored to report the cases as briefly as possible, to bring into prominence the chief factors in question, and to avoid the intolerable length and tediousness of "accurate" clinical histories.

That I have made my own experience the basis not alone of the clinical descriptions, but also of the therapeutical recommendations, will be approved by every physician who has learned, to his disadvantage, the uncritical and confusing agglomeration of numerous remedies and methods noticed in the majority of compendiums. The prescriptions which have been added to the book, and are indicated in the text by the letters P. 1, 2, etc., I do not consider as an offence against science. Older physicians can dispense with them; to the younger ones, to whose wishes I have attached chief consideration, they will furnish welcome hints in beginning children's practice.

THE AUTHOR.

BERLIN, January, 1881.

LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

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LEEDS & WEST RIDING
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LECTURES ON

DISEASES OF CHILDREN.

INTRODUCTION AND METHODS OF EXAMINATION.

GENTLEMEN: Diseases of children are usually regarded as a specialty, but I consider this a mistake, as almost all diseases of childhood are found in adult life. These diseases have been converted into a specialty for the following reasons:

First.—A large proportion of the affections under consideration are observed much more frequently and more strikingly in childhood than in later life (acute exanthemata, pertussis, meningitis tuberculosa, etc.).

Second.—The medical examination of a sick child requires skill, which can only be obtained by frequent practice upon children.

In addition, the young physician, whose sphere of action is at first confined almost entirely to the lower, prolific classes of society, must, in great part, treat sick children upon entering into practice. This circumstance, which was formerly not sufficiently considered, is now more and more recognized, as I am led to judge from the continually increasing number of my hearers. It is so much more remarkable that the Faculties themselves obstinately oppose this view, and deny the right of pædiatrics to a separate chair.

But I cannot deny that even the most persistent study and ripe experience will not save you from disappointment in reference to therapeutic results. The conditions of life in early childhood are of such a nature that even the most rational and faithful treatment of its diseases is useless in an extremely large number of cases.

Statistics prove unmistakably that the mortality of children is greatest in the first months of life, while that of the first year more than twice exceeds that of later years; it gradually diminishes after the second year, and assumes ordinary proportions after the fifth year.

This is best shown by the following table: In my service in the Charité, 3,804 children were received from 1874–78, of whom 2,227 were under and 1,577 over two years of age. Of the former, 1,526—*i.e.*, about 70 per cent.—died, and only 277, or about 19 per cent., of the latter. Of the 1,384 children who had not passed the sixth month, 1,117 died, or about 80 per cent.

This enormous mortality, especially in the first six months, is explained by two sets of causes, the first of which is found in the natural development of the child, the second in extrinsic circumstances. After the birth of the child the development of the body does not cease, but very important changes occur in the organism. I will merely recall to you the closure of certain fœtal channels of the circulation, the differentiation of white and gray nervous tissue, the development of the intestinal glandular system, dentition, the growth of bones—processes which are calculated to cause pathological changes in the corresponding organs. But while children of the upper classes, who are carefully nourished, more readily pass through these developmental changes, the latter are apt to become pathological under the adverse influences to which the lower classes are subject. The foul air of narrow, crowded rooms, the more or less improper food, the influence of cold, hunger, the lack of maternal care—all these influences combine to impede the normal course of development, and to produce the harrowing pictures of disease with which we come in contact. Many of these unfortunate beings carry with them the seeds of death, and die of exhaustion within the first few days after birth ; others succumb to hereditary syphilis ; the majority become puny in consequence of persistent diarrhœa, or repeated attacks of bronchitis with enlargement of the bronchial glands, which finally lead to cheesy degeneration and general tuberculosis. A large proportion of these children are illegitimate, and many mothers send their offspring to the hospital in the hope that they will thus be relieved from them. Of the cases of this category which entered my wards, a large proportion died on the day of admission.

We will now concern ourselves with the medical examination, which, at least during the first few years of childhood, differs essentially from that of adults. One source of difficulty is the absence of speech, or the inability on the part of the children to furnish the physician with a history. In private practice this may be done by the mother, while in hospital practice we are usually restricted to a purely objective examination. The difficulty is increased by the fright and opposition of the child to the strange physician. While it is best in adults to examine one system of organs after another, and then conclude with the previous history, this principle must not infrequently be sacrificed in childhood, as the struggles of the little patient may force you to utilize every favorable moment for the inspection or auscultation of those parts which can alone be examined during quiet intervals—as, for instance, the throat or heart. In this manner the examination may become disjointed and irregular. On the other hand, a *résumé* of the symptoms is more readily obtained on account of the briefer and simpler clinical history. No definite rules can be laid down with regard to the conduct of the physician toward the patient. However sympathetic the physician may be, he will often be disturbed in his examination by the struggles of the patient, and the resistance must be overcome by kindness or force, according to the disposition of the child. Many children can be kept tolerably quiet during the examination by attracting their attention, as by showing them a watch, a toy, a lighted candle, etc.; and in especially important cases we can resort to chloroform, as in examination of the abdomen, bladder, or rectum.

During the first few years of childhood the patients are best examined by having them seated, opposite to the physician, on the lap of the mother or nurse, the face being turned toward the window. If at all justifiable, I even have patients who are suffering from fever, taken

out of bed and placed in this position. Not infrequently, however, the child resists the hands which endeavor to hold it, thus producing the greatest obstacles to percussion and auscultation. When the children are very restless, we can alone perform auscultation with the unaided ear, as the movements of the patient can thus be readily followed, if the physician, grasping the thorax of the child, keeps his head constantly in contact with it. I must insist that the examination not only of the posterior, but also of the anterior and lateral surfaces of the chest, should not be neglected in any case. I have not infrequently found signs of pneumonia below the clavicle, although everything was normal posteriorly, and the tongue-shaped prolongation of the left lung has often presented signs of pneumonia, which I heard much more indistinctly or not at all in other parts of the thorax. The anterior surface of the thorax may be examined in the sitting or recumbent position, the latter method being used especially in very young children ; the posterior surface should be examined only in the sitting or lateral position, but never while the child is on its belly, as the compression of the abdomen will press the diaphragm upward and diminish the thoracic dimensions, and may thus not alone increase the dyspnœa due to thoracic disease, but has also been known to cause sudden death during physical exploration.

Percussion causes much more annoyance to many children than auscultation, and crying interferes very markedly with the clearness of the results. In addition, every unsymmetrical position of the body, every muscular action during the movements of the thorax, causes a slight difference in the percussion-note, and you can therefore understand how careful we must be in the percussion of restless children. In an extremely large number of cases, I have thought, after a first examination, that there was a difference in the percussion-note over both halves of the thorax, although further repeated examinations proved my mistake. In doubtful cases auscultation affords the best control. Moreover, you should never fail to perform percussion both during inspiration and expiration, especially in crying children, as the percussed parts become more or less emptied of air during the cry, and therefore give a dull sound, which disappears during inspiration. This is especially so, as Vogel remarks, over the lower part of the right side posteriorly, where the liver, which has been forced upward during the crying and struggling, may give rise to dulness. Our patience may be sorely tried when we must wait for the rare inspirations for purposes of percussion. But auscultation is not interfered with, by any means, to so marked an extent. On the contrary, I find that, in the deep inspirations which occur during the intervals of crying, the pulmonary sounds are heard much more distinctly than during repose. I do not, therefore, try very long to quiet a crying child prior to auscultation, but insist upon absolute quiet on the part of those in the vicinity.

With regard to percussion, I would also advise the lightest possible strokes of the pleximeter. The conditions of the child's thorax with regard to resonance are so favorable on account of the elasticity of its walls, that strong percussion will cause a sonorous sound from vibration of surrounding parts, though the parts percussed may be empty of air, and therefore dull on gentle percussion. I make use of a small ivory pleximeter and an ordinary hammer. In great emaciation and in percussion of the supraclavicular region, the pleximeter must be superseded by a finger of the left hand.

In order to determine the frequency of the respirations, the child should be examined when quiet, and, when possible, during sleep. If the

hand is carefully placed upon the thorax or abdomen, the number of the respiratory movements can be readily determined. When the child is awake, even if it is not crying, this examination is often disturbed by the fact that the patient holds his breath. It is, therefore, not easy to give the normal frequency of respirations for any given age in childhood. It is greater in children under seven or eight years than in adults, and the frequency is greater, the younger the child, corresponding to the rapidity of the pulse. The heart's action is in itself more rapid than that of adults, very irritable under psychical impressions, and dread of the physician often increases its frequency to such an extent as to render it valueless for diagnosis. This is best shown in children suffering from icterus. The retardation of the pulse, so characteristic of this disease in the adult, I have never observed before the seventh year, and can only account for this fact by the above-mentioned irritability of the cardiac nerves. The pulse, especially in small children, can, therefore, alone be properly counted during sleep. But it must be remembered that, even in perfectly healthy children, the pulse is sometimes somewhat irregular during sleep.

On the average, I believe that a frequency, during the first few months, of 120–140, and, during the second year, of 100–120, should be regarded as normal. After this period a gradual reduction occurs. In children of three to six years the pulse is always above 90, and it is only after the second dentition that it approaches the conditions of adult life. The frequency of the respirations changes in a corresponding manner, its relation to that of the pulse being as one to three and one-half or four. But I repeat that very little is gained in practice by these averages. It is only under very definite circumstances that the rapidity of the pulse assumes a diagnostic or prognostic importance, as, for instance, its retardation in the beginning of tubercular meningitis and its remarkable acceleration at the close, and its extreme rapidity in scarlatina. On the whole, the rhythm and quality of the pulse have always appeared to me to possess greater importance to the physician. This is also true of the relation of the pulse to the respirations, which is, under normal conditions, as three and one-half or four to one. If this is permanently disturbed—if there are 40 to 60 respirations, with a pulse of 120 to 140—you may almost certainly assume that there is some disease of the respiratory organs. Only in exceptional instances is this due to nervous influences. The certainty of the diagnosis is increased if the respirations are not alone more rapid and superficial, but, at the same time, more labored, when certain accessory muscles are brought into play and expiration is stertorous. Bronchitis, pneumonia, or pleurisy is almost always present under these circumstances.

The examination of the heart in early childhood is attended with, perhaps, even greater difficulties. The rapid succession of the beats and the frequently accompanying cries not infrequently render it impossible, at the first examination, to form a decisive opinion concerning the clearness of the sounds and the conditions of percussion. The laryngoscopical examination of the larynx is, however, the most difficult. While there is scarcely any question of such an examination in very young children, even in older ones the physician, as a rule, meets with opposition which can with difficulty be overcome. Even if the laryngoscopic mirror is properly introduced, its surface is soon obscured by moisture, so that a distinct image cannot be obtained. In the large majority of cases, therefore, no results, or very uncertain ones, are obtained by this method. Still more unsatisfactory are the conclusions drawn by the older writers

from the character of the cry. I only attribute any practical significance to hoarseness or the substitution of painful moaning for the voice. You are probably aware that the new-born never shed tears in crying. The secretion of the lachrymal glands must, therefore, be as defective at this time as that of the salivary glands. After some practice the examination of the mouth and pharynx rarely presents any difficulties. If the child, when asked, does not voluntarily open the mouth, it is best to push the lower lip, with the forefinger, over the border of the lower jaw, and exert pressure upon it. The opposition which is made can usually, after a little persistency, be rapidly overcome, especially if the child is forced, by closure of the nostrils, to breathe through the mouth. As soon as the finger is pushed beyond the lower row of teeth, the child usually opens the mouth sufficiently to permit examination of the mouth and pharynx. In the contrary event, resort may be had to a spatula. Above all, good illumination must be secured, either by the bright daylight, or, if this is not possible, by means of a candle, the flame of which is placed in front of a silver spoon held by the same hand. With this simple mirror, which can be everywhere readily obtained, we can secure excellent illumination, to which I very frequently resort. But occasionally we meet with children who resist all our efforts, so that we must finally desist or resort to forcible means to separate the jaws.

In order to derive any benefit from the results of the examination, you must make yourselves acquainted with those factors by which certain appearances in childhood, which are found under normal conditions, are differentiated from those present in the adult, so that you do not consider normal conditions as pathological. I will first draw your attention to the differences in the character of normal vesicular breathing in various periods of life. During the first few weeks and months after birth, the breathing is still very feeble, as the short, superficial respirations are insufficient to force the air vigorously into the alveoli, and, for the same reason, the resonance on percussion is less sonorous. But from the middle of the first year the respiratory murmur begins to assume those characteristics which are also found in the adult under certain conditions, and are known as puerile breathing. It has a markedly sharp, almost blowing character, inspiration is almost alone audible, and expiration very slightly or not at all during repose, while it is more distinctly audible during excitement.[1] The rough, puerile breathing is also increased in those cases in which the thorax is abnormally narrowed by rachitic deformity, and we can therefore imagine that in healthy children also the relative narrowness of the thorax produces the rough blowing character by slight compression of the lungs which are being dilated by inspiration.[2]

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[1] I will also mention that, even in healthy children, fright may temporarily lead to that rhythm which is significant of the respiratory diseases of childhood, viz., the predominance of a prolonged, stertorous expiration over the short inspiration which follows the former like an echo.

[2] The explanation of Sabatier (Etude sur l'Auscultation du Poumon chez les Enfants, Paris, 1863) is ingenious, but not by any means conclusive. According to the measurements of this author, the capacity of the bronchial tree in man and the mammalia increases from the centre to the periphery, but diminishes in children, *i.e.*, the lumen of the two branches of a bronchus combined is narrower than that of the primary bronchus. For this reason the velocity of the current of air in the bronchi diminishes toward the periphery, but increases in children, and the respiratory murmur in the latter therefore appears rougher; this is aided by the greater sharpness of the cartilage, situated between two separating bronchial tubes, and which increases the vibrations of the passing column of air.

Abnormal pulmonary and pleural murmurs are, on the whole, not different from those in the adult, but moderate and fine rhonchi are much more frequent, not infrequently with the peculiarity that they predominate in expiration, while inspiration may be almost entirely free. The abdominal type of respiration predominates until the third year. The diaphragm and abdominal muscles contract with exceptional force, so that, even in the healthy condition, the slight retraction of the epigastrium and the lower ribs foreshadows the pathological change, which is so marked in severe respiratory diseases. Irregularity of breathing in the waking state, and even slight intermissions, should not cause any anxiety, as this occurs not infrequently in small children. The relatively narrow chest contrasts with the large abdomen, which mothers so often consider abnormal, though it is merely produced by the depression of the diaphragm and a tendency to the formation of gas in the intestines.

Among the appearances found in examination of the head, an auscultatory phenomenon deserves mention. In quiet children whose large fontanelle is still open (until the age of two years), the ear or stethoscope, when applied to this part, recognizes, although not constantly, a more or less loud blowing murmur, which is synchronous with the systole of the heart. As the respiratory murmur is also heard at the fontanelle on account of the vibration of the column of air passing through the pharynx, as is also every sound caused by moaning, chewing, and swallowing, the hand should be placed on the pulse during auscultation, in order to prevent mistakes. With some practice, however, we are soon able to differentiate the murmur even without this precaution. In only one case I heard the murmur over the closed fontanelle and other parts of the skull, and others have observed it over the lateral and posterior fontanelles, in the situation of the middle meningeal artery, and even on the spinous processes of the cervical vertebræ. While Fisher (1833) and Whitney (1843), the discoverers of this murmur, attributed a pathological significance to it, and I was also inclined to connect it with rachitis, Hennig and Wirthgen emphasized its normal occurrence from the twenty-second or twenty-third week to the period of osseous closure of the fontanelles. I now agree with the views of these writers, and believe that the murmur is heard so much more frequently in rachitic subjects because in them the fontanelles remain open so much longer. The causes of this murmur are still undetermined. Jurascz'[1] view that it was due to relative narrowness of the sulcus caroticus in the carotids, was opposed by Epstein, who attempted to connect it with a murmur heard in the carotids in the neck. However, the cerebral blowing murmur possesses no clinical interest and is of no diagnostic value.

The relations of the fontanelles and sutures are much more important. In the new-born child the latter are found closed by a cartilaginous ridge, but the former are still membranous, so that the cerebral pulsations can be felt over the anterior fontanelle, and most distinctly when the brain is congested and presses the fontanelle above the level of the surrounding bones. The tense, elastic, pulsating condition of the anterior fontanelle is, therefore, a valuable sign of cerebral congestion, while its depression indicates an anæmic condition of the brain, which is so frequently found in marasmic children or at the termination of exhausting diseases (diarrhœa, cholera infantum). While the lateral and posterior fontanelles are ossified during the first few months after birth, the anterior one

[1] Beiträge zur Kinderheilk., p. 170. Berlin, 1861.

even increases in size during the first six months, becomes gradually smaller during the second half of the first year, and is completely closed about the fifteenth month. But not infrequently the fontanelle shows a slight membranous structure far into the second year, though this cannot always be regarded as a pathological appearance. But other deviations, especially if the great and small fontanelle remain open to a greater extent and for a much longer period, if the sutures are separated and the edges of the bones are unusually movable, must be regarded as pathological. This is also true of some anomalies in the shape of the head, connected with certain diseases (rachitis, hydrocephalus), while individual differences, due to anomalies in the growth of the bones (asymmetry, dolichocephalus, etc.), barely possess any clinical interest. As a main distinction from the adult, you must remember that in children, until the age of two years, the size of the skull is much greater than that of the face, so that their relation is as 6 to 1 (even 8 to 1 in the new-born), and in the adult as 2½ to 1. Many timid mothers consider their children hydrocephalous, especially when there is rachitic thickening of the bones of the skull. Under these circumstances many children only become able to hold the head erect without aid at a very late period, although this is often possible, in the healthy condition, between the third and fifth months. But there are many exceptions to this, chiefly induced by the greater or less amount of muscular strength, especially of the muscles of the neck. Even if the child does not hold the head erect in the fifth or six month, we are not justified in diagnosing a cerebral disease, unless other symptoms are present, such as deficient intellectual development, fixed gaze, nystagmus, awkward movements of the hands, or complete apathy.

In the examination of the buccal cavity of the new-born, you will be at once struck by the dark red color of the mucous membrane, which slowly disappears after a few weeks, and must be regarded as normal. This hyperæmia is combined with a certain degree of dryness, as the secretion of saliva is still deficient. Recent investigations (Ritter, Korowin, Schiffer, and Zweifel) have shown that saliva is present from birth, but in such small quantities that it possesses very little or no glycogenic power. Toward the end of the second month the salivary secretion increases perceptibly ; according to Zweifel, it only begins at this time in the submaxillary glands and pancreas, while the parotid contains ptyalin at birth. On account of the deficient secretion, also, the buccal mucous membrane, during the first few months of life, is almost always somewhat acid in its reaction, and only after being carefully washed does it become neutral, or, very rarely, alkaline.

In very many new-born infants the raphe of the hard palate presents whitish yellow nodules, projecting slightly above the mucous membrane, from the size of a millet-seed to that of a pin's head; they are either separate, or there are several behind one another, which are sometimes surrounded by a narrow red zone. These nodules, which are sometimes somewhat elongated, are frequently found during the first four to six weeks of life, and possess no pathological significance. Bohn regarded them as obstructed mucous follicles, Guyon and Thierry as epidermoid cysts, and Moldenhauer[1] as solid proliferations of the epithelium, and as newly forming glandular coils. But the researches of Epstein[2] have re-

[1] Archiv für Gynäkol. Bd. VII., Heft 2.

[2] Ueber die Epithelperlen in der Mundhöhle u. s. w. Zeitschr. für Heilkunde. 1 Bd. Prag. 1880.

cently shown that they are spaces filled with epithelium, which have been left over after the union of both halves of the palate.

With regard to the tongue, you should bear in mind that it is usually covered, in nursing infants, with a thin, whitish coating, especially after nursing, and that it presents, in many older children, a peculiar appearance, like a chart, *i.e.*, the dorsum shows numerous, curved or straight, whitish gray figures. This appearance, which is perhaps due to irregular desquamation of the epithelium, is often found in perfectly healthy children.

Examination of the heart furnishes results which are almost identical with those found in the adult. For practical purposes, it is sufficient to know that the movements of the normal heart are often perceptible in two or three intercostal spaces, and that the ribs are raised more than in later life; the apex-beat can often be felt a little outside of the line of the nipple, although there is no hypertrophy of the organ.

The employment of the thermometer cannot be praised too highly, but it can alone be thoroughly carried out in hospital and private practice, while it is not feasible in dispensary practice. Under such circumstances we must be satisfied, except in important cases, with estimating the temperature by means of the hand, and relying for information as to the further course of the fever upon the statements of the mother. I always take the thermometric measurements in the axilla. Although this usually lasts from ten to fifteen minutes (twice as long as in the rectum), I cannot persuade myself to choose the latter site for examination, since, despite all precautions, a sudden movement may break the thermometer in the rectum, as I have witnessed. The conditions of temperature are similar in children and adults, except that there is a decided tendency to coolness during the first three or four months. The production of heat appears to be less active at this age, and in many cases of defective nutrition, exhausting discharges, or insufficient action of the lungs, the temperature gradually falls, and may even sink to 30° C. or less. This peculiarity is also shown in the fact that acute febrile diseases often run their course at this age with normal or even subnormal temperatures. But it is unnecessary to create a new disease under the title "Algor progressivus," as was done by Hervieux, since this condition may develop under the most different circumstances, provided they finally terminate in exhaustion.

The examination of the urine is very difficult in the new-born and nurslings, as it is always passed into the diapers, and an estimation of its quantity and color is very uncertain. But in some cases it becomes necessary to examine for albumen or sugar, and the urine must therefore be collected in special vessels (in girls, in well-cleaned sponges placed in front of the genitals; in boys, in condoms or rubber bags fastened around the penis), or by the introduction of an elastic catheter, the latter method being preferred in my clinic. The practitioner is usually satisfied with an estimation of the quantity of urine from the condition of the diaper. Its moisture furnishes him with a criterion of the quantity of nourishment taken, and from the diminished amount of secretion, he usually concludes with justice that there has been an insufficient supply of nourishment or deficient absorption. Only in very recent times has the urine of the new-born been carefully examined by Parrot and Robin,[1] Martin and Ruge,[2] Cruse,[3] Camerer,[4] and others, but the results

[1] Comptes rendus. Bd. 82. No. 1.
[2] Ueber das Verhalten von Harn u. Nieren der Neugeborenen. Stuttgart. 1875.
[3] Jahrb. für Kinderkrankh. XI., p. 393. 1877.
[4] Ibid. XV., p. 161. 1880.

obtained are not entirely uniform. It is especially interesting to the physician that Martin and Ruge occasionally found a small quantity of albumen in the urine within the first ten days after birth, either temporarily or for a number of days, and they are inclined to connect it with the discharge of uric acid concretions from the uriniferous tubules. Cruse's examinations furnished similar results, while Parrot and Robin never found albuminuria in the healthy new-born. Cruse[1] never found albumen in children over ten days old, but a large quantity of mucin.

In the new-born and nurslings the fæces, also, can alone be examined when mixed with urine. In the normal condition they are almost odorless, have about the color and consistency of scrambled eggs, and occur three or four times daily. Deviations from this rule should not be considered as abnormal if the consistence is not more fluid or the smell is not fœtid. In many healthy children the color of the fæces is not like that of the yolk of egg, but has more of a brownish hue. If the diapers are kept for any length of time, the yellow color is often changed to green, because the oxygen of the air converts the brown biliary coloring matter into biliverdin, and the fæces should therefore always be examined as soon as possible. They are usually surrounded by a wet, colorless ring, due to the urine. But I desire to call your attention to the fact that there are certain cases of diarrhœa in which the fæces voided are quite normal in appearance, but which are followed by a more or less copious discharge of serous fluid from the rectum. The wetting of the diapers may then be regarded as due to urine, and the fæces be considered normal. A case of this kind recently came to my notice in a child four months old, which was in a condition of increasing collapse, and in whom the diapers presented the above-mentioned appearance. I convinced myself from personal observation that the evacuation of the firmer fæces was always followed by a violent discharge of a large quantity of thin, opaque fluid from the anus.

Finally, I must consider the manifestations of pain in little children, which can naturally only consist of cries. It is no easy matter to distinguish such cries from those which are the expression of hunger or some other undefinable discomfort. I consider it useless to give you a description of the various modifications of crying, as so many authors have done. Vigorous, continued crying, which does not excite a coughing spell, is a favorable sign in diseases of the respiratory organs, because it indicates a comparatively slight degree of irritation of the respiratory mucous membrane. Loud crying spells, with vigorous movements of the lower limbs, especially flexion on the abdomen, usually, in nurslings, indicate colic pains. It is, however, often very difficult to determine whether the child's cry really indicates suffering. In doubtful cases, when pressure upon the apparently affected parts or any other part of the body causes the cry, or increases it, we can only satisfy ourselves if we succeed in quieting the child, and then begin the examination anew. If we can divert the child's attention from the spot which is being examined, we can often determine the part which is really sensitive to pressure. In determining the condition of the new-born and nurslings, I would recommend you to watch the position of the hands during sleep. Healthy children of such an age sleep with the arms flexed in such a manner that the hands are directed upward, and are held as

[1] Jahrb. für Kinderkrankheiten. XIII., p. 71. 1878.

high as the neck or lower jaw. This position, which is perhaps a relic of intra-uterine existence, changes during a serious illness, and can therefore be regarded as a quieting symptom. I take this opportunity to mention that healthy children usually sleep with firmly closed lids, but that in not a few a slight separation of the lids is noticeable. In individual cases we must make inquiries with regard to this circumstance, which, as we shall see later, more frequently has a pathological significance.

LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

PART I.

DISEASES OF THE NEW-BORN.

The period of nursing continues until the ninth to the eleventh month, when the development of the teeth indicates its close. During the first four to six weeks the child is called new-born, and this period includes a series of diseased conditions which do not occur at a later age, or much more rarely and under a different form.

You are aware that all new-born infants, during the first few days after birth, present a more or less intense red color of the entire skin, due to hyperæmia. In many children this color gradually fades and disappears in about a week ; in others the red color first gives place to a more or less bright yellow hue, which is known as

Icterus Neonatorum

(jaundice of the new-born). This yellow color is usually observed on the second or third day after birth, but is generally not distributed uniformly, being more marked on the forehead, around the mouth, and on the trunk than on the extremities. The more the above-mentioned redness disappears the more distinct and general does the jaundice become; most commonly it has somewhat of an orange tint, and is not very intense. It usually lasts several days, then gradually diminishes, and disappears in eight to fourteen days.

There are marked differences in the symptoms of this affection from those of jaundice in later life. The diapers are wet with colorless urine, the fæces are yellow or brownish, as in the normal condition; the sclerotic coats of the eyes have a yellow tinge in many cases, though not in all, and the temporary pallor produced at the point of pressure by the finger upon the gums does not always show the yellow hue which we are accustomed to find in the icterus of adults. Apart from the yellow color of the skin, there are no other symptoms. On account of its innocuousness and great frequency, icterus neonatorum has been regarded as an almost physiological condition.

It is above all necessary to determine whether the yellow color is really due to a bile-pigment which has formed in the liver. Opposing views are still held on this subject. The old view of the French writers that the jaundice was the result of a yellow pigmentation caused by the red color of the new-born, finds no adherents at the present time. Not

alone the skin, but also the internal organs are yellow, and Orth [1] describes a case in which even the brain had a deep yellow color. Orth's investigations lend new support to the view that the coloration of the tissues is due to a pigment which is very like that found in the bile. He corroborated the previous observations of crystalline pigment in the blood and various organs of the new-born, and found that it was only present in cases of general icterus or those in which the jaundice was disappearing.[2] The pigment, which was present in the blood, kidneys, liver, and other organs, in the shape of red, rhomboid plates or needles collected into plumes, showed the microchemical characteristics of bilirubin, and Orth believes that they have formed after death from the biliary pigments dissolved in the blood. But how does this pigment enter the blood? While some regard the jaundice as hæmatogenous and caused by the formation of yellow pigment in the blood, others regard it as of hepatogenous origin, as in ordinary obstructive jaundice. Although I have often found small mucous plugs in the ductus choledochus, the biliary contents of the intestines and the normal color of the urine, showed that these plugs were insufficient to cause any considerable retention of bile and absorption in the liver. On the other hand, the ductus choledochus and hepaticus are often found entirely free of mucus, and on this account the view that icterus neonatorum is of hæmatogenous origin is continually gaining fresh adherents. We are, however, ignorant of the factor which gives rise to such an abundant production of yellow pigment in the blood. This must necessitate the destruction of a large number of red blood-globules, which set free a corresponding amount of blood-pigment, from which the bilirubin and hæmatoidin are derived. If the investigations of Porak [3] and others are confirmed, they would indicate the effect of a slow ligature of the funis, performed after the cessation of the pulsation, upon the production of the icterus. When this method is adopted a larger amount of blood is said to enter the circulation from the placenta than when the funis is rapidly tied, and, therefore, there will be a greater destruction of red blood-globules and more abundant formation of bilirubin and hæmatoidin in the blood, the elimination of which proves insufficient, especially in weak children. In this manner, then, the pigments accumulate in the organs and give rise to the yellow color. It is still unsettled, however, whether these views are correct. But the hæmatogenous doctrine has recently received a severe blow from the latest work of Cruse,[4] who showed by microchemical reactions that the yellow bodies found in the urine of icteric new-born were really bile-pigment, and that dissolved bile-pigment could be shown in the urine upon shaking it with chloroform. Cruse, therefore, regards the jaundice as hepatogenous, due to hyperæmia of the liver and desquamation of epithelium in the finer bile-ducts, these influences being not active enough, however, to separate the bile entirely from the intestine. The author has not hitherto furnished any anatomical proof of this view.

The development of the jaundice is aided by weakness of the infant, the action of cold, atelektasis, and bad air. Treatment is unnecessary, as the disease recovers spontaneously.

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[1] Virchow's Archiv, Bd. 63. Ueber das Vorkommen von Bilirubinkrystallen bei Neugeborenen Kindern.

[2] In 37 cases of icterus Orth found the pigment in 32, and in the remaining 5 the former presence of jaundice could not be absolutely denied.

[3] Porak: Considérations sur l'Ictère des Nouveau-Nés, Paris. 1878. Schücking, Berl. Klin. Wschr., 1879. No. 39. Violet: Virchow's Archiv, Bd. 80, p. 353.

[4] Archiv f. Kinderheilkunde. I., p. 353.

In not a small number of cases, however, the jaundice is complicated with other more serious diseased conditions. Many of these patients are extremely emaciated and feeble at birth, they suffer from sprue in the mouth and on the gums, and are affected with diarrhœa and vomiting. Under these conditions I have repeatedly observed a yellow color, *i.e.*, biliary matter, in the vomit. The most unfavorable complication is that of sclerema neonatorum, which is fortunately quite rare. The following case is noteworthy on account of the obscurity of the etiology and the unexpected favorable termination:

A child, two weeks old, suffered for ten days from jaundice, which had suddenly increased a few days previously. The fæces were dark brown and scanty; the spots of urine in the diapers had yellowish green borders. In addition, marked development of sprue, extending into the pharynx, livid color of the mucous membrane, increasing exhaustion despite excellent nursing. There were numerous red spots on the neck, back, and extremities; they did not disappear on pressure, were slightly prominent here and there, and disappeared later after slight desquamation. Under the use of an infusion of bark, with hydrochloric acid, mouth-washes of a solution of chlorate of potash, and aromatic baths, the infant unexpectedly recovered.

Whether the exanthem was due to small hemorrhages or to collections of bilirubin crystals in the skin, I am unable to determine.

Icterus neonatorum proper must be distinguished from that form of jaundice which, in rare cases, is due to obliteration or congenital absence of the excretory bile-ducts. During my entire practice, three cases of this kind have, at the most, come under my notice, and an autopsy was obtained in the following one:

A child, four months old, suffered since birth from jaundice, with dry evacuations of an almost milk-white color, and dark bile-stained urine. The left hepatic lobe could be felt in the epigastrium. Despite all remedies, the color of the skin became greener, and the child died, extremely emaciated, five weeks after it came under observation. At the autopsy, the liver was found at least a third smaller than normal; both lobes were of the same size, the left one flattened and extending to the left hypochondrium, of moderate consistence and olive-green color throughout. The gall-bladder was rudimentary, no trace of the biliary passages could be found, and the opening of the ductus choledochus into the duodenum was also absent.

Trismus or Tetanus Neonatorum.

Though the symptoms of this disease are similar to those of tetanus in the adult, they are more or less modified by the tender age of the patient. It begins most frequently between the fifth and ninth days after birth, although, in a few instances, I have seen the first symptoms develop on the twentieth day. As a rule, the earliest symptom is difficulty or impossibility of nursing; every attempt gives rise to rigid contraction of the muscles of mastication and the orbicularis oris, rendering suckling impossible. The other facial muscles also take part in the contraction, and the countenance then becomes distorted. In the beginning these symptoms only appear paroxysmally upon attempts at nursing, and it is sometimes possible to give the child milk by means of a teaspoon; in a few hours, however, the symptoms rapidly increase in severity. The paroxysms now occur spontaneously, the forehead being thrown into transverse folds, the eyebrows wrinkled, the lids firmly closed, the lips puckered like a snout and surrounded by radiating folds. The pharyngeal muscles soon become affected, and the ingestion of milk is thereby

hindered, this being often accompanied by spells of suffocation, with cyanotic face and cessation of the respiratory movements; the latter are usually extremely rapid and superficial in the intervals of the attacks. If we endeavor to insert the finger into the mouth, we come in contact with the jaws, firmly pressed together by the rigid contraction of the masseter and temporal muscles, and any attempt to overcome the resistance of the jaws is always followed by the development or increase of the spasmodic phenomena. In very few cases are these restricted to the above-mentioned parts; they are usually followed by rigidity of the muscles of the neck and back, with retraction of the head and stiffness of the vertebral column, which become especially evident if the child is grasped by one hand around the middle of the body and held in a horizontal position. The muscles of the upper and lower extremities are also more or less affected, the arms and legs are extended, the muscles hard and unyielding, and forcible flexion almost impossible. These symptoms show intermissions, or at least remissions, but as the disease progresses they become more persistent and frequent, though by no means constant, and are markedly intensified upon touching the patient, giving nourishment or introducing enemata. Rapid convulsive twitchings are also occasionally observed in the trunk and limbs. Under such circumstances it is impossible to nourish the child by the breast or bottle; in one case, however, the patient drank from the bottle at the height of the disease, though not in sufficient quantities.

The spasmodic phenomena, together with the absence of nutrition, lead to a rapidly increasing collapse. The temperature either remains normal, or shows moderate elevations to 38.5–39°, and in many cases this is not exceeded during the entire course of the disease. But occasionally the temperature rises quite rapidly, and finally reaches 40°, 41°, and even more. As a rule, the disease shows a uniformly progressive character, though occasionally an apparent improvement occurs either spontaneously or after the exhibition of remedies; usually this is soon followed by a fresh aggravation of the spasms. Finally the child becomes comatose, the pulse is imperceptible, and death occurs from exhaustion or from asphyxia, due to tetanic contraction of the inspiratory muscles. The duration of the disease varies from one to five days.

By far the largest proportion of cases of trismus neonatorum prove fatal; but complete recovery is not impossible, and a few such cases have occurred in my experience. Those accompanied by high temperature present an extremely unfavorable prognosis, though death is also very frequent when the temperature is low (37.1–37.8° throughout the attack). In favorable cases improvement always occurs gradually; the rigidity of the muscles and the spasms slowly disappear, and in two of my own cases I could detect some rigidity of the muscles of the limbs at the end of three weeks. But both cases were not very severe, even at their height; the temperature was elevated only a few tenths of a degree, and in one of the children milk could be administered after the second day by forcing a spoon between the jaws.

Post-mortem examination furnishes no characteristic results. In those cases in which extravasations of blood are found in the spinal canal they must be regarded as the results of venous stasis caused by the interference with respiration. Small hemorrhages, due to the same cause, are not infrequently found in the cerebral meninges and in other serous membranes. The central organs are normal, apart from more or less marked venous congestion and its sequences (œdema, small extravasa-

tions). We must therefore regard trismus and tetanus neonatorum as a neurosis, though this term expresses very little. It is undoubted that the reflex irritability of the spinal cord is increased in tetanus, though the increase of the spasmodic phenomena by irritation of sensory nerves (feeling the pulse, touching the child, etc.) is not equally well marked in all cases. In trismus neonatorum, also, this symptom is sometimes more, sometimes less developed, and this is understood so much more readily as the reflex impulse is predominant at this age even in the normal condition. If Soltmann's experiments on new-born animals are verified, it would appear that during the first period of life all movements are reflex and produced without volitional effort, and that all the reflex inhibitory centres in the brain and cord are still wanting. This would explain the enormous frequency of reflex spasms at this period, but not the factor which impresses upon this uncontrollable reflex action the peculiar and dangerous form of trismus. But Soltmann's view, that the irritability of the peripheral nerves is less in the first period of life than in adults, does not tally with the frequency of this form of disease, since no one will deny that the exciting cause of tetanus is to be found in these nerves. As in adults, so also in the new-born, tetanus is the result of various influences which irritate either the distribution of a single nerve or a number of sensory fibres, and which, on account of the predisposition which is present, gives rise to the disease by rapid transmission to the spinal cord. Among the causes I will mention:

First.—Injuries (tetanus traumaticus) which usually affect the umbilicus. In two of my own cases the remains of the cord had been forcibly torn off on the morning after birth, and the wound of the umbilicus was surrounded by an inflamed zone. I will add, however, that I attach importance only to real injuries, not to the "inflammation of the umbilical arteries" mentioned by Schöller, which is merely a partially broken-up thrombus, and has no connection with trismus.

Second.—The effects of thermal irritants on the skin, on the one hand, early exposure to cold air (for instance, on account of baptism), and, on the other hand, too hot baths. This occurred in Elbing, where, in the practice of a busy midwife, trismus was endemic for a number of years, and hundreds of children died in consequence. It was finally discovered that the midwife could not distinguish a temperature of 33° from that of 28°, and not until the thermometer was employed did the "epidemic" terminate.[1] It can be readily understood that many other irritants remain undiscovered, and the disease apparently develops spontaneously. Perhaps its development by foul air (the vapor of oil in Iceland, the Maternity Hospital in Dublin, from which trismus is said to have been driven by proper ventilation), and its epidemic occurrence in certain islands of the West Indies, can be explained by some of the causes mentioned. Attention must also be paid to the condition of the kidneys. The occurrence of albumen in the urine of the new-born was referred to above (p. 9), and it was stated that some observers (Martin and Ruge) are inclined to regard the discharge of uric acid concretions from the renal tubules as the cause of nephritic processes in the new-born. It may therefore also be taken into consideration whether uræmic processes may not appear at this time of life under the form of trismus. We may thus, for instance, compare the following case[2]:

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[1] Bohn: Jahrb. f. Kinderheilk. IX., p. 307. 1876.
[2] Centralzeitung f. Kinderheilk. Berlin, Jahrg. I., p. 57.

Beginning of the disease on the thirteeeth day after birth with severe trismus and general convulsions. Temperature 41.8° on the third day of the disease. Death on the sixteenth day. The urine drawn by the catheter on the third day contained a large quantity of albumen, with numerous hyaline and granular casts. The autopsy showed capillary hemorrhages in "the cortex of the kidneys and the glomeruli, and thrombi in the renal tubules. Umbilicus normal."

The view that trismus neonatorum, like epilepsy, is a spasmodic disease which is specific only in appearance, and can be produced by various irritants, is therefore not without foundation. It is difficult, however, to determine its causes, and this can be done only under favorable circumstances, as in injuries, wounds of the umbilicus and thermal irritants. But even then therapeutics must contend with the greatest difficulties, as the disease, when it occurs in adult life, which is much more capable of resistance, is one of the most dangerous known. Chloral hydrate is the only remedy under whose use (0.06 hourly) I saw two cases of trismus neonatorum recover. If the drug cannot be swallowed, 0.1 should be given hourly by enema. In other cases this remedy proved as useless as the administration of chloroform, which, at the most, produced temporary relief of the lock-jaw. I have obtained temporary relief from opium alone (tinct. thebaic., gtt. j. every two hours) while the narcosis lasted. Equally poor results were obtained by extr. calabar, of which I used 0.005 hypodermically three to four times daily (0.05 to 10.0 water); although others (Monti) had good results from this remedy. On account of the extremely unfavorable results of all treatment in this disease, so much more importance must be attached to careful prophylaxis.

Apart from trismus, other convulsive attacks, partial and general, occur in the new-born, and are identical with the eclamptic symptoms of older children. I merely mention this for the reason that, based on certain of Virchow's discoveries, the latter are made responsible for these cerebral symptoms. Under the title "Encephalitis and Myelitis Interstitialis," Virchow[1] described the diseased condition of the brain and spinal cord which he observed in the new-born or shortly after birth, and which occurred under the influence of infectious diseases (syphilis), and also without any distinct cause. It consisted essentially of a proliferation and fatty infiltration of the neuroglia-cells, which was sometimes visible macroscopically in the form of small yellow hortensia-colored soft spots. These observations, which were confirmed by Hayem and Parrot, but were not directly regarded as inflammatory, were interpreted by Jastrowitz,[2] in an article based upon sixty-five cases, as indicative of a physiological fatty degeneration of the neuroglia-cells, especially in certain parts of the mid-brain and the posterior columns of the medulla; this reaches its maximum at the seventh month of intra-uterine life, then diminishes and disappears soon after birth. Jastrowitz regards the fatty degeneration as abnormal only when it continues beyond the usual period, or involves other parts of the brain than the white substance of the centre, as, for instance, the large basal ganglia, the gray cortical substance, the nuclei of the cerebral and spinal nerves. We are ignorant of the etiological relations of this incomplete absorption of fat. Hitherto these observations possessed an anatomical interest alone, as no relation to definite clinical symptoms has been detected, nor has the keratitis ul-

[1] Archiv, 1867, Bd. 38, p. 129; 1868, Bd. 44, p. 472.
[2] Arch. f. Psych. u. Nerv., II. u. III. 1872.

cerosa [1] said to occur from the second to fifth month as the result of such an "encephalitis" been rendered certain.

This is also true of certain macroscopical changes—œdema, hyperæmia, small ecchymoses of the pia—which are occasionally found in the new-born. If the cases in which these appearances are observed post-mortem are compared clinically with one another, we do not find any characteristic symptoms, but oftentimes a general symptomatology, which may be termed that of "congenital deficiency of vitality." More or less marked emaciation, grayish yellow color of the skin, extreme weakness and apathy, pitiful whimpering instead of the normal cry, superficial, rapid respirations, slight cyanosis of the tips of the extremities, fall of temperature—these are the symptoms which the unfortunate beings present soon after birth, and to which most of them succumb during the first days or weeks of life, if they are not accidentally brought under especially favorable conditions. The lot of the majority, unfortunately, is to fall into bad hands or into a children's hospital. My wards in the Charité always contain a number of these children, almost all of whom, despite every effort, die in collapse from progressively increasing heart-failure, with or without convulsions. The œdema, hyperæmia, and small ecchymoses of the pia, which are not infrequently found under such conditions, should be regarded, in my opinion, as the results of venous stasis caused by weakness of the heart and the pulmonary atelectasis, which are almost always present, but not as active processes, and should therefore not be regarded as causes of the terminal convulsive symptoms.

Cephalhæmatoma.

Your aid will not infrequently be sought by anxious mothers with regard to a tumor on the head of the new-born, which is known as cephalhæmatoma, and is caused by an extravasation of blood between the bones and pericranium. It appears to be produced by pressure upon the skull during the passage of the fœtus through the pelvis. In many cases this pressure only involves the scalp and the subcutaneous and subaponeurotic connective tissue, giving rise to a sero-bloody extravasation, with the formation of a moderately large doughy tumor (caput succedaneum). If the pressure is more severe or prolonged the hemorrhage will occur between the pericranium and the bones, as a rule the parietal bones, especially the right one, which in the ordinary position of the child is most frequently subject to pressure during delivery. The extravasated blood gradually raises the pericranium from the bones, and forms a fluctuating tumor, which does not reach its maximum forthwith, but as the hemorrhage slowly continues, gradually enlarges and becomes stationary about the third day. The tumor not infrequently involves the entire parietal bone, but its further extension is prevented by the sutures to which the pericranium is very firmly adherent. A bilateral cephalhæmatoma has not hitherto come under my notice, though cases are mentioned in literature. Upon examination, you will find a more or less tense, distinctly fluctuating tumor, usually on the right, more rarely the left, parietal bone, or, perhaps, on other cranial bones. The skin is normal in color; more rarely has a bluish tinge, or is even infiltrated with hemorrhages.

[1] Graefe u. Hirschberg: Arch. f. Ophth., XII., S. 250, and Berl Klin. Wochr., S. 324. 1868.

When very tense, you will not be able to feel the subjacent bones, but can detect in the first few days a hard, somewhat projecting rim around the tumor, which, especially when the swelling is small, may simulate the edges of an opening in the skull. Cephalhæmatoma appears to cause very little annoyance to the new-born, but if pressure is made upon it, the little one will cry out, which is readily explained by the sensitiveness of the stretched soft parts. But the health remains good, and the absorption of the blood usually occurs with rapidity. This is aided by the fact that the blood remains at least partly fluid in these tumors for a very long time, sometimes over a month. At the end of a week the tumor is considerably smaller; the bones can be distinctly felt through it. Two to four weeks are required for complete absorption. During this time the previously mentioned hard ring around the tumor is still distinguishable, but becomes narrower, and in many cases in which absorption occupies a long time, you can, by pressure upon the soft parts, feel a creaking like that of parchment, until finally absorption is complete and the pericranium is again firmly adherent to the bones. The cause of this hard ring must be sought in the new formation of bone, which occurs upon the inner surface of the detached periosteum, and which is, at first, most abundant at the place where periosteum and bones are still in contact, *i.e.*, the edges of the tumor. At a later period plates of bone are found on the inner surface of the detached periosteum, giving rise to the parchment-like creaking referred to above.[1]

Cephalhæmatomata of a similar nature may occur at a later age from traumatic influences. I have observed them occur from traumatic causes —occasionally without any distinct cause—in children of two, four, and eight years of age. The tumor was situated upon the parietal or occipital bone, the latter being covered over its entire extent. Gradual enlargement of these tumors was also observed, and in a boy aged eight years, a hemorrhage occurred into the subcutaneous connective tissue of the forehead and eyelids, a week after the injury, and after the cephalhæmatoma had attained its maximum. A week later the subcutaneous hemorrhage was merely indicated by greenish yellow pigmentation, while the colossal cephalhæmatoma was entirely absorbed, with the exception of a small spot, about as large as a mark piece, surrounded by a hard, bony wall.

The treatment should be purely expectant. Formerly I often made incisions, emptied the blood, and then applied pressure by means of adhesive plaster. Suppuration could not, however, always be prevented in such cases. But although this danger is now very much diminished by the antiseptic method, I see no necessity for opening a tumor, which I have always found to disappear completely by absorption in a few weeks. I would, therefore, advise an incision only when the tumor threatens to suppurate and discharge, a termination which is extremely rare, and has never come under my notice. It is well, under all circumstances, to place a soft covering (cotton) over the tumor in order to protect it from injury.

Only the entirely inexperienced could mistake a cephalhæmatoma for congenital encephalocele, a hernia of the brain, or at least of its distended membranes (meningocele), inasmuch as the apparent or real fluctuation of such a tumor and the surrounding hard edge of the osseous opening might render the mistake possible. One difference is found in the fact that the cerebral hernia occurs generally at a site which is not implicated

[1] Virchow: Geschwülste, I., p. 130.

in cephalhæmatoma, viz., in the occipital bone—more rarely the glabella, or parietal bone. The size of the encephalocele is smaller, as a rule, and distinct pulsation can be felt in a number of cases. I will not detain you with a detailed description of this affection, but will merely narrate a case of unusually large meningo-encephalocele, which is very characteristic, both clinically and anatomically.

Ida B., eight days old; admitted to my wards December 23, 1873. The mother had previously had a child during the sixth month of pregnancy, in whom "the occipital bone was open." The skull is flat, the face frog-like, the diameters of the head very small. A tumor starts with a large pedicle from the middle of the occipital bone, its diameter is more than twice as large as that of the head, sways to and fro like a balloon, and is distinctly fluctuating, except over the pedicle, around which a bony wall is indistinctly felt. The skin of the tumor is only hairy over the part nearest the occiput, becomes bald and atrophic toward the vertex of the tumor, is thinned in places to an almost translucent lamella, and traversed by dilated veins and hemorrhages. No pulsation or respiratory movements in the tumor, which is not translucent. The fluid cannot be forced back in the least from the tumor into the skull. I therefore concluded that the communication with the skull was obstructed by brain-tissue, and that the tumor was not a meningocele, but a meningo-encephalocele. The marked flattening of the vault of the skull supported this view, as it indicated a deficiency in the cranial contents. The child was otherwise well formed; functions normal. Puncture of the tumor with a fine trocar discharge 200 c.ctms. of a reddish brown fluid, which contained a large amount of albumen and red blood globules; the tumor then became less tense, but very little smaller.

During the next two weeks its circumference again gradually increased, and frequent contractures occurred in the upper and lower limbs. Frequent twitchings were also observed in the muscles of the eyes and limbs. Pulse, 152 to 192. The child soon refused the bottle, and collapse occurred; temperature 36.9 to 36.5°. To facilitate the examination, I removed by puncture, on June 5th, 410 c.ctms. of bloody fluid, and could then distinctly feel a compact, doughy mass, which projected into the cyst from an opening in the occipital bone. Death occurred on January 6th, the Cheyne-Stokes breathing having developed during the day.

The autopsy showed that the cyst was composed of the extremely thin integument and of the immediately subjacent congested, and partly hemorrhagic dura mater. The sac contained brain-tissue, whose congested pia is adherent to the cyst-wall in numerous places. The sac also contains an abundant opaque red fluid. The squamous portion of the occipital bone presents a heart-shaped opening, 2½ ctms. broad and 3½ wide. The skull itself contains brain-tissue of normal appearance. The cerebellum and medulla are situated in the normal sites. The pia mater contains a few purulent deposits. Close examination shows that the largest part of the posterior cerebral lobes had passed through the osseous opening and lay in the sac. The posterior horns of the lateral ventricles situated within the sac were enormously dilated and filled with serous fluid, and distended the hyperæmic posterior lobes like cysts. Those portions of the lateral ventricles situated within the cranium contained puriform fluid. Otherwise the brain and remaining organs are entirely normal.

It is worthy of note that such a colossal meningo-encephalocele did not interfere with delivery to any extent. But the enormous pressure to which the tumor was subjected during labor was undoubtedly the cause of the hemorrhages into the integument and dura mater. The meningitis was probably the result of the double puncture.

Hæmatoma of the Sterno-Mastoid.

The act of delivery may also give rise to other anomalies which affect the neck. Children, during the first few weeks of life, will not infrequently be brought to you presenting a hard, round, or band-shaped nodular tumor on the lateral aspect of the neck, corresponding to the anterior part of the sterno-mastoid muscle. Its size varies, oftentimes as

large as a pigeon's egg, but frequently much larger and elongated, so that I have occasionally found a large part of the anterior edge of the muscle hard and nodular, whence band-shaped projections ran into the adjacent parts of the muscle. Occasionally there are two or three isolated hard spots in the edge of the muscle. As a rule, the upper half of the muscle is much more frequently affected than the lower, but in one case I found almost the entire anterior half of a cartilaginous hardness. The right sterno-cleido-mastoid is much more often affected, sixteen of my cases occurring on the right and only five on the left side.

The youngest child which I found suffering from this affection was three weeks old, the majority were four to six weeks old; three cases occurred at the ages of three, five, and twelve months. It produced no annoyance, and was usually discovered accidentally while the child was being washed. In rarer instances, the mothers found that the child's head was not held straight while lying down, but deviated to one side, usually toward the right. But this position was not constant, and appeared to me to be so much more infrequent the younger the child.

The nature of this tumor becomes evident upon discovering that almost all the affected children had an abnormal presentation, which either delayed labor or rendered artificial aid necessary. In twenty-one of my cases a breech-presentation occurred in fourteen, and delivery was performed by forcible traction. Of the remaining seven cases, five had a normal presentation, but the delivery lasted very long, and strong traction was necessary, because the shoulders of the child did not develop. There is very little doubt, therefore, that the cause is to be found in a partial rupture of the muscle during birth, and that the affection consists of a hemorrhage into the muscle (hæmatoma), and a myositis encapsulating it and leading to a fibrous cicatrix. The employment of force also caused other accidents, and in one of my cases gave rise to a fracture of the arm.

So far as my observation goes, the swelling always takes a favorable course, becomes gradually smaller, and finally leaves a more or less firm cicatrix in the muscle, which interferes very little, or not at all, with its functions. I have never observed suppuration, but it cannot be denied that a caput obstipum might result. The natural process of recovery by the formation of a cicatrix renders treatment unnecessary. At the most, an ointment of iodide of potassium may be rubbed over the tumor as a placebo, or in order to keep the case under observation.

In very many new-born you will observe during the first weeks of life an

Enlargement of the Mammary Gland,

which is interesting in several respects. In the situation of one or both mammæ you will find a round or blunted, conical, quite hard tumor, about the size of a small walnut and of the normal color of the skin. It appears to be painful on pressure, as this causes the child to cry. If the base of the tumor is grasped by two fingers, and lateral pressure exerted, you will see a whitish, opalescent drop exude from the little nipple situated at the summit. Under the microscope this shows fat-globules, with conglomerate masses of fat.

In order to understand the development of this tumor, it must be remembered that, in all new-born babes, a milk-like secretion from the breasts begins on about the fourth day after birth; increases until the

ninth day, usually accompanied by slight swelling of the mamma, and then gradually subsides, so that it has disappeared by the twentieth day. Natalis Guillot[1] removed by pressure about one cubic centimetre of whitish fluid, which showed, under the microscope, all the characteristics of colostrum. According to Sinéty's observations,[2] sections of the breast of the new-born show, near the skin, milk-canals, which are filled with epithelial masses. Farther down these become dilated, divide and form cavities, which contain a fluid like colostrum. This process is said to begin during fœtal life, reaches its acme from the fourth to tenth day after birth, on account of increased development of the milk-canals and cavities; may be increased by pressure upon the breasts, and continue six to eight weeks in individual cases. Epstein[3] connects this process with the active-cell proliferation and epithelial desquamation which occurs during fœtal life in other parts, that must be regarded as involutions of the skin, especially in the sebaceous glands, and appears in the form of smegma and seborrhœa. According to Guillot, the secretion of milk occurs only in weak children.

The secreting mamma may become the site of diseased processes in the new-born as in the adult woman. But it is unnecessary to assume, as Bouchut does, a puerperal state in the former. The local process may simply become intensified into inflammation, which first causes enlargement of the gland, and may then lead to the formation of abscesses. The little tumor then becomes reddened, is very sensitive, fluctuates, and discharges pus spontaneously or after incision. As I have observed this result several times from strong and repeated pressure, I now abstain from manipulation, and simply cover the tumor with cotton dipped in oil. If redness and suppuration occur, the discharge of the abscess may be hastened by warm cataplasms and incision. Guillot observed three deaths from complications, and Bouchut[4] a fatal case, in which there was considerable undermining of the pectoral muscle. I have hitherto had no unfavorable result, but in one case I found the mammæ much enlarged, nodular, and containing milk at the end of the third week.

Among the affections of the skin at this period, we will discuss erysipelas, sclerema, and pemphigus, because they develop in a peculiar, almost specific form, and usually run a grave course. There is now a general tendency to deny any independent character to

Erysipelas Neonatorum,

and to regard it as a complication of the condition known as "puerperal infection" of the new-born. I possess no extensive experience concerning this condition, which usually occurs in maternity hospitals, but I believe that I am justified in the conclusion that erysipelas neonatorum is not always a symptom of puerperal infection, but presents similar conditions to those occurring in later life. As in adults, the erysipelas sometimes develops as a symptom of serious general diseases—for instance, pyæmia, septicæmia, typhoid fever, etc.; at other times as a primarily local affection, concerning whose nature, especially its infectious character, we are still in the dark. I think that two forms of erysipelas neonatorum

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[1] Archiv. de méd. 1853. [2] Gaz. méd., No. 17. 1875.
[3] Central-Zeitung für Kinderkrankh., II., No. 4, p. 53.
[4] Traité Prat. des Maladies des Nouveaux-Nés, p. 719. 1867.

should also be distinguished. The first and most dangerous is that form which is combined with the previously-mentioned puerperal infection of the new-born, whose symptoms are added in various forms to those of erysipelas: rapid collapse, very high temperature (to 41°), jaundice, vomiting, and diarrhœa, inflammation of various serous membranes (pleura, peritoneum, joints), convulsions, coma. This form of erysipelas, which does not merely develop in the children of women suffering from sporadic puerperal fever, but which also attacks large numbers of new-born at such times and in such lying-in wards, in which epidemics of puerperal fever are raging. The second form, however, has, as I believe, nothing in common with puerperal infection, or, at least, such a connection cannot be proven. The starting-point of the erysipelas is an injury to some part of the body. A true erysipelas traumaticum develops with the well-known tendency to attack other localities.

But we may assume that, as at certain times different wounds readily give rise to erysipelas, while this rarely occurs at other times, so, in the body of the new-born, injuries have a special tendency to the development of erysipelas, under the influence of a poisoned atmosphere, uncleanliness, unknown infectious influences, which, in these cases are assuredly not of a puerperal nature. For this reason, also, the second form of erysipelas is found much more rarely in private practice among the well-to-do than among the poor. Erysipelas neonatorum may develop, however, even with the best of care and under the most favorable influences. As an example, I will mention the case of a Jewish child of wealthy parents, in whom erysipelas developed after circumcision, at the end of two weeks caused localized gangrene of the scrotum, then an enormous abscess on the back, and proved fatal after general collapse, jaundice, and symptoms of peritonitis. There could be no question of puerperal infection in this case.

The traumatic form of erysipelas may begin a few days after birth, though occasionally it occurs much later. The wounded surface of the umbilicus is very often the starting-point, the genitalia with almost equal frequency, more rarely the anus. In these cases, apart from circumcision, we have to deal not so much with wounds as with the red excoriations formed on intertriginous parts of the skin from the contact of urine and fæces. Erysipelas may also develop from wounds of other parts of the skin, though much less frequently. Most frequently you will first observe the erysipelas at the umbilicus or in the pubic region as a more or less intense redness of the skin, and quite resisting swelling which often has sharply defined, somewhat prominent edges, and feels warm to the touch. All pressure, which temporarily diminishes the redness, but does not make it disappear entirely, is evidently painful to the child. The process is rarely confined to the primarily affected integument; the edges gradually project in various directions, sometimes uniformly, usually more to one side than to another. Thus, it frequently happens that its extension inferiorly predominates, the thighs, legs, and feet being implicated, while it does not, at first, extend above the umbilicus. But even in these cases we not infrequently see the process starting upward from the anus, and involving the nates, back, and thus the upper half of the body. The erysipelas may come to a standstill at any point, or affect the entire body, even the face and scalp. Wherever it appears the skin is bright or dark red, often shining, œdematous, and firm, even as hard as a board, so that the finger with difficulty forms a slight depression. As a rule, however, the redness and tension of the skin are not so intense in the parts affected

at a later period, and the prominent edges gradually become less marked. In many places vesicles filled with yellowish serum form, as in older individuals. The œdema of the skin and subjacent tissue is most marked in flaccid parts, so that the penis, scrotum, vulva, eyelids, hands, and feet may be considerably swollen. Lines drawn with a blunt object on the red skin are visible for a long time as white stripes—in one of my cases for more than fifteen minutes. During the progressive advance of the redness the previously affected parts become pale, and the chest, neck, and legs are sometimes bright red, while the intervening parts have resumed their normal color, though the latter parts may again become affected with erysipelas. After the progress of the disease has stopped, therefore, we not infrequently find irregularly distributed, isolated patches of redness, between which the skin is normal or is more or less œdematous and covered with desquamated epithelium. After the skin has become perfectly pale, œdema may remain, and be distributed over the entire integument.

Remittent febrile movement is present during the entire course of the disease, the evening temperature reaching 39° to 41°, the morning temperature being about one degree less. The pulse is extremely rapid (170 and more) and small, respiration correspondingly rapid and superficial. Many children refuse nourishment at an early period, while I have seen others nurse almost as well as in the healthy condition. The fever usually disappears rapidly after the cessation of the erysipelas, and the patients recover more or less rapidly. But if the affection continues to advance, complications with diseased conditions of other organs readily occur, especially profuse diarrhœa, pneumonia, and peritonitis, and prove fatal. I also observed the latter complication in two non-puerperal cases, with marked distention and tenderness of the abdomen and frequent vomiting. In such cases the inflammatory process probably extends from the integument of the abdomen directly to the peritoneum, through the wounded and swollen umbilicus. But, apart from complications, the high fever may so reduce the vital energies that a fatal result ensues. But we must not lose courage, as children may recover from widespread erysipelas even after weeks of suffering ; while others, after recovering from the erysipelas, fall victims to abscess-formation and necrosis of the tegumentary coverings. I have repeatedly observed these terminations in the scrotum, malleoli, back, arm, and external ear. Smaller necroses of this character may recover.

In a child three weeks old, erysipelas had developed twelve days previously at the umbilicus, and spread upward and downward over a large portion of the body. An abscess remained as a residuum on the left side of the scrotum, which opened and left a deep loss of substance as large as a two-mark piece. The penis and lower extremities were œdematous, and there was extensive red infiltration of the left cheek. Under the use of warm poultices the gangrenous tissue of the scrotum was thrown off at the end of four days, while the erysipelas suddenly affected the left upper limb and resulted in a large abscess. Complete recovery finally occurred.

This case also shows that, after apparent cessation of the extension of the disease, fresh parts of the skin may suddenly become affected.

Treatment is almost entirely unavailing in this affection. In the beginning we may attempt to ameliorate the inflammatory process by large fomentations of lead-wash. Internal remedies, aside from mild purgatives, are entirely useless. If the erysipelas begins to extend, we possess no remedy which will check its spread. Nothing remains but the use of

tonic remedies, wine, and infusion of bark, from which, however, I have obtained no appreciable benefit. Complications must be treated according to their character. Abscesses must be poulticed, opened when distinct fluctuation is felt, and antiseptic dressings employed.

I will also add a few words with regard to erysipelas of infancy and later childhood. In these cases also careful examination usually discloses a wounded surface, which has served as the focus for the reception of the unknown infectious matter. Among these I have most frequently found vaccination, eczema of the scalp, excoriations on the genitals or anus, often as the result of erythema intertrigo; finally, in older children, especially scrofulous ones, chronic rhinitis, with excoriations of the nasal mucous membrane. Nothing is more common under the last mentioned circumstances than an habitual erysipelas (occurring every year or even several times a year), in which the affection spreads from the nostrils over both cheeks, without, as a rule, any further extension. But, despite careful examination, it is not always possible to detect a wounded surface at the point of origin.

In a child fifteen months old, I observed an erysipelas to start from the right labium majus, in which there was not the slightest injury to the skin, and then spread in spots, with free interspaces, to the internal malleoli and the surface of the abdomen. The attempt to check its spread with collodion was entirely unsuccessful; it continued to extend for twenty-two days, when recovery occurred. In another child, five months old, the erysipelas appeared to develop from the vagina, which was the site of fluor albus; the process extended over the entire body and terminated fatally. In an infant, three months old, the affection started from an incision on the right side of the neck, and spread to the right ear and cheek, both eyelids, the forehead and scalp, when it terminated. The treatment consisted of the use of compresses of ice-cold lead-wash, an ice-bag to the head, and quinine internally (0.03 every two hours).

If the erysipelas starts from an eczema capitis, it is not infrequently, in the beginning, concealed under the hair and crusts on the scalp, and manifests itself by fever, whose origin is only recognized when the process spreads beyond the scalp. In these cases we not infrequently observe relapses or rather extension to different sides of the eczema.

A boy, four years of age, with eczema capitis, especially on the left side, was received into my wards in September, 1873. Fever, restlessness, headache, during the night of September 26th. September 27th, continuation of these symptoms without any definite local cause. Temperature, 39.7°; at night, 39.9°. On the next day, redness and swelling on the left side of the head, extending beyond the scalp to the temples; anorexia, tongue thickly coated. Emetic. Temperature at night, 40.6°. During the next few days the erysipelas diminished in intensity, the fever diminished, and, on October 1st, the temperature was 37.5°, and all that was left of the erysipelas were some vesicles on the forehead. On October 11th the fever again began, reached 40.5° on the next morning and evening, and an erysipelas again started from the eczema, extending 3 ctms. beyond the border of the hair. Under the application of an ice-bag to the reddened parts, the erysipelas remained stationary, became pale on the next day, and the fever had disappeared on the 14th.

I have repeatedly noticed the starting-point of erysipelas in tracheotomy wounds made in diphtheria, or even in incisions which had become affected with diphtheria. In one infant it started from a puncture of the scrotum made with an insect-needle, on account of hydrocele. In many cases the erysipelas develops in consequence of vaccination, rarely in the first few days (early erysipelas), usually at the end of the first week or even later. As a rule, the erysipelas only affects one arm, and its extension is less to be dreaded than if both arms were erysipelatous. It is often im-

possible to decide whether we have to deal with the ordinary, though unusually extensive areola of vaccination, or with erysipelas limited to the arm. At times, especially in certain localities (foundling asylums), vaccination erysipelas may develop epidemically, and it is immaterial whether animal or humanized virus has been used. The treatment of all these forms corresponds with that mentioned on page 23.

The physician has much less opportunity of becoming acquainted with the dangerous affection known as

Sclerema Neonatorum,

which is confined almost exclusively to maternity hospitals and foundling asylums, and is even here a rare disease.

The characteristic feature of this affection is the hardness and rigidity presented to the pressure of the finger by the integument of a large part of the body. In the most extreme forms the body feels as hard as if frozen, though not uniformly in all places. It is accompanied by greater or less diminution of temperature. The affected children are feeble, prematurely born, emaciated, and all of them die.

These are the main features of a disease concerning which there has been extreme diversity of opinion up to the present time. As a result of its rarity and imperfect description by most writers, not only did the opinions of physicians differ with regard to the character of the disease, but many had no distinct conception of what was meant under the term sclerema. The credit of having brought light into this chaos belongs chiefly to Parrot.[1] In his work on "Athrepsia" he shows that two entirely distinct affections, true induration and œdema of the new-born, have always been mistaken for one another. He explains this confusion from the fact that true "induration of the cellular tissue" (sclerema) was first described by Underwood, and that this term was soon after (1781) applied by Andry to the œdema of the new-born which is often observed in the Paris Foundling Asylum.

1. True induration (sclerema) occurs exclusively, according to Parrot, in very emaciated (or, as he expresses it, athreptic) new-born babes, especially when the emaciation affects children of moderate stoutness soon after birth. While the skin of atrophic infants shows broad folds around the limbs, in this affection it becomes very tense and smooth, loses its softness, and finally, cannot be raised from the subjacent parts, to which it appears to be firmly united. This change usually begins in the lower limbs, spreads upward over the lumbar region and back, and may finally involve the entire body and even the face. The tension and hardness of the skin increase daily and soon impart to it the feel of hard leather. The soft parts then appear as rigid as stone or wood, pressure with the finger leaves no depression, the color of the skin is usually dirty yellow, and slightly cyanotic at the tips of the limbs. The limbs become immobile, are in continual extension, and only the feeble movements of the thorax, perhaps also the facial muscles, distinguish it from rigor mortis. If the child is held in the air by the neck, he can be placed horizontally like a rigid body, exactly as in cases of trismus neonatorum, for which it may be mistaken, especially when the implication of the lips and cheeks causes closure of the mouth and prevents nursing. But even

[1] Clinique des Nouveaux-Nés, p. 116. Paris, 1877.

when this is not the case, we may be led to suspect tetanic contractions of all the muscles. I especially remember two children who lay for weeks in my wards in a rigid condition, and emaciated to an extreme extent, but were still partially able to nurse, and finally died after the temperature had fallen to 30.0°, and in one case to 28.5° C. At the autopsy, the brain and spinal cord were found perfectly normal, while the integument presented the appearances of sclerema. In a few other cases it was not diffused so widely, but confined to the region of the calves, adductors of the thighs, nates, cheeks, or arms and forearms, the depression of temperature being readily perceptible to the hand or to the finger placed in the mouth. Almost all of my cases were more or less jaundiced.

Parrot found the following post-mortem appearances: marked atrophy and condensation of the skin and rete Malpighii, the cells of which were scarcely visible and formed a compact mass with indistinct contours. In the subcutaneous adipose tissue the connective-tissue bands were more numerous and thickened, the fat had disappeared to a considerable extent, the fat-cells were atrophied, and their nuclei distinct. The blood-vessels, especially in the papillæ, were narrowed to such an extent that their lumen could not be distinguished.

The disease consists, therefore, according to Parrot, of a drying of the skin, with condensation of its layers and atrophy of the adipose tissue. Similar results were obtained in some of my own cases.

2. The second variety, œdema of the new-born, presents an entirely different picture. In this the integument is thickened and removed from the subjacent parts by an œdematous infiltration of the subcutaneous connective tissue. We find all the clinical appearances of œdema, especially swelling of the affected parts, and this may affect only one part of its surface or its entirety. The swelling spreads most frequently from the legs to the thighs, penis, scrotum, or the vulva, the calves being occasionally affected before the feet. Not infrequently the trunk, upper limbs, and cheeks, are implicated, or the swelling only appears in the palms of the hands and soles of the feet. All the œdematous parts are swollen, and feel doughy or hard, according to the degree of infiltration. In severe grades, the affected portions may be so hard as to yield but little or not at all on pressure. The skin is then generally shining, in less marked forms, dull and usually reddish or yellowish, occasionally like bluish marble. When the skin is very tense, a certain degree of stiffness of the limbs and face, with impairment of mobility, may occur, but never attains the same intensity as in sclerema. The temperature of the body usually falls more or less, and in fatal cases may even sink to 30° C., or lower. Post-mortem examination shows infiltration of the subcutaneous connective tissue with a yellowish, serous fluid, while the adipose tissue has been converted into a yellowish red, or brownish, granular mass.

Despite all these differences, there are certain points of similarity between the two varieties with respect to the concomitant symptoms, viz.: the progressive weakness, the small, imperceptible pulse, disappearance of the second sound of the heart, and especially the fall of temperature. I have measured 28.5° in the axilla, others only 22°. External heat produces no warmth, or only very temporarily. The voice becomes weak and whimpering, the respiration slow and interrupted, or frequent, superficial and stertorous in consequence of an attendant pneumonia, which is unable to raise the depressed temperature. The children are usually in a completely apathetic, somnolent condition, and some of them finally have partial or general convulsions; many also suffer from diarrhœa,

which materially increases the weakness. According to the predominance of these or those symptoms, we will find various complications at the autopsy, especially bronchitis, pneumonia, more or less extensive atelectasis, pleurisy, enteritis, congestion, and small hemorrhages of the cerebral meninges and other parts. Hemorrhagic gastritis was found in one of my cases. Other complications may also arise, such as jaundice, diseases of the umbilicus, pyæmia, puerperal conditions, etc.

The pathogenesis of œdema neonatorum is as varied as that of œdema in later life. In some cases it is due to previous erysipelas, and to this class belong those instances in which a dark red color of the genital region, or other parts, has been described, with occasional purulent infiltrations of the connective tissue and partial necroses. In another class, the œdema must be regarded as the result of extreme heart-failure, or extensive atelectasis, followed by stasis in the veins and serous transudation. At times the œdema is caused by nephritic processes, as Elsässer[1] had shown. I observed the following case:

Child, aged four weeks; admitted March 24, 1874. Intertrigo in all folds of the skin; marked œdema of face and limbs. Pulse, 136; temperature, 36.5°. Urine cloudy, albuminous, and extremely scanty. March 27th, severe dyspnœa, cyanosis; pulse, 144 to 160; temperature, 38.4°. March 29th, death. Autopsy showed parenchymatous nephritis, dropsy of the pleuræ, pericardium, and peritoneum; compression of left lower lobe.

You will notice that there is at least one pathogenetic factor common to sclerema and œdema neonatorum, viz., extreme weakness, which may either be congenital or acquired. The diminished energy of the heart, which is sometimes in a condition of fatty degeneration, the interference with circulation, the feeble respirations, and the atelectasis pulmonum, produce the extreme depression of temperature; this perhaps gives rise to the peculiar change in the subcutaneous adipose tissue, like firm mutton suet, which is occasionally found in not very emaciated children.

It follows naturally from the pathogenesis that you will find sclerema and œdema in children who are born prematurely, or are subject to unfavorable influences, such as cold, bad air, and poor food. All other causes mentioned are hypothetical. From the character of the etiological conditions, it is evident that cases may occur in which both varieties of disease occur simultaneously, or at least in succession, in one individual. Parrot describes an instructive case of this kind.

Cases of sclerema are incurable; the patients die in extreme collapse, but sometimes not until the lapse of two to three weeks. The prognosis is somewhat more favorable in œdema, if the cause can be removed. It is, on the whole, most favorable after erysipelas, although fatal cases are not rare. It is less favorable in passive œdema due to extreme weakness of the heart, pulmonary atelectasis, or nephritis. Recovery is exceptional in such instances, and the treatment must be confined to dietetic and hygienic measures. Obtaining a good wet-nurse is a main indication (if the child cannot nurse, it must be fed with mother's milk drawn from the breast, or with good cow's milk), combined with means for artificial warmth (frictions with warm flannels, warming-pans, aromatic baths). Internally we may administer small doses of wine (10–15 drops of Tokay every hour).

The confusion existing with regard to "induration and œdema of the

[1] Archiv für physiol. Heilk., XI., 3. 1852.

cellular tissue" was increased by Bouchut, who brought the scleroderma of adults and older children into connection with sclerema of the new-born. These two affections are so different in their history and course that we cannot conceive how Bouchut's error found any adherents. With regard to scleroderma,[1] I must refer you to works on dermatology.

A third affection of the skin in the new-born,

Pemphigus Neonatorum,

presents numerous varieties, but I will only distinguish two forms, simple (acute) and cachetic pemphigus.

I will illustrate the first form by a few cases from my practice.

The child of a physician, born healthy, had an eruption of pemphigus on the ninth day after birth, which developed on the throat, neck, trunk, and limbs. The hands and feet were unaffected. Some vesicles were as large as a two-mark coin, others like a pea or hazel-nut, filled with yellow serum. There were perhaps thirty to forty in all. The intervening skin had a bright red color. Within a few days the contents of some of the vesicles, which continued to form for twelve days, became cloudy. The general health was good, with the exception of moderate tracheal catarrh. The vesicles burst, the thin crusts became dry, and at the end of another week the disease had disappeared.

A child, aged two weeks, to whom I was called January 8, 1874. The father had a chancre twelve years previously, but no subsequent symptoms. On the ninth day after the birth of child, pemphigus suddenly developed. Under slight rise of temperature, successive crops of vesicles occurred over the entire body; they varied in size from that of a half mark-piece to a thaler, were hemispherical in shape, and of a transparent yellow color. The face was also affected, and the vesicles coalesced in this region into enormous elevations of the epidermis. The integument of the body was very red. The soles of the feet and palms of the hands escaped, except that one vesicle formed in the left palm. General condition unimpaired. The formation of vesicles continued for ten days, and recovery occurred as in the previous case, so that in a few days the site of the vesicles was indicated by thin, dry scabs, after the desquamation of which the skin remained red for some time.

I think that these cases will suffice to indicate the course of the disease in the new-born, as it is not my purpose to dilate upon the description of pemphigus in general. Upon more than one occasion the large number of vesicles and the redness of the skin made me fear the development of complications similar to those of extensive burns; but my fears were always groundless. Apart from restlessness and pruritus during the healing stage (indicated by the movements), no abnormal symptoms were manifested. But a favorable termination does not always occur. Accidental complications, with inflammation of internal organs, sudden collapse, or furunculosis following the disease, have been repeatedly observed as causes of death. I will mention as an important characteristic that, in this form, the soles of the feet and palms of the hand are either unaffected or implicated to a very slight extent.

What are the causes of this disease in the new-born? Occasionally it occurs endemically in maternity hospitals. Thus, Ahlfeld[2] observed twenty-five cases in Leipzig within a period of two months, in children born of healthy mothers, and within two to fourteen days after birth. In these cases, also, the soles of the feet and palms of the hands were

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[1] Cruse: Oester. Zeitschr. f. Päd., II., p. 189. 1876; Jahrb. f. Kinderheilk., XI., p. 318. 1877; Ibid., XIII., p. 36. 1876; Silbermann: Ibid., Bd. XV. 1880.

[2] Arch. f. Gynäkol. V., Bd. I., p. 150.

unaffected, while the fingers were occasionally implicated to a marked degree. Ahlfeld supposes the disease to be of an infectious, or at least miasmatic nature, but adduces no proof in support of his view. Koch [1] thinks that the contagion is spread by midwives, as he observed within a period of three months eight cases of pemphigus in the practice of one midwife; and, in a later report,[2] states that twenty-three additional cases occurred in the practice of the same woman, and none in that of others. He also observed its transmission to adults, and states that, after many negative results, he succeeded in producing a vesicle upon his own arm by inoculation with the contents of a pemphigus vesicle. Vidal [3] also reports successful inoculations. The epidemic described by Moldenhauer [4] (the same as that observed by Ahlfeld) disappeared after strict isolation of the patients. Nothing is known, however, concerning the character of the contagion.

The epidemic, or at least endemic, occurrence of pemphigus neonatorum has not come under my notice. In none of my cases has the disease been conveyed from the child to any of the attendants. The attempt has therefore been made to discover other than infectious causes. Bohn [5] brings it into connection with the exfoliation of the epidermis, which begins on the third day and usually terminates at the end of the first week. He believes that during this time every irritation of the skin, as by articles of clothing, but especially by baths, is capable of converting the physiological action into a pathological one, under the form of vesicle formation, and justly warns against estimating the temperature of the bath by the hand. Bohn refers to a case of pemphigus produced by baths of 31°, and which disappeared soon after the employment of cooler ones. Dohrn [6] expresses himself in a similar manner. The rarely observed transmission of the disease loses its significance as an indication of contagion if we remember that even the secretion of simple eczema not infrequently transmits the disease to the mother or nurse of the affected children.

Our opinion with regard to the infectious character of acute pemphigus neonatorum must therefore still be held in abeyance.

The treatment is extremely simple. I confine myself to lukewarm baths (26–27°), with the addition of bran or gluten.

Pemphigus cachecticus is distinguished from the simple acute form by the fact that it generally attacks the thin portions of the skin—the neck, axilla, and groins, and also the soles of the feet and palms of the hands. The vesicles, which are situated upon a livid macula, are usually only half full, and rarely exceed the dimensions of a pea or hazel-nut. Their contents are less clear, often purulent or bloody, and their number is usually smaller. The patients often present the remains of these vesicles, or the consequent superficial ulcerations, at birth, and they are then regarded as indicative of hereditary syphilis. I have seen cases in which this connection was undoubted. In a child, aged six months, the formation of vesicles began shortly after birth, and had spread to numerous portions of the body. The dirty color of the skin, the chronic rhinitis, and the broad condylomata around the anus, proved the existence of syphilis. But I am not inclined to regard this form of pemphigus as a positive indication of

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[1] Jahrb. f. Kinderheilk., p. 413. 1873.
[2] Ibid., p. 425. 1875.
[3] Gaz. Méd., 29. 1876.
[4] Arch. f. Gynäkol., VI., p. 369. 1874.
[5] Jahrb f. Kinderheilk., IX., p. 304. 1876.
[6] Arch. f. Gynäkol., IX., 3.

syphilis, but agree with Caillault[1] that the formation of vesicles, in the manner peculiar to this variety, is merely the expression of a profound cachexia, which, however, is often due to syphilis. General tonic treatment is indicated, especially breast milk, Tokay wine, and good air. Baths of corrosive sublimate (1.0 to the bath) should only be added when there are other positive indications of syphilis.

Aphthæ of the Palate.

In a previous lecture (page 7) I called attention to small nodules in the mucous membrane of the palate which are observed in many children during the first four to six weeks of life. Very often, also, a round or more oval, whitish yellow patch, surrounded by a red border, is found on each side of the palatal arch, at the level of the apophysis pterygoidea, and immediately behind the alveolar arch of the upper jaw, where the bone shows through the thin mucous membrane; they are usually symmetrical, occasionally larger on one side than the other, now and then shaped like a roll, evidently from the confluence of two patches. The greatest diameter rarely reaches one centimetre. These patches, which bleed readily on contact, are found with extreme frequency in perfectly healthy children. They gradually lose their grayish yellow color, become red, and disappear without leaving a trace. In atrophic and cachectic children, I have occasionally seen them increase in size and depth, and become converted into ulcerations which even extended to the bones. The buccal and palatal mucous membrane is then often covered with sprue, and the children die in consequence of the general disease from which they are suffering, or from complications. You must distinctly bear in mind that these symmetrical patches or "aphthæ" have no connection with syphilis; I would not mention this, were it not for the fact that cases come under my notice in which physicians make this diagnosis. I have always been of the opinion that they were produced mechanically, in nursing, by the pressure and friction of the tongue upon the mucous membrane, which is very thin in this situation. According to Parrot, the epithelium becomes loosened, proliferation of the nuclei in the mucous membrane occurs, followed by separation of the epithelium and the formation of a slight erosion. Occasionally superficial or even deeper ulcerations occur in the raphe, and may be regarded as starting in the little nodules to which we have referred above. But these ulcerations are much rarer, and, with few exceptions, I have only found them in very emaciated children, in whom sharply defined, round, whitish yellow or gray ulcers are often found in the palate, and often lay the bones bare. I do not share Parrot's belief in the syphilitic nature of all ulcers outside of the raphe.

Aphthæ of the palate only require treatment when they become larger under the influence of poor nutrition. I then usually apply a solution of sulphate of zinc (1 to 10) or nitrate of silver (1 to 15). I have never observed them after the age of three months, but Parrot saw a case at two and one-half years.

Only in exceptional instances have I seen such an extension of the aphthæ as to cause them to be mistaken for diphtheria. This occurred in a somewhat emaciated child, aged three months, in whom there

[1] Traité Prat. des Maladies de la Peau chez les Enfants. Paris, 1859.

were originally two patches at the sides of the palate, which gradually increased to such an extent that they finally coalesced, and the entire posterior part of the arch of the palate was covered with a yellowish gray layer. The uvula and tonsils were entirely normal, and this, together with the course of the disease, disproved its diphtheritic character. Indeed, the disease disappeared within ten days without leaving any loss of substance.

Melæna Neonatorum

is a rare affection, of which I have only observed a few cases. It is characterized by hemorrhages from the stomach and intestinal canal, usually from the first to the seventh day after birth, rarely later. Occasionally there is only repeated vomiting of dark blood, from which the children gradually recover. In other cases the hæmatemesis recurs, and blackish discharges of blood from the anus also appear. At times hæmatemesis is entirely absent, and we merely observe rapidly following bloody stools, which at first contain meconium or fæces, but afterward consist purely of fluid and coagulated blood. Other symptoms may be entirely absent, and examination of the abdomen reveals nothing abnormal. In most cases deathly pallor develops within twenty-four to forty-eight hours, followed by cold skin, disappearance of the pulse, and death; but a small proportion of the cases recover after the cessation of the hemorrhage. The mortality varies, according to different authors, from thirty-five to sixty per centum.

The views with regard to the development of this condition vary greatly. Billard explains the hemorrhages from the congestion of the alimentary mucous membrane, which is normally present during the first few days of life, and may be increased by disturbance of the venous circulation, as in asphyctic conditions, pulmonary atelectasis, congenital disease of the heart, enlargement of the liver and spleen. Others attributed melæna to early ligature of the umbilical cord. Attention has recently been directed to small, round ulcerations of the gastric and intestinal mucous membrane as connected with melæna. But there are also differences of opinion with regard to the development of these ulcers. While some regard their origin as inflammatory, others attribute them to ulceration of the follicles or to fatty degeneration of the small arteries. Landau,[1] basing his opinion upon a case of duodenal ulcer, with thrombosis of the umbilical vein, believes the ulcers to be of embolic origin, and produced by thrombi which enter the small arteries of the gastric mucous membrane from the ductus Botalli or umbilical vein. The action of the acid gastric juice upon the portion of mucous membrane not supplied by blood also takes part in the process. Asphyxia and insufficiency of the first respiration present some significance, as they favor stasis of blood and thrombosis in the umbilical vein. Almost as a matter of course the ulcerations have also been regarded as parasitic, and due to colonies of micrococci (Rehn[2]).

Merely from the diversity of opinion with regard to its pathogenesis, we might draw the deduction that melæna is only a symptom of various anatomical processes, as it is in later life. There is no doubt that ulcer-

[1] Ueber Melæna der Neugeborenen u. s. w. Breslau, 1874.
[2] Centralz. f. Kinderkrankh., p. 227. 1878.

ations of the gastric mucous membrane occur quite frequently in the new-born, while melæna is rarely observed, and furthermore that, in those very cases in which multiple ulcers were found on autopsy, blood was not discharged either from the stomach or bowels. I have observed in such cases that the contents of the stomach may appear of a bloody black, and the small ulcers may be covered with a layer of blackish mucus, although no bloody discharges occurred during life. In six fatal cases, Kling[1] found gastric and duodenal ulcers only twice. Landau's case of duodenal ulcer and the fact that intestinal hemorrhages may occur in the adult from embolism of the mesenteric artery, should lead us to examine the gastric and intestinal arteries in all cases of melæna. On the other hand, hemorrhages must be regarded as possible without ulceration, if the venous pressure is increased by obstructed respiration, as was shown by Epstein, who produced hemorrhages into the gastric mucous membrane of animals by suspension of respiration.[2] It may also be mentioned that intestinal hemorrhages may be connected with a hemorrhagic diathesis, especially with "puerperal infection."

The history of a number of cases shows that recovery may occur even though the hemorrhage has been profuse enough to cause symptoms of collapse, general coldness, disappearance of the pulse, and upturning of the eyeballs. We must therefore endeavor to check the hemorrhages, even under apparently the most unfavorable circumstances. The application of cold fomentations or an ice-bag to the abdomen, and warm flannel to the arms and legs, is to be especially recommended. If the children cannot take the breast, milk cooled in ice should be given by the spoon, and this latter plan is indeed preferable when severe hæmatemesis occurs. Liquor ferri sesquichlorati (gtt. j. every two hours in a teaspoonful of oatmeal gruel) should be administered, or ergotin (0.03–0.05 at a dose) either internally or subcutaneously. Enemata are not advisable, as they do not reach the upper portions of the intestines, and are apt to cause tenesmus and a renewal of the hemorrhage. With respect to prophylaxis, Landau warns against too early ligature of the umbilical cord, which should only be performed after respiration has been thoroughly performed and vigorous cries have been uttered.

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[1] Ueber Melæna neonatorum. Inaug. Diss., München, 1875.
[2] Archiv f. Experim. Pathol., Bd. II.

LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

PART II.

DISEASES OF INFANCY.

I.—The Atrophic Conditions of Children.

At no time does nutrition play so important a part as in the period from birth to the end of the first year. You are aware that a number of obstacles may prevent the nourishment of the child at the breast of the mother, as should naturally occur. Diseases of the mother, poverty, undeveloped condition of the nipples, are some of the most frequent and excusable obstacles, while, on the other hand, many mothers, especially among the higher classes, simply neglect this duty. Among this class, a wet-nurse can readily be obtained, but, among the poor, resort must be had to artificial feeding. I do not deny that the latter plan, when carefully and properly carried out, often furnishes satisfactory results. But the struggle for existence, illegitimate birth, frivolity and ignorance, foolish superstition—all these factors act as disturbing elements, and explain the enormous frequency of disorders of nutrition and the terrible mortality among infants belonging to the poorer classes. But this is caused not only by insufficient and improper nourishment; it is also due in part to the bad air which these children are compelled to breathe, to the want of cleanliness, and to neglect during the first stages of diseases from which they may be suffering. Some of these causes are also active in children's hospitals and foundling asylums, where we have abundant opportunity of observing their effects. These are included under the term atrophy. The symptomatology of this condition naturally varies according to the stage in which it comes under observation. The first sign is the cessation of development, which can be determined only by carefully weighing the children from week to week. But the deterioration soon becomes evident of itself, the adipose tissue disappears more and more, the integument of the face and entire body becomes flabby, wrinkled, yellow in color, and not infrequently presents a branny desquamation of the epidermis. At this stage the functions of the organs, especially of the digestive tract, are still entirely intact or nearly so, and proper nourishment and care may lead to a favorable termination. But, in the majority of cases, this is prevented on account of the poverty of the patient; disturbances of the digestive organs, especially vomiting and diarrhœa, are superadded and lead to the development of the most severe forms of emaciation, which exclude all hope. The face is pale yellow, pointed, with marked prominence of the bones, and numerous

transverse and vertical wrinkles, especially around the nose and mouth and on the forehead; these become deeper with every movement of the facial muscles. The eyes are widely open and staring, or half closed with a dull expression, a picture of perfect apathy, interrupted occasionally by painful distortion of the wrinkled features, feeble cries or hoarse whimpering. The flabby, sallow skin hangs loosely over the bones, and the shoulder-blades, vertebræ, ribs, and pelvic bones distinctly show the outlines of the skeleton. Upon the neck and abdomen the skin forms large folds, which, on account of their loss of elasticity, preserve their shape for some time, as if they were made of dough. The adipose tissue appears to have disappeared entirely, and the muscles feel like thin, flabby cords. The integument of the genitals, anus, and heels is not infrequently reddened, and contains larger and smaller abscesses and furuncles in various places. Sprue is often found in the mucous membrane of the mouth and palate.

You must remember that defective nutrition of the tissues may arise from various causes. Even when all the circumstances seem to point to simple atrophy, *i.e.*, due to improper and insufficient nourishment, we should always ask ourselves whether other causes may not also be operative; and among these I refer especially to tuberculosis. I will now merely mention that tuberculosis occurring during the first years of life gives rise to symptoms which vary greatly from those in later life, on account of the simultaneous implication of many organs which are intimately connected with the formation of the blood, *viz.*, the lungs, lymphatic glands, spleen, etc., and the local symptoms are subordinate to those of general failure of nutrition. The positive proof of hereditary predisposition to tuberculosis, and the physical signs of consolidation of lung-tissue, are alone decisive in diagnosis, as various râles due to bronchitis may be heard in simple atrophy, and a diarrhœa which is present may be due to chronic intestinal catarrh as well as to intestinal tuberculosis. In individual cases repeated examination is often necessary in forming a diagnosis. From the beginning there are very often disorders of digestion, either frequent vomiting immediately or shortly after feeding, or changes in the evacuations, which become more frequent and fluid, and contain yellowish or greenish lumps. The amount of urine generally diminishes, chiefly on account of the lessened appetite. The child is irritable, cries a good deal, and sleeps less than usual. These symptoms increase in intensity with the progress of the disease. The evacuations from the bowels become more and more fluid, of a dirty green color, and very offensive; normal or constipated bowels are rarely observed. The appetite is lost, and the child grows too weak to feed from the bottle or breast. The quantity of urine continually grows less, becomes darker, and Parrot's examinations not infrequently showed the presence of albumen or a small amount of sugar. If no complications are present, the respiratory organs show no abnormal physical signs, but respiration is very superficial and weak, like the action of the heart, which may fall to sixty beats per minute or even less. The temperature of the body may finally fall to 35.0°, and even lower. On account of the heart-failure the previously dirty yellow skin becomes slightly cyanotic at the extreme parts (lips, fingers, toes, nails). The large fontanelle is depressed, and may diminish more or less in circumference, because the edges of the bones are pushed across one another; this is due to the diminution in the volume of the brain. The half-closed eyelids complete the picture of fatal collapse, which often develops almost unnoticed, as the condition of the child may be like that

of death during the last few days of life. True sclerema may develop in the new-born during the final stage.

No definite statement can be made with regard to the duration of the affection, as this depends upon so many circumstances. In the new-born it often proves fatal within the first few weeks or months of life, while older children may eke out their miserable existence for many months, and succumb to an exacerbation of diarrhœa or an acute pulmonary affection. Broncho-pneumonia is one of the most frequent causes of death, and may be due to accidental colds or the prolonged dorsal decubitus. I may also draw your attention to the fact that when care is not taken in administering nourishment, milk may be drawn into the respiratory passages and give rise to bronchitis and pneumonia, if death does not rapidly ensue from asphyxia. This may also happen from regurgitation of the contents of the stomach into the mouth.

Post-mortem examinations of simple atrophy have shown an almost complete disappearance of the fat beneath the skin and around the internal organs, atrophy and pallor of the muscles (even the heart), and usually marked anæmia of all parts. Pulmonary atelectasis is often found. Among the complications, broncho-pneumonia, catarrh, and follicular inflammations of the intestinal canal are the most common. We occasionally find thrombosis of the venous system, especially in the sinuses of the dura mater and in the renal veins.

The prognosis depends mainly on the severity of the disease and the possibility of placing the child under better vital conditions. If the atrophy has not progressed too far, and no severe complications are present, if tuberculosis can be excluded, and you possess the means of furnishing good nourishment and nursing, a favorable prognosis may be given. The rapidity with which recovery may occur under such circumstances is astonishing. Little hope can be entertained, however, in practice among the poor, that such a result will be obtained.

In discussing treatment I do not fail to recognize the difficulties connected with the subject. Its thorough elucidation would necessitate a discussion of the care of the healthy child from birth to the period of weaning, as all errors with regard to diet, cleanliness, clothing, etc., will be reflected in the nutritive condition of the infant. I would also be compelled to enter the domain of sociology and of public health.

Under these circumstances, I must therefore confine myself to an explanation of the most important factor, viz., nutrition. Concerning the breast-milk of the mother or nurse, I have little to say, nor will I dilate upon the physiology of nutrition, etc. I may state that even children nourished in the natural manner may become atrophic, if the ingested milk produces continual dyspeptic symptoms (vomiting or diarrhœa) as the absorption of the necessary amount of chyle is therefore interfered with. Cases also occur occasionally in which a nurse's milk does not agree with the child, so that the latter is continually suffering from digestive disorders, or, although these do not attain any considerable severity, does not progress favorably in its development. The same nurse will then successfully nurse another child, so that the first infant must possess some peculiar idiosyncrasy, since it also progresses finely after another nurse has been obtained. If vomiting and diarrhœa are constantly recurring in an infant, if the weight of the body does not increase, or perhaps even diminishes, we should not delay in securing another nurse. In order to determine the diminution of weight at an early period,

the child should be carefully weighed every week, allowance being made for the clothing, condition of the stomach, bowels, and bladder.

The appearance of the child, the dyspepsia, and beginning atrophy, are, in my eyes, more significant than the methods by which we endeavor to determine the character of the mother's or nurse's milk. The microscope will inform you with regard to the number, form, and size of the milk-globules, but the results of these examinations do not agree with reference to the influence which this or that microscopical deviation possesses upon the condition of the child. The chemical examination of the milk is even more difficult. I therefore advise you to make the condition of the child the criterion in your judgment of the milk, as in the selection of a nurse you should be guided by the appearance of her own child. It may also be remarked that the insufficiency in the quantity of the nurse's milk can be determined less by manipulation and pressure of the mammæ, than by the dryness of the diapers and the continuous crying of the infant after nursing; after the child has been satisfied it should fall into a quiet sleep.

The situation is much more serious when a child, suffering from beginning atrophy, must be fed from the bottle. I have previously called your attention to the small quantity of saliva secreted during the first months of life, and you will therefore understand that during this period, *i.e.*, until the tenth week, no amylaceous food should be administered. When mother's milk cannot be obtained, cow's milk is the only proper substitute during the first three months of life; the latter, however, contains more casein, twice as much fat, and less sugar. The most important difference, however, is the fact that the casein of cow's milk forms tough coagula which are soluble with difficulty, while that of the mother forms loose, small coagula. The coagula of mother's milk are much more readily dissolved by the pepsin and hydrochloric acid of the gastric juice, than those of cow's milk, and the fæces of children fed on the latter contain more undigested casein than those fed at the breast. The less serious differences can be compensated by suitable dilution. During the first three months the proportion of milk to water should be as 1 to 3, in the second three months, as 1 to 2, and in the third, half and half. After the ninth month you may give two parts of milk to one of water, or undiluted milk; both the milk and water used should be first boiled, in order to destroy any fermentation germs which they may contain. The proportions given above must, of course, be modified according to the quality of the cow's milk. The well-being of atrophic children of the poor depends, above all, upon obtaining pure, fresh cow's milk, and more attention should be paid to this subject by the community than has hitherto been done. Cow's milk is the best substitute for mother's milk during the entire period of infancy. I consider the administration of other substances advisable only when good cow's milk cannot be obtained, or when it gives rise to constant vomiting and diarrhœa. But this does not occur often, and these bad effects cannot infrequently be obviated by allowing the milk to be taken cold, after it has been boiled. But there are some cases in which even the cold milk is not digested, and we must therefore resort to other substitutes. Condensed Swiss milk is apparently the most convenient and best. When it is placed under the microscope, the entire field is covered with crystals of milk-sugar, which disappear as if by magic when water is added to the object-glass, and we then only see numerous well-formed milk-globules. Although I have in some cases used the condensed milk to advantage for months, I do not

favor this form of nourishment, as the enormous amount of sugar (about sixty per cent.) necessary to preserve the milk very often produces acid fermentation and diarrhœa. If this does not occur, we may employ the milk without fear. During the first three months, one part condensed milk is given to twenty-two parts water, from the third to eighth months, 1 to 18, and later, 1 to 12. Oatmeal- or barley-water may also be used for dilution.

Among the many artificial substitutes for milk, Nestlé's flour has obtained an especial reputation. This is composed of wheat flour, the yellow of egg, condensed milk and sugar, 1,000 parts containing 20 parts nitrogenized matter and seven parts of salts. One tablespoonful is usually boiled with nine to ten tablespoonfuls of water. I must, however, call your attention to the fact that it may spoil under certain circumstances, and then act injuriously. According to my own experience I can recommend Nestlé's flour as a very suitable article of diet after the tenth to twelfth weeks. But I believe that other similar foods, such as those of Gerber, Giffey, Liebig, Frerichs, the preparations of Cham, Vevey, and Montreux are equally valuable.

Among other renowned substitutes for mother's milk, I will here mention Liebig's soup[1] and Biedert's cream mixture.[2] The preparation of these substances is too intricate to meet with general introduction among the poor, in whom artificial nourishment must be especially considered. I have repeatedly made faithful trial of both preparations in my clinic in a number of atrophic children, but could not convince myself that they were more efficacious than feeding with cow's milk or Nestlé's flour.

Wine, especially pure Tokay, is an admirable aid in the nourishment of atrophic infants. Whether other kinds of wine possess equal merit, I do not know. I always prefer the Hungarian wine, of which my teacher, Romberg, was accustomed to say that it was not alone "lac senile," but also "lac juvenile." During the first few months of infancy twenty to twenty-five drops may be given three or four times a day, either pure or in a teaspoonful of water. At a later period the dose may be increased to a couple of teaspoonfuls pro die. A lukewarm bath (27° to 28° R.) should be given daily for purposes of cleanliness, and, if the weakness increases, aromatic substances may be added (a few handfuls of chamomile and calamus). Well-aired rooms, cleanliness, careful nursing, are desiderata which can only be obtained in the minority of cases.

Nothing need be expected from the use of drugs in the treatment of atrophy, except when complicated with diseases of the respiratory organs or intestinal canal.

II. Sprue.

This is observed most frequently in the new-born and during the first months of life. The symptomatology of the disease varies according to its severity and the circumstances under which it occurs.

First Grade.—Isolated, white, slightly prominent points and spots, which can be readily removed with a spatula, but leave a drop of blood

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[1] Wheat flour and ground barley malt, āā half an ounce. are mixed with gtt. xxx. of a solution of bicarbonate of soda (2 : 11 parts of water); then stirred with one ounce of water and five ounces of milk, and boiled until thick. It is then removed from the fire, allowed to stand for a few minutes, and again heated until it is thin and sweet, *i.e.*, until the starch has been converted into sugar.

[2] Virchow's Archiv, Bd. 60, Heft 3 u. 4.

behind if violence is used, are found upon the mucous membrane of the lips, tongue and cheeks, especially in the folds between the cheeks and alveolar border, and between the lips and gums. The mucous membrane is otherwise unchanged, and no other disturbance is present. This form occurs often in perfectly healthy children, when the mouth is not kept clean. It is sometimes difficult to distinguish, at first, between sprue and the remains of milk, but the latter can be at once removed, while the former is more adherent to the mucous membrane.

Second Grade.—The entire mucous membrane of the mouth extending into the pharynx is dark red, purple, and strikingly dry. Numerous round or irregular white dots and spots are seen everywhere, especially on the tongue, cheeks, lips, and hard palate, and here and there, particularly in the folds and on the tongue, they coalesce into larger patches. The mouth appears to be painful, as the children distort the face during nursing, or refuse to nurse. In a still more severe degree the tongue, cheeks and hard palate are covered by a white membranous coating, while numerous patches are visible on the lips, gums, velum palati and tonsils. These severe forms only occur in atrophic children or those exhausted by serious diseases, and this explains the fact that the mucous membrane grows pale, on account of the increasing anæmia. In the last stages I have often found the sprue upon a perfectly pale, almost livid mucous membrane. Occasionally the white color changes to a dirty gray or yellowish, the latter especially on account of bile-staining from vomited matter. The longer it continues, the more firmly does the sprue adhere to the mucous membrane. Under such conditions, the new-born often present the ulcerations mentioned on page 30. Under the microscope the sprue is found to consist chiefly of threads of spores. These threads appear as long, straight, or variously curved, transparent, sharply defined cylinders, about fifty to sixty millimetres long and three to four millimetres wide, composed of a number of members. The mature ones possess one or more branches, starting from those portions of the trunk at which the coaptation of the individual parts is marked by boundary walls. Internally the threads usually contain some molecular granules, and perhaps a few oval bodies, probably developing spores. Around the origin of the threads, masses of round or oval spores, from which the former develop, are almost always found. The common term, "oïdium albicans," is not a proper one, according to the investigations of Grawitz,[1] as the fungus is identical with mycoderma vini. In addition to the fungoid elements, the microscope also shows numerous epithelium-cells, more or less fat-globules and red blood-globules.

All the symptoms which have been ascribed to sprue belong to the primary disease in whose train it occurs. It is not by any means confined to those parts which are open to clinical examination, but also occurs in the deeper parts of the pharynx, and often in the œsophagus, especially in the lower two-thirds, where it appears either in the same manner as in the mouth, or as a more or less complete cylinder. As a rule, it is not of a pure white, but pearl or yellowish gray, and terminates in a sharply defined line immediately above the cardiac end of the stomach. I have only found sprue on the gastric mucous membrane in one case, but the stomach was not carefully examined in all post-mortems. I mention this because Parrot[2] not infrequently found it in the stomach.

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[1] Zur Botanik des Soors u. s. w. Deutsche Zeitschr. für Prak. Med., 20. 1877.
[2] L. c., p. 223.

In order to recognize it, the mucus covering the fungus must first be removed by a stream of water, when the sprue will appear in the form of isolated or conglomerate papillæ, which are in part only distinguishable with a magnifying-glass. The larger patches often show a central depression, and thus present a decided similarity to a favus-crust. The sprue is usually found on the posterior wall of the stomach along the lesser curvature and near the cardiac end. It is so adherent that it is difficult to remove it from the mucous membrane by a stream of water or by scraping. It is very rarely found beyond the stomach, but Robin observed it in the small intestine, and Parrot twice in the cæcum.

It is a noteworthy fact that though sprue may be strongly developed in the pharynx, it never extends to the posterior part of the nasal cavity, even in cases in which there is a communication between the latter and the buccal cavity, on account of the presence of cleft palate; occasionally, however, it is found on the mucous membrane of the glottis. As this is the only portion of the respiratory mucous membrane affected by sprue, we must agree with the opinion of Berg and Lélut, that pavement-epithelium, but not ciliated epithelium, is a favorable soil for the development of the fungus. It has been found in the lungs in a few cases, probably by aspiration from the pharynx. A portion of the fungus lies superficially between the epithelium-cells; another part is situated deeper, so that the threads are distinctly seen to enter perpendicularly into the mucous membrane (Wagner,[1] Parrot). It also appears from some observations of Zenker and Ribbert,[2] in which the spores were found in the brain, that they may proliferate into the blood-vessels, and then be carried as emboli to other parts.

It never appears to occur, or at least attain any considerable development, on the perfectly healthy mucous membrane of the mouth. Even in mild cases there has probably been partial irritation of the mucous membrane by decomposing particles of milk. This is more clearly seen in cases belonging to the second grade, which are always preceded by a striking dryness and redness of the mucous membrane, and roughness of the tongue, the fungus starting in these situations; this is furthered by the deficiency in alkalinity. The small amount of saliva secreted in the first months of life must also aid the formation of acids in the mouth. Atrophy and feebleness of the child are especially favorable to the development of the spores, and this has been directly shown by the experiments of Delafond[3] upon animals. This also agrees with the clinical experience that similar eruptions of sprue not infrequently occur in later life in the last stages of phthisis and in severe cases of typhoid fever. Perhaps many a case of "diphtheritic complication" of typhoid fever, in which no autopsy is held, may depend upon sprue in the pharynx.

The spores are probably chiefly conveyed to the buccal mucous membrane by means of the food, but direct transmission from badly cleaned bottles is also possible. Observers are at variance as to whether the fungus may be conveyed from the mouth of the child to the nipple of the mother or nurse. Seux[4] did not observe a single instance among 1,600 cases of sprue; but Mignot,[5] basing his opinion on a few observations,

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[1] Jahrb. f. Kinderheilk., I., p. 58. 1868.
[2] Berliner klin. Wschr., p. 618. 1879.
[3] Gaz. Hebdomad., p. 909. 1858.
[4] Recherches sur les Maladies des Enfants Nouveaux-Nés, p. 29. Paris, 1855.
[5] Traité de Quelques Maladies pendant le Premier Âge, p. 223. Paris, 1859.

maintains the possibility of its occurrence, and Delafond also observed it in his experiments on animals. The attention of the nurse must therefore be called to the possibility of such transmission, and the nipples be kept perfectly clean and frequently washed with alkaline fluids. In cases of doubtful diagnosis the microscope will decide. A membrane-like desquamation of epithelium from the mucous membrane of the tongue, and especially of the gums, in the form of thin, grayish white patches, is occasionally mistaken for sprue. The microscope then reveals epithelium-cells and an amorphous granular mass, but no fungous elements. In a few instances these conglomerations of epithelium are found exclusively under the tongue, where they form a rolled-up, milky white, tranverse thread.

I observed this in two nursing infants, one of whom was blooming and plump, the other atrophic and presented numerous abscesses of the skin and a bed-sore on the elbow. Neither of the infants had any teeth, but the mucous membrane of the mouth was reddened throughout, and bled readily on contact. The white deposit under the tongue could be removed quite readily; it was somewhat more firmly adherent to the frenum, and there left a drop of blood behind. Under the microscope, I could only recognize fat-globules (probably milk), epithelium-cells, and an amorphous connecting substance, but no trace of sprue elements; and I presume that the increased desquamation of epithelium, due to the congestion of the mucous membrane, had assumed this shape from the repeated gliding of the lower surface of the tongue over the edge of the alveolus in nursing.

The local treatment of sprue only promises success in cases of the first grade. Mechanical rubbing is usually sufficient. The attendant should vigorously rub the patches off with the finger, which has been surrounded with fine line and dipped in water, even if this gives rise to a slight hemorrhage. Whenever a new eruption becomes visible, this operation is repeated. In cases of the second grade the matter is different. You will also succeed in removing the fungus by the above method (still better if the finger is dipped in an alkaline solution, 5 parts of chlorate of potash, borax, or benzoate of soda, to 100 of water); but the general disease is the chief factor, and relapses will therefore continually occur. Under these circumstances, I have, with advantage, repeatedly brushed the entire mucous membrane of the mouth with a solution of nitrate of silver (1 or 2 parts to 100 of water) after the sprue had been rubbed off.

III. Hereditary Syphilis.

The period in which the beginning of atrophic conditions and the development of sprue are most frequently observed also affords the best opportunity of becoming acquainted with the symptoms of hereditary syphilis.

The children presented to your notice are usually two to three months old, and are well or poorly nourished, according as they have been nursed at the breast or bottle-fed. A marked degree of atrophy is not by any means a characteristic of infantile syphilis, as a number of children under my observation, especially those fed at the breast, presented normal fulness of the body, while those fed artificially showed a tendency to atrophy. But the severer forms of atrophy are due not to syphilis alone, but to the coexistence of other factors.

One of the earliest symptoms in many cases is snuffling during respi-

ration, caused by swelling of the nasal mucous membrane in parts which are not open to inspection. There is also usually an obstruction of the nares by yellowish or brownish crusts, and a sero-mucous discharge, occasionally mixed with blood; the nose may be somewhat swollen externally (coryza syphilitica). This is one of the most constant symptoms, which either precedes the others or accompanies them. It is soon followed by bright red, but chiefly somewhat brownish round spots as large as a five- or ten-penny piece, which are at first isolated and appear especially in the region of the eyebrows, chin, anus, palms of the hands and soles of the feet (roseola syphilitica). Many of these spots show slight branny desquamation of epithelium, or are covered with large scales; others, and sometimes nearly all, appear shining when viewed from the side. The spots on the chin and buttocks are irritated by the saliva, urine, and fæces, and become converted into red, moist excoriations, whose specific characters may be obscured by erythema of the surrounding parts (intertrigo). The position of these excoriations, the presence of patches which are not thus complicated, and the existence of the coryza, however, will justify the suspicion of syphilis and a resort to anti-syphilitic treatment.

If this is not done, the progress of the disease soon removes all doubts. The patches spread over a large portion of the body, especially the forehead, the vicinity of the mouth, and the extremities, and coalesce in many places into large, dark red or yellowish brown, more or less desquamating patches, covered here and there with crusts due to the drying of excoriations. The palms of the hands and soles of the feet are diffusely reddened and covered with desquamated epithelium, and the heels especially are often tense and of a shining red color. Excoriations occur at the angles of the mouth, and are covered with a whitish deposit; there are fissures in the mucous membrane of the lips, which bleed readily, and these, together with the crusts on the eyebrows and the coryza, form a picture which permits even the inexperienced to make a diagnosis. In many cases, also, the hair falls out, especially that of the eyebrows. I have also corroborated, in a number of cases, Trousseau's observation of a brownish color of the skin peculiar to infantile syphilis.

But you must not expect all the symptoms of the disease to be as well marked as I have described above. Frequently only a part of the symptoms is present, and deviations are also not uncommon. Instead of roseola, I have repeatedly observed dark red, round papules on the soles of the feet, lower limbs, and around the anus, or dark red, infiltrated patches, covered with thin whitish scales; here and there the remains of pemphigus (page 29). In many cases, especially in very young children, the entire skin was diffusely reddened and covered with large flakes of yellow epidermis mixed with sebum. As the most rare symptom I have noticed vesicle-like, moist (eczematous) eruptions; they appeared to me to be due to maltreatment of papular eruptions, especially from scratching or the contact of irritating secretions and excretions. I have more often observed the development of ulcers from the previously mentioned excoriations around the anus, scrotum, etc., and the intertrigo of the inguinal regions also manifests a tendency to the formation of whitish gray ulcerations, surrounded by an infiltrated red border. But I cannot subscribe to Caillault's[1] opinion that only the presence of condyloma latum justifies the diagnosis of congenital syphilis, as this eruption has not been present in a considerable number of cases. Nor do I regard

[1] Traité Prat. des Maladies de la Peau chez les Enfants. Paris, 1859.

condylomatous formations as one of the earliest symptoms, since, except in a few cases, I have always observed them at a later stage in children who were a few months old, or had suffered from a relapse. Under such circumstances they occurred quite frequently, especially at the angles of the mouth, on the tongue, in the dimple of the chin, the inguinal folds, around the anus, on the scrotum and vulva, at times on the inner and upper part of the thigh, most rarely on the alæ nasi and outer canthus of the eye. Their appearance is the same as in the adult, and they have a marked tendency to maceration by secretions, so that they gradually terminate in whitish gray, fissured ulcerations after desquamation of the epidermis. In some cases the condylomata form conglomerate masses. I have also repeatedly observed onychia, combined with thickening and claw-like deformity of the nails, which are finally shed by suppuration of the matrix.

The mucous membranes also present abnormal appearances. Apart from the almost constant coryza, I have also observed conjunctivitis with purulent secretion, fluor albus, occasionally redness and swelling of the meatus urethræ, with pain on micturition. Condylomatous, or rather, gummy, hard, dark-colored deposits occur on the dorsum of the tongue, especially posteriorly, and the tonsils are occasionally the site of flat ulcerations, which have resulted from condylomata. But these affections are not frequent, and I must again warn you against regarding the often-mentioned (page 30) ulcers of the palate as syphilitic. More or less marked hoarseness, advancing exceptionally to complete aphonia, may sometimes be combined with the cutaneous syphilides. In the following case the loss of voice was almost the only symptom of syphilis.

Charles C——, aged four months, suffered for two months from hoarseness, and lately from complete aphonia. No cough; normal respiration. Laryngoscopic examination showed nothing abnormal. The child was well-nourished, but had some brownish cicatrices around the anus. Further investigation showed that the child had suffered, at the age of two months, from coryza and a macular, desquamating eruption, which were relieved by calomel. Diagnosis: syphilitic affection of the vocal cords (condylomatous ulcer). I ordered hydrarg. oxydal. nigr. 0.007 twice a day. At the end of four weeks the voice was entirely normal.

Slight enlargement of the lymphatic glands is often found, at times separately behind the ears, at the lower end of the arm, or in masses in the cervical, axillary, or inguinal chains. These enlargements always belonged to the most obstinate symptoms, and often continued after recovery, although it then remained doubtful whether they were not accidental complications. But I cannot agree with Bednàr that these glandular enlargements are extremely rare.

Syphilitic affections of the osseous system were formerly regarded as very rare. A case of this kind was observed and described by me as early as 1861:[1]

Anna B——, aged two months, atrophic, although nursed by the mother; presented April 4th, because she had not moved her arms for two weeks. Both upper extremities lay flaccid and immovable. The left arm, when lifted and then dropped, fell as if powerless, while slight resistance could be detected in the right arm. Sensation and temperature normal in both arms. The condyles and lower third of left humerus much swollen, and a movable gland as large as a pea found on its inner side. Cervical, axillary and inguinal glands swollen and hard; palms of feet red, shining, and slightly desquamating. Nares clogged, at times a slight bloody purulent discharge. Treat-

[1] Beiträge zur Kinderheilk., p. 192. Berlin, 1861.

ment: Hydrarg. oxydat. nigr. 0.015 twice a day, inunctions of ung. kali. iodid. over the swollen surface. At the end of a week the swelling had disappeared, the coryza improved, the arms slightly movable. April 16th, mobility of arms normal, coryza disappeared, and the mercury was then changed for syr. ferri. iodid. (gtt. v., t. i. d.). All syphilitic symptoms had disappeared by April 21st.

Two other cases observed by me were entirely similar to the above. In these instances only the lower end of the humerus was the site of syphilitic periostitis and ostitis, but the following cases show that other long bones may also be affected:

Child, aged ten weeks, suffering from coryza, with crusts in nostrils, and shining, red, flat umbilicated papules around the anus and on the nates. Painful swelling of lower epiphysis of left radius and ulna, middle phalanx of left middle finger, first and second phalanges of right middle finger. Mercurial treatment. Almost complete recovery in five weeks.

Child, aged three months, well nourished. Intertrigo, with erosions, around anus and genitalia. Coryza, with purulent discharge and crusts. During past four weeks swelling of upper epiphyses of bones of right forearm; tenderness on pressure. Right arm flaccid, and is moved very little. Mercurial treatment. Marked improvement in three weeks.

You thus see that not only the long bones present swelling of their epiphyses, but also the phalanges of the fingers, which then present the picture of scrofulous osteomyelitis (pædarthrocace), *i.e.*, a hard swelling, covered at first with normally colored and movable skin, which grows red in the course of time, forms small fistulous openings, and finally, after suppuration lasting years, heals with a funnel-shaped cicatrix. I have observed this form a few times, but always in the fingers, never in the toes. Taylor[1] and Lewin[2] have recently discussed this "dactylitis," which must be regarded as comparatively rare. But you must not forget in cases of pædarthrocace that this affection is not only scrofulous, but may also be due to hereditary syphilis. On the other hand, you should not consider all epiphyseal swellings, especially at the lower ends of the radius and ulna, as syphilitic, even if other suspicious symptoms are present, and particularly in children over six months of age, since they may be due to rachitis. In these cases the swelling of the epiphyses are unaffected by mercurial treatment, while the true syphilitic symptoms disappear. You should, therefore, endeavor to determine whether rachitis and syphilis are not present in combination. During the first six months of life you may consider the epiphyseal swellings as syphilitic, and treat them accordingly. I do not recognize any difference in the form of the swelling (Taylor states that the syphilitic variety is characterized by a sudden, abrupt beginning); but the epiphyseal swelling of syphilis may be unilateral, and this is never true of rachitis.

You will have been struck in most of the above-mentioned cases with the difficulty in motion, or complete immobility of the upper limbs, which also causes the arms to drop, after having been lifted up, like dead, heavy bodies. Bednàr[3] was the first author who attached any importance to this symptom; among sixty-eight cases of hereditary syphilis he noted paresis of the arms sixteen times, of the legs once, and of all the limbs twice. His description agrees entirely with the symptoms noted above. Bednàr appears inclined to regard the paresis as due to a myopathic

[1] Syphilitic Lesions of the Osseous System. New York, 1875.
[2] Charité-Annalen, Jahrg., IV.
[3] Krankheiten der Neugeborenen, u. s. w., iv., p. 227. Wien, 1853.

affection and to a flaccid condition of the muscles. In my opinion the immobility of the limbs is a result of the painful affection of the bones. A proof of the correctness of this view lies in the fact that the mobility returned, in all my cases, with the diminution in the swelling of the bones. It may be said in opposition that Bednàr does not mention any swelling of the bones in his cases, and I can also adduce some cases in which the paresis occurred without any discernible osseous affection:

A child, aged six weeks, with roseola on the arms and legs, face, and trunk, coryza and conjunctivitis. Both arms lay perfectly flaccid; a few slight movements in the fingers. No swelling of the bones. At the end of a week, mercurial treatment had produced disappearance of the eruption, and improved mobility of the upper limbs.

A child, aged three months; arms and legs immovable and flaccid almost from birth. Coryza, with snuffling and discharge; slight roseola of face and around the arms. No swelling of the bones. Mercurial treatment. Coryza and roseola cured in less than three weeks; arms moved well, legs also, but latter cannot be completely extended on account of resistance in knees.

Wegner's[1] investigations have made us better acquainted with the significance of these pareses without any discoverable disease of the bones. In syphilitic new-born and young children, the long bones at the transition of the diaphysis into the cartilage of the epiphysis constantly present a diseased process, due to excessive proliferation of the cartilage-cells and retarded ossification of the already calcified tissue. The new formation of vessels in the bones does not occur at all or only imperfectly, and in consequence of the deficient nutrition the cells slowly undergo shrinking and fatty degeneration. On section, this process manifests itself by a narrow yellowish or orange-colored, somewhat zigzag line running along the border of the epiphyseal cartilage, and which, according to Wegner, is formed by the necrotic tissue, separates the diaphysis and epiphysis, and may lead to complete separation of the epiphysis by an "inflammatory suppurative complication." The process is always multiple, is especially frequent at the lower end of the femur, in the bones of the legs, arms, and in the ribs, but occasionally in all the long bones.

These observations are corroborated by Waldeyer and Köbner,[2] but they think the process is a gummatous one, due to excessive cell proliferation, which results, on account of compression of the vessels, in necrosis of the intermediate tissue and thus in separation of the diaphysis and epiphysis. Whichever explanation is correct, the important clinical fact remains that there is a diseased process at the boundary of the epiphysis which produces discoverable symptoms (swelling, pain, immobility) only in the smallest proportion of cases during life, but whose influence on the mobility of the limbs should not be underestimated, even when other symptoms are absent. Separation of the epiphysis, which can be determined during life, only occurs in exceptional instances, and is manifested by abnormal mobility at the border of the epiphysis and an unusual "looseness" of the hand (Köbner and Waldeyer). I myself could prove crepitation at the point in question in only one case. The changes described do not always occur in a uniform degree in all the bones. I could never positively notice the propagation of the diseased process from the epiphysis of the bones to the adjacent joints. But other authors (Güterbock,[2] Parrot) state that they have repeatedly observed purulent arthritis

[1] Virchow's Archiv., Bd. 50, p. 305.
[2] Langenbeck's Archiv., Bd. XXIII., Heft 2.

as an attendant of hereditary syphilis. Bouchut often found the diaphysis of the long bones uncommonly dense and hard, and frequently covered with periostitic deposits; Wegner, in rare cases, saw gummy periostitis on the inner surface of the cranial bones or small gummy nodes of the pericranium.

The action of infantile syphilis is not restricted to the skin, mucous membranes, and osseous system. As in the adult, other organs may also be affected. The implication of the testicles was practically unknown until very recently. Désprès[1] described three cases in children from the ages of seven months to three years, in one of whom Cornil found, on autopsy, hypertrophy of the tunica vaginalis and albuginea, in addition to interstitial orchitis and epididymitis. At least ten cases have come under my own notice since 1874. The testicle is more or less enlarged, hard and tough, somewhat uneven or nodular; its size varies from that of a hazel-nut to a chestnut. I found both testicles affected four times, the left also four times, and the right twice. The youngest child was three months old, the oldest two and one-half years, in whom an autopsy was obtained.

A child, two and one-half years of age, was admitted to hospital with broad condylomata around the anus and psoriasis syphilitica. Both testicles considerably enlarged and nodular. Inunction treatment. In a month all symptoms disappeared, but testicles unchanged. Death in three months from vomiting and diarrhœa. Autopsy.—Testicles very large and firm; extensive interstitial connective-tissue hypertrophy, most marked in corpus Highmori; no gummata discoverable.

In this case, therefore, as in those of Désprès and Hutinel,[2] the affection was an interstitial orchitis, partly epididymitis, which will resist treatment if it has advanced to fibrous proliferation. The liver may also become affected with interstitial inflammation and, in a portion of the cases, this will only be recognized at the autopsy.

A girl, aged seven days; father syphilitic. Child had roseola and psoriasis, marked atrophy; no hepatic enlargement. Death from exhaustion. Autopsy.—Interstitial hepatitis. Liver somewhat enlarged, very firm, flat. Acini not visible; parenchyma traversed throughout by whitish bands of connective tissue. Cortical substance of kidneys extremely tough. Fundus of stomach hemorrhagic, and its mucous membrane covered with a layer of bloody mucus. Yellow epiphyseal zone in many of the long bones; periostitis of right humerus.

In other cases there is a noticeable enlargement of the liver, which permits a diagnosis. In a few children, during the first months of life, I observed, in addition to eruptions, condylomata and coryza, a more or less marked enlargement of the liver, which disappeared after mercurial treatment. But those cases are rare in which disease of the liver can be determined clinically. This is still more true of the spleen, kidneys, and pancreas.

Implication of the nervous centres in syphilis appears to me to occur much more frequently in adults than in children. In a few isolated cases I have observed contractures, which were improved or cured by specific treatment. The first case[3] of this kind occurred in a boy, aged fourteen months, presented at my clinic on November 24, 1867.

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[1] Bullet. de la Soc. Chir., 1875. [2] Revue mensuelle, 2, 1878.
[3] Beiträge zur Kinderheilk., N. F., p. 421. Berlin, 1868.

Examination showed contracture of right arm at elbow, the fingers of right hand, and both lower limbs at the knees. Standing, sitting, grasping with right hand impossible. Biceps of arm and flexors of legs tense, and every attempt at extending the parts causes sharp cries. Papules around arms and on scrotum, coryza, enlargement of clavicular and axillary glands. The history showed that the child had suffered for months from coryza, vesicular eruption and ulcers, and had had epileptiform attacks at the age of three weeks, after which the contractures slowly developed. Mercurial treatment. December 23d, the right hand could be opened and knees slightly flexed. February 27th, treatment changed to iodide of potassium. March 30th, child began to walk and use the right arm.

The influence of antisyphilitic treatment was unmistakable, but the question arises whether the contractures were not myopathic (due to interstitial myositis) and independent of the nervous system. That this may occur in hereditary syphilis appears to me to be proven by the following case:

A syphilitic child, aged four months, had rigid contracture and hardness of the flexors of the legs, so that the legs were constantly semiflexed; extension only partially possible. Mercurial treatment for a number of weeks produced complete recovery.

I have never observed, in infantile syphilis, any true cerebral symptoms, paralysis of one or more limbs, or any convulsive attacks, and the following case appears so much more noteworthy:

A child, aged two years, had, in addition to pædarthrocace, an unusual psychical condition, an alternation of precocity and dulness, without motor disturbances. Death occurred from diphtheria. The autopsy showed a number of nodular tumors as large as a cherry beneath the pia mater, in various parts of the cerebral tissue and cerebellum; they were translucent gray at the periphery, and in a condition of fatty and calcareous degeneration in the centre. As no tubercles were discoverable, and periostitis of the tibiæ was present, we are led to regard the growths as syphilitic gummata, and so they were declared in the Pathological Institute of the Charité.

A few other similar cases are reported in literature, but were merely based on microscopical examination. But, as the diagnosis between gummata and tubercles cannot be made with certainty by means of the microscope, we must be very sceptical with regard to these cases. Changes have also been found in other parts of the vascular system, which remind us of the "syphilitic affections" of the cerebral vessels. Thus, Schütz[1] describes the small arteries of the kidneys and skin as markedly narrowed, their walls much thickened from hypertrophy of the muscular coat and adventitia, and attributes to these changes the numerous small hemorrhages which were found on the skin, in the subcutaneous cellular tissue, muscles, kidneys, and other organs. Further investigations are necessary to decide whether these vascular changes are due solely to syphilis. The course and termination of hereditary syphilis are affected less by the character of the symptoms than by the nutritive condition of the patient. Syphilitic infants, who are nourished in the natural manner, usually do well under specific treatment. Artificially fed children, especially those who are feeble and atrophic from birth, are in great danger. Not infrequently death occurs suddenly, but this is not by any means remarkable, as sudden deaths occur quite often in atrophic children. Under favorable circumstances the disease often takes a happy turn with surprising rapidity. We are astonished to find the eruptions, condylomata and swelling

[1] Prager med. Wochenschr., Nos. 45, 46. 1878.

of the bones diminished, under the influence of mercury, after the lapse of five to six days, and entirely disappear in a few weeks. But relapses are frequent in infantile syphilis, and we can satisfy ourselves of this fact in dispensary practice, in which the children so often pass out of our observation soon after the first disappearance of the symptoms. Treatment should therefore be continued for at least some weeks after the disappearance of the symptoms, though even then the danger of a relapse is not removed. In the majority of cases, however, we can entirely cure the disease within the first, or, at least, the second year. But you must always be prepared for a reappearance of the affection in the later years of childhood, and doubts may then arise whether we have to deal with a relapse of hereditary syphilis, with direct infection, or with the so-called "syphilis tarda." But even in those cases in which the disease has been thoroughly cured, it often leaves a disturbance of the organism which predisposes it to the development of rickets.

It is often very difficult to determine with certainty the origin of infantile syphilis. With extremely rare exceptions, all cases developing in the first two months of life must be regarded as hereditary. It is sometimes manifested as pemphigus in the first few days, and as other eruptions and coryza in the first two weeks of life. Much more frequently, however, the children present no striking symptoms for the first four to six weeks. The development of the disease after the second or third month is rare, and when the period is still longer, it is always doubtful whether we do not have to deal with a relapse or with direct infection. Parents often attempt, by false statements, to lead the physician to believe that the child has been infected by a syphilitic nurse or attendant. I do not deny the possibility of such infection, but in no case of this kind, coming under my own notice, have I been able to positively exclude the hereditary origin of the disease. I have, however, observed direct infection of infants in poor families, by syphilitic females who lived in the same rooms, perhaps by the use of sponges and other toilet articles used in common. The formerly often accepted view of the infection of the child during delivery, from syphilitic disease of the maternal genitals (syphilis adnata), is very doubtful. I have observed no case of this kind, or of infection by vaccination. That a communication of the disease from inoculation with vaccine-lymph obtained from a syphilitic child (whether the virus has been mixed with blood or not) is possible, will not be denied, but this point is still disputed; nor do I consider myself justified in expressing a decided opinion, since not a single well-authenticated case has come under my notice. I have, however, seen many cases in which ulcerations and various eruptions developed after vaccination, and would readily be mistaken for syphilis by the inexperienced or superficial observer. Much less do I dread infection from the milk of a syphilitic nurse, if her nipples are healthy.

Apart from isolated exceptions, therefore, all cases developing in the first months of life must be regarded as hereditary. Although the study of this heredity has been vigorously prosecuted, the views of authors are still at variance in many respects. We know with certainty, however, that syphilis may be inherited either from the father or mother. The father communicates the disease directly in the semen with which he impregnates the female, the mother communicates it through the ovum from which the fœtus is developed. The parents must therefore be in the secondary stage of syphilis; and primary affections can only infect the child by leading to the development of secondary symptoms in the mother

during pregnancy, a source of hereditary syphilis which is denied by many authors. Whether this view is correct, or whether infection of the fœtus is not possible from the blood of the mother, who has become syphilitic at a later period, I consider as still undecided. Those who deny communication through the blood also dispute the possibility of infection of the mother from the blood of the fœtus which has been infected by the father. Others regard this as possible, and Hutchinson calls attention to the experience that women who are married to syphilitic men not infrequently become affected only when they conceive, but not so long as the marriage remains unfruitful. However this may be, it is positive that syphilitic mothers abort with uncommon frequency, or do not produce viable children. This tendency to abortion is significant from a diagnostic point of view, as it often tips the scale, in doubtful cases of hereditary syphilis, in favor of the latter.

After a lapse of time, and especially after specific treatment, a temporary improvement or recovery of the parents may occur, and this explains the fact that the tendency to abortion is often strongest in the first period of these marriages, and then disappears more and more. A remarkable alternation of healthy and syphilitic children is not infrequently observed, and can only be explained by the supposition that the syphilis of the mother manifests itself from time to time, and then again becomes latent. In this manner heredity may be possible for a very long time, and the following case shows that even twenty years may elapse.

The father of the child in question had a chancre when he entered matrimony. The first child, born a year after marriage, suffered repeatedly from swelling of the tibiæ, and at the age of seventeen still had extensive periostosis of the left humerus. During her twenty years of married life the mother suffered repeatedly from suspicious angina and obstinate ulcers around the knee-joints. During this period she gave birth to two perfectly healthy children, then aborted several times, and in the twentieth year of her married life was delivered of a boy who manifested the most marked symptoms of hereditary syphilis.

I doubt whether it is possible to determine from the form of infantile syphilis its paternal or maternal origin. The view of Bärensprung, Hecker, and Keysel,[1] that internal syphilis, especially implication of the liver, shows inheritance from the father, is not by any means proved with certainty.

I now come to the treatment of the disease. I can condense my large experience in this field into the short proposition : Mercury is the only sure remedy for infantile syphilis. Its effects are often astonishing, and neither iodide of potassium nor iodide of iron can be compared with it. Of the mercurial preparations I prefer calomel and hydrarg. oxydat. nigrum in doses of 0.01–0.015, given morning and evening. The latter sometimes causes emesis, especially at the beginning of the treatment. I regard every other form of administration of mercury as improper, whether by mercurializing the nurse or some of the lower mammalia. Nor can I recommend to you inunction with gray ointment or the subcutaneous injection of corrosive sublimate. They should only be taken into consideration when extensive syphilitic eruptions are not present, or intestinal complications contraindicate the internal use of mercury. All children in whom I resorted to the inunction treatment had passed the second year, and were suffering from relapses of syphilis, characterized

[1] Bayer: Aerztl. Intelligenzbl., No. 21. 1876.

by condylomatous formations rather than extensive exanthemata (1.0 unguent. ciner. daily). I also used injections of corrosive sublimate in these cases with good effect. I can only recommend baths of corrosive sublimate (1.0 to a bath) in those cases in which marked atrophy, vomiting, or diarrhœa appear to render the internal administration of mercury dangerous.

Condylomatous growths were powdered with calomel, or brushed daily with a solution of nitrate of silver (0.5 : 15 of water) if they had ulcerated. I also advise the latter plan for the nasal mucous membrane, if the coryza resists internal treatment.

The great importance of the natural diet for syphilitic infants was referred to above. If the mother is syphilitic, she may, of course, nurse her affected child. If the mother presents no evidences of the disease, she should only be allowed to nurse the child if the lips and buccal cavity of the latter present no abnormal appearances. Even the secretion of coryza must be regarded as a not altogether safe factor in nursing from the breast (Roger). Although Günsburg[1] concludes from his observations that hereditary syphilis is not conveyed to the nurse, but that all cases in which this appears to have been the fact must be explained by *acquired* syphilis of the children, I must regard this conclusion as very problematical, and therefore advise you to be cautious. In my opinion the physician is bound to inform the nurse that her nursling is suffering from syphilis, and it then remains for her to decide whether she will run the risk or not. Although grave family secrets may thus be revealed, and the physician be guilty of an indiscretion, I nevertheless believe that all these reasons should not induce us to subject a healthy nurse to syphilitic infection without her knowledge. But the matter is not so serious as it seems to be at first sight, as I have had no opportunity of observing an infection of the nurse which has developed in this manner. The nurse should be enjoined to be very cleanly, and watch carefully for any excoriation of the breast which may develop.

Finally, a few words with regard to the conduct of the physician toward the parents. I should advise, in practice among the higher classes, if you do not receive spontaneous confessions, to keep your opinion to yourself, informing only the nurse; and, if you are convinced of the entire innocence of the mother, to take the father into your confidence. Fortunately, the symptoms of the disease are themselves sufficiently characteristic to permit a diagnosis without any confession of the parents.

But cases occasionally occur in which even the experienced physician cannot make a diagnosis with certainty, and in which it would be very unwise to excite the parents by vague questions. In doubtful cases you may relieve your conscience by employing the mercurial treatment, which will soon decide whether syphilis is really present.

I will conclude this chapter with a few remarks on syphilis of later childhood.

The twenty-one cases which I take as the basis of this description occurred between the ages of two and twelve years, and, with the exception of three, in girls. The history showed that in five cases the symptoms were due to a relapse of hereditary syphilis; in the others it remained doubtful whether the disease was hereditary or had been contracted at a later period. That the occurrence of the first manifestations of hered-

[1] Oester. Jahrb., II., p. 169. 1872.

itary syphilis in later childhood (syphilis tarda) is indeed possible I will not deny, but no undoubted case has hitherto come under my notice.

In five girls, from four to twelve years old, the symptoms could be traced to a rape, or, at least, to an attempt at rape, though the statement of the oldest child (twelve years) that she had been surprised by a man while sleeping on a pair of stairs, seemed very suspicious in view of her bold conduct. In only one of these cases did examination show a torn hymen, but the vicinity of the hymen was often reddened and sensitive, and more or less leucorrhœa was present.[1] In two sisters, aged nine and eleven years, the disease was said to have been derived from the syphilitic nurse by one of the girls, who transmitted it to her sister.

The symptoms of syphilis of later childhood are not materially different from those of later life. The predominance of the condylomatous forms alone appears to me to be noteworthy, although I cannot agree with Violet[2] that syphilitic exanthemata never occur under these circumstances. The mucous papules appear in more or less dense masses, not infrequently partially macerated and ulcerated at the surface, around the anus and on the labia majora, where they occasionally form nodular masses, which deform the vulva. The inner surface of the thighs, the nates, even the folds between the neck and breast, were occasionally the site of these new formations, in addition to which whitish condylomatous proliferations, partially eroded and traversed by fissures, appear very often at the angles of the mouth, the tonsils, and adjacent palate, more rarely on the cheeks. Gummy changes in the dorsum of the tongue occurred with special frequency in the shape of round or more rectilinear infiltrations of the mucous membrane, which were sharply defined from the surrounding parts by their dark color and greater resistance, projected occasionally above the surface, and in this comparatively rare event appeared of a whitish color or ulcerated.

The rarity of syphilitic exanthemata has been previously mentioned, but their occurrence is proven by several cases in which roseola of the forehead and scalp, psoriasis palmaris and plantaris were observed. As a rule, small, movable enlargements of the lymphatic glands were present, and in a number of cases those visible externally were distinctly swollen. I had frequent opportunity of observing affections of the osseous system.

A girl, aged twelve, had complained for a year of severe pain in the right arm, especially at night. The humerus was swollen to double its usual size, uneven and angular, very tender on pressure. Syphilitic infection at the age of three years. Enlargement of some glands in neck and axillæ. Had been treated formerly, but relapses always occurred.

Girl, aged eleven. Tender enlargement of right tibia since a year and a half ago, and severe nocturnal pains. Enlarged glands under the maxilla; no other syphilitic symptom. Iodide of potassium given. Decided improvement in three weeks, and in eight months no trace of the disease was left.

Considerable losses of substances in the pharynx, complete destruction of the uvula, adhesion of the velum palati to the posterior wall of the pharynx, and ulcerative destruction of the hard palate I have only

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[1] In three girls, aged respectively four, six, and twelve years, I observed, as the result of attempted rape, more or less marked inflammation of the vulva, with leucorrhœa and numerous sharp condylomata on the labia, without syphilis.

[2] Syphilis Infantile. Paris, 1874.

observed exceptionally. I regard the dental changes (short, narrow incisors, separated from one another and notched), described by Hutchinson, so much the less as a positive sign of late syphilis, since this condition of the incisors is not infrequently found in children who are entirely free from syphilitic disease.

The treatment was always mercurial, except in the very rare cases in which an affection of the bones was alone present. I then tried iodide of potassium (2:120), which rapidly relieved the pains and caused disappearance of the swelling of the bones, but did not prevent relapses. Otherwise mercury was employed from the beginning, either in the form of inunction with unguent. ciner. (1.0 to 2.0 daily), of which, on the average, 25.0 to 40.0 were used, or as injections of corrosive sublimate (0.004 pro die), which were continued for about two weeks.

IV. The Dyspeptic Conditions of Infants.

Before I turn to the diseased conditions included under the term "dyspeptic," I will direct your attention to a symptom which occurs so often that it can hardly be considered pathological, viz., the vomiting of infancy. This is simply the result of over-hasty nursing, causing overloading of the stomach, which then relieves itself of the surplus by a sort of regurgitation. According as this occurs immediately after nursing or a few minutes later, the milk is ejected, either in the fluid state or mixed with casein coagula. This may be repeated constantly after nursing, or more rarely, according to the quantity of nourishment taken. Any movement will promote its occurrence. This regurgitation is also aided by certain peculiarities of the infantile stomach, viz., its more vertical position and the slighter development of the fundus and larger curvature, which causes a relatively smaller capacity of the organ. So long as the children remain healthy in other respects, medical interference is unnecessary, and we should merely direct the mother to feed the child less frequently and for a shorter period, and to let him lie quietly in bed after nursing.

But cases occur not infrequently in which primarily simple vomiting acquires a more serious significance, inasmuch as the growth of the child ceases, and the beginning signs of atrophy show that we have to deal with something more than mere regurgitation of superfluous milk. Under these circumstances emesis occurs after the ingestion of comparatively small quantities of milk, and the physician may be in doubt for days whether the condition is simply a dyspeptic one or a beginning cerebral affection, especially tubercular meningitis. I will here mention that dyspeptic vomiting is often preceded and accompanied by eructations, indicative of an unusual amount of gas-formation in the stomach. As a rule the vomited milk is mixed with more or less tough mucus, an appearance which I regard as specially important. During the first few days or weeks of this condition, which I call *gastric dyspepsia*, the fæces may retain their normal character, and, at the most, show a greenish or brownish color, but they generally present an admixture with mucus, and an unusually fœtid smell. They may not be increased in frequency. As a rule, these children suffer a great deal from wind, and before this is discharged, they usually show tympanitic distention of the abdomen, especially in the region of the transverse colon.

In another series of cases (*intestinal dyspepsia*), vomiting is either

entirely absent or plays a subordinate part, and the dyspeptic symptoms are, from the beginning, of an intestinal character. Many children have severe crying spells, draw themselves together, distort the eyes, and only become quiet after loud sounding flatus has been discharged (flatulent colic). The passages, which at first present the above-mentioned appearances, soon grow more frequent and more fluid, contain numbers of yellow or green undigested coagula of casein and lumps of fat, more or less tough mucus, have a greenish, even spinach-green color, and a very nauseous odor. Fifteen to twenty passages may occur in a day, but usually their number, especially in the beginning, is limited to five or ten. The appetite is diminished, the tongue sometimes clean, sometimes coated grayish white, and the urine is scanty.

As soon as these symptoms become manifest, you should not delay in investigating their causes. As a matter of course artificially fed children are most frequently subject to these dyspepsias; improper constitution or adulteration of cow's milk is often to blame, and, still more frequently, nourishment with mealy substitutes at a period when the insufficient salivary secretion forbids their employment (page 36). But children fed at the breast do not escape; it is a matter of experience that a change in the character of the breast-milk, whether caused by emotional disturbance, overwork, deficient nourishment, appearance of the menses, may give rise to dyspepsia in the child. As a striking example I may mention the case of a child, aged four months, who did splendidly until his nurse became affected with suppurative tonsillitis, which caused great pain and robbed her of sleep. The child immediately suffered from diarrhœa until the nurse's tonsillar abscess ruptured. The child's dyspepsia disappeared upon the same day. Among the poorer classes infants at the age of five or six months often eat various articles such as potatoes, cabbage, etc., and it is not surprising, therefore, that dyspeptic conditions should be among the most frequent affections of infancy. You will accordingly find dyspeptic diarrhœa most frequently at the period of weaning, whether this occurs at the end of the first year or a few months after birth.

What occurs, under these conditions, in the stomach and intestines? The old idea of a "formation of acid" in the digestive organs gave place, as pathological anatomy developed, to the anatomical explanation that the dyspeptic symptoms were due to catarrh of the gastric and intestinal mucous membrane. But this theory proved insufficient, so that resort was again had to the chemical explanation which, in my opinion, is the only correct one. We evidently have to deal with fermentative processes in the contents of the stomach and intestines, the final result of which is the formation of fatty acids. We cannot, at present, state with certainty in what manner this process is carried on. Perhaps the irritation of the injurious food causes an irritated condition of the gastric and intestinal mucous membrane with profuse production of mucus. The alkaline mucus neutralizes the hydrochloric acid of the gastric juice which is necessary to normal digestion, and as this cannot act upon the contents of the stomach in a normal manner, the sugar of the milk or that which is formed from amylaceous articles of diet is converted into lactic acid, from which butyric and fatty acids are finally produced. This process of fermentation either occurs in the stomach alone (gastric dyspepsia), or, and much more frequently, also in the intestinal canal (intestinal dyspepsia); for it is readily understood that if the fermenting gastric contents are not entirely discharged in vomiting, the process will continue as soon as these contents pass into the intestines. The acid smell from the

mouth, the masses of mucus in the vomited matter, which usually smells sour, the fœtid evacuations, their acridity (they readily produce erythema around the anus), the flatulence and discharge of fœtid gas from the anus, as well as the eructations from the stomach,—all these symptoms constitute the clinical expression of the abnormal chemical process. In a child a few months old I had the opportunity of observing not alone a distinctly ascertainable dilatation of the stomach, but also stinking eructations and flakes as yellow as butter in the vomited masses of milk and mucus. By means of a stomach-pump these masses were repeatedly removed from the stomach, and this was always followed by rapid depression of the previously markedly distended gastric region. At a later period of life, and even in adults, we see similar conditions develop from overloading the stomach with food and drink which are injurious with regard to quantity and quality. But while, in older children and adults, this process usually terminates with the discharge of the fermenting masses, and therefore occurs almost always in an acute form, this rapid termination only occurs in infants if the diet is immediately regulated in a normal manner. Less frequent nursing, substitution with water boiled with a little gum arabic, greater dilution of cow's milk usually suffice to remove the disturbance within a few days. But the dyspeptic symptoms are too often unnoticed for a long time, and an attempt is made, without the aid of the physician, to remove them by mealy articles of diet (oatmeal gruel, flour soups, etc.). The abnormal fœtid evacuations and often also the emesis thus continue for weeks, and result in the progressive atrophy which I have described above (page 34). An anatomical process is then superadded, inasmuch as the continued irritation of the fermenting contents finally causes a catarrhal affection of the mucous membrane. Post-mortem examination shows congestion and swelling of the mucous membrane in patches, and the solitary follicles as well as Peyer's patches are unusually prominent. Occasionally the changes in the mucous membrane are extremely slight despite the continuance of the disease for months.

The further course of this condition depends chiefly on circumstances, especially on the possibility of obtaining proper nourishment and care. In some cases improvement and exacerbation alternate for months, usually because the instructions of the physician are carried out more faithfully at some times than at others. The more the chronic intestinal catarrh and the follicular changes develop, the more persistent and profuse the diarrhœa becomes, and the passages are, in large part, very watery, yellow or green, and contain extremely slight quantities of solid detritus of the food, numerous flakes of mucus, and occasionally small punctate collections of blood. The following case is an illustration:

Emma S., aged two months, admitted February 19, 1873, with hereditary syphilis. Mercurial treatment. On the 29th, diarrhœa dyspeptica, vomiting rare, alternating improvement and relapse, the latter continuing from January 18, 1874; four to five passages daily of a green color, streaked with blood, and occasionally reddish brown. Increasing atrophy, sprue. Rectal enemata of a solution of acetate of lead, with which a teaspoonful of thick, blackish blood was often discharged. January 21st, temperature 35.6°, collapse and death. Autopsy showed fatty liver and catarrh of the large intestine with five follicular ulcerations in the neighborhood of the ileo-cæcal valve.

The atrophy now progresses rapidly, the mouth becomes covered with sprue; the face, hands, legs and feet are frequently œdematous. Fever may be entirely absent or a very slight rise of temperature occurs in

the afternoon and evening, but thirst is often very much increased on account of the copious losses of blood. The further course is the same as that already described under the head of "atrophy."

Much more rarely the dyspepsia begins so acutely that a grave and even fatal condition of exhaustion may develop in a few days. The symptomatology is the same as that of infantile cholera, but these cases always occur sporadically and even in midwinter. A gross dietetic error may almost always be detected as the cause. Severe vomiting, profuse, rapidly repeated, thin and stinking evacuations, which gradually become clearer and more colorless, intense thirst, changed features, especially sinking of the eyes, cool skin, disappearance of the pulse and depression of the fontanelle are found in this affection as in cholera. The cause of the rapid collapse noticeable in these cases is apparently the profuse serous evacuations, which are produced by the irritation of fermenting masses upon the mucous membrane and by the reflex action upon peristalsis. These enormous serous losses explain the rapid absorption of the parenchymatous fluids which cause the emaciation of the features and the depression of the fontanelle, and also the extreme weakness of the heart which finds its expression in the apathy and somnolence (arterial anæmia and venous hyperæmia of the brain), in the disappearance of the pulse and the depression of temperature. Such cases may prove as fatal as the epidemic cholera of the summer months, but, as a rule, their prognosis is more favorable, because the affection usually ceases after the discharge of the deleterious contents of the intestines. In fatal cases, the autopsy shows no changes or, at the most, slight catarrhal changes of the gastric and intestinal mucous membrane, occasionally extreme pallor with slight swelling of the follicles.

Under these conditions you may expect to find the peculiar gastric change known as gelatinous softening of the stomach, or gastromalacia. The slightest grade, which is found quite often, consists of a pulpy softness of the mucous membrane of the fundus and posterior wall of the stomach, so that it can be removed with the handle of the scalpel like a thick solution of gum; these portions are the ones which are most subject to the action of the gastric contents in the usual position of the corpse. More rarely the softening involves all layers of the stomach, which become converted into a gray, reddish or dark brown, semitranslucent jelly, which smells like butyric acid and reddens litmus-paper. The serous coat usually remains intact, but this also may readily tear before the autopsy. No trace of inflammatory changes can be found and the microscope shows a mucoid substance containing a few epithelium-cells and some intact blood-vessels filled with dark clots. It is now positively known that gastromalacia is a chemical change in the stomach occurring after death, a post-mortem self-digestion of the walls of the stomach by its contents, which can only be expected in cases in which nourishment has been taken and death occurred during digestion. This also explains the fact that adjacent organs, the spleen, left kidney, omentum, diaphragm and even the lower lobe of the left lung are found more or less digested and softened.

The dangerous consequences which we have found develop from a dyspepsia which is at first neglected, make early and careful treatment our duty, which can only be fulfilled with hopes of success when the surrounding conditions of life are favorable and our directions are carefully carried out.

In many cases you will only appear at the bedside after nature has

removed the deleterious contents of the digestive canal by profuse discharges through the mouth and anus. You merely find the child enfeebled, and need simply regulate the diet. If the child is nursed at the breast, the possibility of any deleterious change in the milk must be considered. Emotional disturbances and overwork only affect the milk temporarily, and the child can be restored to the breast as soon as the dyspeptic evacuations have ceased. But it is always wise to let the breast be taken less often than usual for a period of twenty-four to thirty-six hours, and to give instead some thin oatmeal or barley gruel. If the appearance of menstruation in the nurse always produces dyspepsia in the child, the nurse must either be changed or the child weaned. In the majority of cases, however, I have observed no bad effect of the menses on the milk. The same remark holds good of acute diseases in the nurse which may give rise to dyspeptic conditions though by no means always. Unless the nurse's disease promises to be short and mild, you should endeavor to secure another nurse. If the child has been fed on artificial diet, you may, after the attack has passed, cautiously make trial of its accustomed food. If relapses occur, a change of food becomes necessary and the question then arises whether a nurse should be taken instead of the previously used artificial food. We should always advise the former plan if circumstances permit, though we may encounter many difficulties as the children prefer the bottle to the unaccustomed nursing at the breast, and often refuse the latter decidedly. But with patience we will usually succeed in accustoming the child to the breast. But even then the nurse's milk may, for various reasons (page 52), not agree with the child and cause dyspepsia, so that we are again forced to change nurses.

The main rules for the dietetic treatment of infantile dyspepsia can only be given in a general way, as cases often occur which cannot be fitted to these rules. Thus, I have occasionally seen dyspepsias which continued despite several changes in the nurses and only ceased when the children had been weaned. Others reacted against cow's milk with dyspeptic symptoms, so that other substitutes, especially the children's flours mentioned above, became necessary. But I would advise you to be led rather by practical experience than theoretical considerations, and to make repeated experiments with cow's milk before resorting to other substitutes. Experience teaches that, under these conditions, the milk is better borne cold than warm; after being boiled, it should therefore be allowed to cool or, especially in acute dyspepsia, placed on ice. The majority take it with willingness and even with eagerness, and the period at which the children again prefer the warm milk, has always been a favorable indication of beginning recovery. As long as emesis continues, it will be well to give the cold milk in spoonfuls as drinking from the bottle readily induces overloading of the stomach and vomiting.

A child, aged ten months, weaned for six weeks, suffering from diarrhœa for one and one-half weeks. December 19th, sudden increase, numerous thin, yellow discharges, vomiting infrequent, abdomen tender on pressure. Calves' broth given solely during last few days. Within twenty-four hours, twenty evacuations and frequent vomiting occur ed, great heat and thirst. December 22d milk given with arrow-root, followed by repeated vomiting and more severe diarrhœa. I then gave hourly a few teaspoonfuls of milk, cooled in ice, and small bits of ice and ice-water mixed with a little sugar, to relieve the thirst. The only medication was an emulsion of almonds, cooled on ice, given in teaspoonful doses. Decided improvement on the next day; vomiting had only occurred once after crying, and three normal evacuations occurred. December 24th complete convalescence; child now refused the cold milk and preferred warm milk and arrow-root.

But although I have seen a large number of cases like the preceding, the use of ice-cold milk is not always successful and we must then give barley gruel, infusion of salep, arrow-root or children's flours. In uncontrollable vomiting we may be led to administer nourishment per rectum, and I have a few times tried enemata of pepton (prepared according to Adamkiewicz' method, about one teaspoonful to half a cup of meat broth) but have seen no good result, probably because the peristaltic movements of the intestines were increased by the enemata, which were soon ejected in an almost unchanged condition.

With regard to medicinal measures, I would recommend in fresh cases (not more than three or four days' standing) calomel as the first remedy (0.005—0.015 every three hours, with pulv. gummos. 0.5 according to the age of the children) (P. 2). The effect of this remedy is probably anti-fermentative. Mialhe's view that calomel is converted into corrosive sublimate by the chloride of sodium of the gastric and intestinal contents is only correct in so far as this change occurs very gradually and only where large quantities of calomel remain in the intestines for a long time. But both circumstances are absent in the case under consideration. Nor do I believe that calomel has a favorable influence on the catarrhal irritation of the mucous membrane. Let us hold fast, therefore, to the therapeutic effect which has been practically determined! Relief of emesis, diminution of evacuations, decrease of fœtor, and a more pulpy consistence of the stools are often noticeable on the second or third day of its use, and in a number of cases no other remedy is necessary. Perhaps the mild purgative action, which even these small doses possess in infants, also has a favorable effect. If the affection has continued four days or more, we cannot promise such favorable effects from the calomel, but even then it is worth a trial.

Next to calomel comes hydrochloric acid (P. 3) which also yields good results in cases no longer acute. This remedy antagonizes fermentation as Schottin[1] has shown. In addition "it dissolves the protein substances in the stomach and can act vicariously for the deficient gastric juice."

In recent cases you should not add opium as its constipating action causes tympanitic distention of the intestines. But if several days have elapsed and the watery evacuations have not ceased, the addition of tinct. thebaica (about three to four drops to the mixture) is to be very highly recommended, since by diminishing peristalsis, it gives the hydrochloric acid time to exercise its action.

There are many physicians who prefer alkaline remedies, especially bicarbonate of soda. Although this may temporarily neutralize the acidity of the fermenting gastric contents, it cannot control the fermentation itself, and I cannot therefore recommend this or other alkaline remedies.

When calomel and hydrochloric acid are of no avail, I would advise the use of creosote on account of its decided anti-fermentative action, especially in cases in which emesis is prominent; in those cases, also, in which after the subsidence of acute symptoms, thin, stinking evacuations continue, against which hydrochloric acid has proven useless, this remedy proved itself effectual, as soon as given in sufficiently large doses (P. 4). The following case shows that even large doses need not be feared:

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[1] Köhler: Handb. der physiol. Therapeutik, p. 882. Göttingen, 1876.

Boy, aged seven months, bottle-fed. During the last few days vomiting of milk, partly fluid, partly coagulated, with sour smell; frequent thin, sour-smelling stools, looking like weiss bier. Hydrochloric acid alone, and with addition of laudanum is ineffectual. I then tried creosote gtt. viij., aq. commun. 45.0, syrup simp. 15.0, one teaspoonful every two hours. At the end of two days, cessation of vomiting; the diarrhœa continued and was relieved later by small doses of opium.

In addition to these remedies, I have also tried other drugs which are said to have an anti-fermentative action, viz., chloral hydrate (1.0 and more to 100.0), carbolic acid and aq. chlorica. Only the first did good service, although not constantly, in cases of dyspeptic vomiting; I have entirely discontinued the use of the other two, and do not consider the continued employment of carbolic acid as entirely devoid of danger. Pepsin has also disappointed my expectations, probably because we cannot determine its exact indication in individual cases. This remedy can evidently only be of assistance when the dyspeptic fermentation is caused either by diminished secretion of gastric juice or a diminution in its amount of pepsin. But how can we diagnose this change with any amount of certainty? The use of pepsin, therefore, in infantile dyspepsia always remains an experiment, whose success must be regarded as a lucky accident. I prescribe pepsin either pure or combined with hydrochloric acid (P. 5). As a matter of course pepsin can only prove effective when protein substances, especially milk, continue to be ingested, and it should be given half an hour after feeding.

R. K——, aged ten months, bottle-fed, poorly nourished; presented December 7, 1866. For a few days insomnia, frequent colic, ten to twelve thin, green evacuations daily, slight meteorism, no vomiting or fever. Calomel unsuccessful. Pepsin (0.06 three or four times a day) produces cure after twelve doses. April 13, 1867, again brought to dispensary on account of vomiting after every act of feeding. This has continued for some weeks; sprue in the mouth. Pepsin, 0.06 four times a day. Marked diminution of vomiting on April 16th, complete recovery by April 23d.

Under certain circumstances, therefore, pepsin may prove successful, and we may be compelled to use one tried remedy after another. To those previously mentioned may be added others, which will be discussed later, especially the subnitrate of bismuth. This seems to me to be especially indicated when the presence of mucus in the stools shows the beginning of a catarrhal condition of the intestinal mucous membrane. During the first year of life we may give 0.05–0.2 of this drug, with pulv. gummos., 0.5, five or six times a day, and when the affection lasted for weeks I often saw increased effect from the addition of extr. opii aquos., 0.002 to 0.005. Bismuth also constitutes one of our most reliable remedies at a later period when the symptoms of chronic intestinal catarrh come into the foreground. Nitrate of silver (0.05 : 100) is also of undoubted service in many cases of dyspeptic diarrhœa, and may therefore be tried when the latter proves obstinate. After recovery I would recommend rhubarb as a digestive tonic, which must be given for several weeks in the form of tinct. rhei. vinosa (five to fifteen drops three or four times a day, according to age).

V. Coryza of Infants.

The great sensitiveness of the nasal mucous membrane is manifested in the new-born, who soon react to the contact of atmospheric air by frequent, reflex sneezing. Every cold affecting the new-born, especially in

incautious washing or bathing, readily produces coryza with snuffling breathing and sero-mucous discharge, which dries into yellowish brown crusts if the nostrils are not kept carefully clean. This tendency to coryza continues during the first year of life. In all such cases there may be a suspicion of hereditary syphilis, especially as coryza may constitute the first symptom of syphilis, and precede all others for weeks. In protracted coryza, therefore, you should examine the child and parents in this respect.

Although syphilitic coryza may lead to the same dangers as the ordinary form, this occurs but rarely. But in simple coryza we much more often see symptoms develop which may prove dangerous in several respects. The inflammation may rapidly spread, at this age, to the mucous membrane of the larynx, trachea and even the bronchi. Hoarseness of the cry, cough, fever and dyspnœa not infrequently develop within a few days, and examination shows more or less diffuse bronchitis and bronchopneumonia. On the other hand, the swelling of the nasal mucous membrane, which considerably stenoses the already narrow nasal cavity, may cause more or less severe dyspnœa, which gives a disquieting appearance to every tracheal and bronchial catarrh when combined with coryza, though auscultation and percussion do not justify our fears. But sudden attacks of dyspnœa may also occur in cases of pure, uncomplicated coryza. Bouchut describes asphyctic symptoms which are said to occur from the fact that the child, finding it impossible to breathe through the nares, breathes through the mouth with such force that the tongue is suddenly drawn backward by aspiration and the under surface of its tip is pressed against the hard palate, thus preventing the entrance of air into the pharyngeal space. I have only observed aspiration of the tongue once, not in a case of coryza, but in spasm of the glottis; only with difficulty could I reach, with the forefinger, to the root of the tongue, which was rolled over and pressed firmly against the hard palate, and thus draw it forcibly forward. I have always been compelled, however, to attribute the dyspnœa in infantile coryza to obstruction of the nares, and have seen it obtain such intensity as might lead it to be mistaken for croup.

In March, 1861, I was called to a child, aged seven weeks, in whom severe spells of suffocation had appeared for an hour and a half. According to the parents, the child had been perfectly well a few hours before and had been taken out in a strong east wind; the attacks had occurred without any provocation almost immediately after returning. As the severe symptoms had ceased on my arrival, I thought of attacks of spasm of the glottis, and, in order to study it, had the child placed at the breast. A bad attack occurred immediately, almost as severe as in croup. With an expression of extreme terror in the cyanotic face, open mouth and violent action of all the inspiratory muscles, the child gasped for breath, giving rise to a whistling noise, which evidently had its origin in the nose. After a few minutes, gradual abatement, then sleep, during which inspiration and expiration were accompanied by snuffling; lower part of nose somewhat swollen. During the next twelve hours I only allowed nourishment to be taken from the spoon, ordered frequent inunction with warm oil over the nose, and calomel 0.015 every two hours. On the next day a mucopurulent discharge occurred from the nose, and disappeared after a few days.

In the rare cases of this kind, the sudden development of the swelling of the mucous membrane is specially noteworthy, analogous to that occurring in adults in severe coryza, particularly during the prone position. Here also the secretion ceases with the increased swelling, and, as a rule, relief is only obtained in the sitting position, as occurred in the little patient just referred to. In my opinion there is a decided analogy between these cases of acute coryza, so-called pseudo-croup, and certain

very acute attacks of bronchial catarrh, to which I shall recur later. A second danger consists in the interference with nursing, because during this act the child is forced to breathe through the nose, and as this is not possible, must often let loose the nipple in order to breathe through the mouth.

Coryza almost always affects both nares and is rarely confined to one side. I observed one child, aged eight weeks, who had been previously perfectly healthy, but had suffered for two weeks from a yellowish serous discharge from the right nares, while the left was entirely intact. Snuffling, breathing and dyspnœa were present during nursing. Pencilling the right nares with a solution of nitrate of silver produced recovery within two weeks.

The above-mentioned examples contain everything that I have to say concerning the treatment of coryza. Above all the nutrition of the child demands your care. If nursing is interfered with by dyspnœa, the milk drawn from the breast or cow's milk should be given in a spoon. Kussmaul's case, in which a child, aged six months, was nourished for a week through the œsophageal sound on account of the above-mentioned aspiration of the tongue, is probably unique. When the development is very acute, I would recommend the internal administration of calomel, 0.01–0.015 every two hours. In milder cases you need simply keep the lumen of the nostrils free by brushing them with oil and removing crusts. In more chronic cases, pencilling the nares with a solution of nitrate of silver (1 : 50) is usually successful.

VI. Retropharyngeal Abscess.

That this affection, despite its description by many physicians, is almost entirely unknown, is due to its rarity; notwithstanding the large material at my command, I have only seen from thirty to forty cases. Only a physician who has been fortunate enough to carefully observe a case of retropharyngeal abscess, is safe from a diagnostic error, as the symptomatology will be indelibly impressed upon him.

The affection consists of an almost always insidious abscess-formation in the connective tissue between the cervical vertebræ and pharynx, with the final formation of a tumor, which, extending more or less into the pharynx, may produce considerable disturbances of deglutition, and finally, also, of respiration.

I observed my first case in 1850,[1] and owe my diagnosis to having read, a few days previously, the histories of two cases of this kind, reported by Fleming, in the *Dublin Med. Journal*, February, 1850. Almost all my cases affected children who were in the first year of life or very little older: the youngest child was only four months old. In two cases, the children were two and three and one-half years old. The disease is extremely obscure at the outset; inclination to cry, restlessness, frequent refusal to nurse are the first symptoms. We may indeed suspect that pains are present from the beginning in swallowing, but this dysphagia is a symptom which cannot be determined, in the beginning, in such small children. Painful distortion of the features while drinking may arouse suspicion, but it is not infrequently absent even after the full development of the tumor. The first really suspicious sign is a snoring

[1] Casper's Wochenschr., June 22, 1850.

sound in respiration, especially during sleep. Inspection of the pharynx, which every conscientious physician should perform under such circumstances, usually reveals nothing, or at most a thickening and redness of the pharyngeal mucous membrane, which is coated with mucus. As a rule, one and one-half to two weeks or more elapse before the abscess, by its increase in size, interferes seriously with respiration. A new series of symptoms then appear, which may be mistaken by the physician for severe laryngeal catarrh or perhaps even croup. Respiration is labored, the inspiratory accessory muscles act vigorously, and inspiration and expiration are accompanied by a snoring sound. On attempting to drink, slight attacks of suffocation occur, and the fluid is often regurgitated through the nose and mouth. In severe grades, the face may be slightly cyanotic. Contrary to my former experience, I have now learned that cases may occur in which hoarseness and cough are present on account of an attendant laryngeal catarrh. In many cases of retropharyngeal abscess a diffuse swelling is observed externally on one or both sides of the upper region of the neck, and several swollen lymphatic glands are present, which appear to have been pressed outward. The external jugular veins are often very turgid. But all these symptoms are not characteristic; a positive diagnosis depends entirely upon a local examination of the pharynx with the finger. In children who already have teeth, it is best to protect the finger by a leaden ring. In very severe dyspnœa the local examination may produce not only symptoms of asphyxia, but also convulsions, as Fleming has observed. But I have always been able to feel the abscess distinctly as a tumor projecting into the pharynx from the vertebræ, either above or further down at the level of the epiglottis or even deeper. The tumor is usually hemispherical, more rarely oval, distinctly fluctuating, about as large as a walnut, and is situated either in the median line or more to one side. If it has once been felt, the diagnosis is positive, and the treatment is defined at the same time. As soon as fluctuation is distinctly felt, I would urgently advise you not to delay with incision of the tumor, since you are not sure that the tumor may not open spontaneously and its contents partly enter the larynx by aspiration. I know of a case in which a colleague, who wished to keep the patient in question until the next day for clinical demonstration, paid for the delay by the sudden death of the child during the night from suffocation.

There is therefore only one remedy viz., rapid incision. In all of my cases, I have done this with a straight or, when the abscess was situated deep, curved bistoury or tenotome which was surrounded with paper or adhesive plaster nearly to the tip. The child's tongue is depressed with the left index finger so that its tip touches and distinctly feels the tumor, the head being firmly held by an assistant. The finger is used as a director, the knife carefully carried along to its tip, *i.e.*, to the tumor, and then boldly plunged into the latter, whereupon the pharynx is immediately filled with yellow pus ; the little wound is enlarged in withdrawing the knife. In order to facilitate the discharge of pus, the head of the child should be immediately inclined forward. After the incision all is usually at an end and complete euphonia is at once presented. I have almost always seen the dyspnœa disappear as if by magic; the external swelling rapidly subsided, and after a few minutes the child looks comfortable and takes the breast.

However, affairs do not always pursue this course. In a number of cases, much greater difficulties were presented, chiefly on account of the

deep situation of the abscess; I could then with difficulty reach the abscess with the tip of the index finger and bend the curved bistoury downward. This is especially true of very young children with very narrow buccal and pharyngeal cavities, in whom severe attacks of suffocation were produced by the repeated movements of the finger over the larynx. Respiration then ceases, the children become blue, distort the eyes, the pulse becomes irregular and small, and nothing remains but to withdraw the finger. But I never hesitated to renew the attempt, and was always successful except in one case, in which the abscess was situated so deep behind the lowest part of the pharynx that I despaired of success at the start. In operating for deep-seated retropharyngeal and retroœsophageal abscess, a concealed pharyngotome is advisable, though I have never used one hitherto. I have also repeatedly observed that a single incision proved insufficient, the abscess having again filled up by the next day, on account of the small size of the opening, the symptoms again reappeared, and a second operation had to be performed. Only in one case was I compelled to open the abscess three times, but will remark that I employed my finger-nail in the second operation, a method which I cannot recommend. After the incision I would advise repeated injections of lukewarm water into the nasal and pharyngeal cavities, in order to clear out the blood and pus as much as possible. The danger of aspiration during the operation cannot be excluded, but this has never happened in any of my cases; nor have I seen any evil results on account of the entrance of milk into the wound.

If the operation is not performed in time, spontaneous rupture may occur during sleep, with aspiration of pus into the air-passages, and fatal suffocation. Or the pus may pass down behind the pharynx or œsophagus even into the mediastinum, and death occurs finally from exhaustion. In the following case the suppuration extended to the outer parts of the neck:

A thin, feeble child, aged ten months, brought to the dispensary on April 2, 1875. Had difficulty in swallowing for the past two weeks; snoring, rattling respiration, copious secretion of mucus in the pharynx, and diffuse swelling of both submaxillary regions, in which a couple of lymphatic glands were felt as large as a walnut; temporal veins unusually distended. At the level of the epiglottis the finger came in contact with a fluctuating tumor as large as a walnut, which projected into the pharynx from behind, and which I immediately incised. Profuse discharge of pus. Decided improvement of symptoms in the next few days, but the discharge of pus continued, and external swelling but little diminished. April 9th I felt a large fluctuating swelling on both sides of the lateral and upper part of the neck; the left one was opened at once, the right on April 11th. An immense amount of pus was discharged, but suppuration continued and collapse occurred. Death on April 19th. Autopsy showed a large deposit of pus extending behind the pharynx down to the œsophagus and into the submaxillary regions on both sides, where it had been opened externally. In addition, small amount of broncho-pneumonia, hyperplasia of the mesenteric glands, small tubercles in the liver. Vertebral column normal.

Only in one case did I observe rupture of the abscess into the pharynx. When this happens, the diagnosis may be rendered impossible as the pus is swallowed by the child and therefore does not appear externally, nor does any tumor necessarily form in the pharynx.

Even rarer than retropharyngeal abscesses, in my experience, are those which form on the lateral wall of the pharynx, between it and the soft parts of the neck, and form a fluctuating tumor on the right or left lateral wall behind and below the tonsils. In one of these cases the abscess ruptured into the external auditory canal.

With rare exceptions all of my cases were idiopathic, *i.e.*, they occurred in healthy children independently of any other disease. A few of the children were, at the most, somewhat emaciated, but presented no abscesses in any other part of the body. There was no affection of the vertebræ or any constitutional disease, and the etiology is therefore involved in complete obscurity. The assumption of Bokai and others that the inflammation and suppuration of the retropharyngeal connective tissue starts primarily in the lymphatic glands situated in front of the vertebræ, is not by any means positively determined.

I have only once observed the development of the abscess-formation from spondylitis of the cervical vertebræ, in a child aged one and one-half years, who had suffered, since December, 1874, from difficulty and pain in moving the head, and manifested a strikingly stiff position. April 5, 1875, I found these symptoms intensified, and also difficulty in swallowing, labored and snoring respiration during sleep, and an abscess, as large as a walnut, situated low down on the posterior wall of the pharynx. This was incised on the same day and a considerable amount of pus evacuated. The diagnosis of vertebral caries was afterward confirmed by the appearance of congestive abscesses on the back and neck, by paralysis of the arms and paresis of the lower limbs. Finally I will mention that I observed, in a case of scarlatina, the formation of an abscess in the submaxillary connective tissue which finally ruptured into the pharynx, but also required an incision externally.

VII. Dentition and its Symptoms.

Although the appearance of the teeth indicates in general the termination of the age of infancy, and Nature herself indicates that sole nourishment with fluids may give place to a more consistent diet, it does not by any means signify any necessity for a change of diet. As a rule, the first teeth appear between the seventh and ninth months, but it is customary to continue nursing at the breast at least until the end of the ninth month, usually somewhat later, even if the children have all the incisors. The mother may, however, suffer from the child biting the nipple, and in one case, observed by me, the sudden outcry of the mother, who had been bitten by the child, caused the child to fall into convulsions.

Every physician knows that the most varied disorders of infants are attributed to teething, and are therefore neglected or even regarded as salutary. In the opinion of the majority of physicians, however, teething is a physiological process which cannot give rise to any morbid phenomena. But it is questionable whether this decided negation is always justifiable, and, although fully recognizing the services thus rendered in restricting "teething" diseases, I cannot suppress certain doubts regarding the universality of this view. We know that the perforation of the teeth is due to the fact that the growing root of the tooth gradually presses the crown forward and pushes through the alveolus after perforation of the overlying gum, which grows continually thinner from the increasing pressure. Is it inconceivable that this slow process may have an irritating effect upon the dental branches of the trigeminal nerve and give rise to reflex symptoms not only in motor but also in vaso-motor nerves? I think this question may be answered in the affirmative. I will, at a later period, mention cases in which, for example, partial spasms of the throat and neck muscles were undoubtedly connected with the perforation of a

group of teeth. The undeniable fact, also, that obstinate vomiting, diarrhœa, or even a spasmodic cough, which have resisted all treatment, suddenly disappear as soon as one or a few teeth have passed out of the alveolus, can only be explained by reflex stimulation of peristalsis or of the vagus, starting from the dental branches of the fifth nerve.

Examples of premature teething are not lacking, and I have repeatedly observed cases in which one or two incisors had appeared at the end of the third month, or a little later. More frequently the process is retarded, and not infrequently the first tooth does not come through, even in perfectly healthy children, until the tenth or eleventh month.

Another anomaly, which was regarded in certain historical characters as the precursor of an energetic, violent character, is also probably known to you: I refer to the congenital appearance of the teeth. According to my experience, two forms may be differentiated. In the first we find one or two sharp, more or less hook-shaped teeth, which are simply embedded in a fold of the gum, and are loose and readily movable. In such cases the rudiment of the tooth has probably not only been prematurely developed, but has also an abnormally superficial position, so that the crown has reached the outside before the root has developed. I have always removed these teeth at once with a pair of forceps, because they usually injure the tongue or the nipple of the nurse. In the second variety I found teeth firmly embedded in the alveolus, but which could be distinguished from the normal and later teeth by the rough surface and yellow color, that is, by an absence of the enamel. These teeth require greater force for their removal, and I therefore advise you to let them alone as long as they are not loosened. As soon as this has occurred, it is better to remove them, as I have always observed, in such cases, a diseased process of the alveolus, which can heal only after the removal of the tooth. The following cases are examples of this form:

Girl, aged three months, presented April 2, 1875. A tooth had been present in the left upper jaw, and was extracted three days later; soon after, swelling of the left cheek. Examination showed considerable swelling of the left upper jaw, fistulous openings at border of alveolus, with discharge of pus; purulent discharge from left nares and from fistula under edge of orbital cavity. Fluctuating abscess in region of left malar bone. April 20th, separation of several splinters of bone from edge of alveolus, and later a larger sequestrum was removed. Further course unknown.

Girl, aged five months. After forcible extraction of a tooth, which was present at birth, from the left upper jaw, a painful swelling of left cheek developed. The upper jaw was found thickened and tender, fistulous openings at left alveolar border and discharge of pus from left nares.

Boy, aged two months, presented January 4, 1878. Left half of upper jaw swollen, tender on pressure, gums red and swollen. Pressure under the jaw caused discharge of pus into mouth. A small opening in region of first molar tooth found to be the source of the pus, and a sound introduced met with a hard object. The history showed that the first left incisor appeared at age of six weeks, accompanied with swelling and suppuration. January 15th the first molar also emerged from the opening mentioned above. Both teeth were quite loose.

This case appears to me to shed light upon the entire process, inasmuch as it is rendered probable that a periostitis of the alveolar border, whether in the upper or lower jaw, pushes the crown of the tooth outward by swelling and exudation within the alveolus. I regard the periostitis as primary, and not, as I formerly believed, the result of forcible extraction of the tooth. The cause of this osseous affection I must leave undetermined. The extraction of these teeth is therefore not alone devoid of danger, but is necessary in order to relieve the alveolus of an

irritating foreign body. A case of periostitis of the orbital cavity in a child two weeks old, observed by Samelsohn,[1] also favors this view. "The cause of the disease, which was accompanied by enormous protrusion of the eyeball, was the premature appearance of the first molar, after whose extraction the affection ran a favorable course." I believe that the premature tooth was not the cause of the process, but rather that a periostitis of the upper jaw, due to unknown causes, pushed the tooth out prematurely.

The process of dentition at the normal period may also be accompanied by abnormal local symptoms, which must be regarded as due to irritation by the teeth. General redness of the buccal mucous membrane is very often noticed, especially in the gums, which are covered here and there with small flakes of desquamated epithelium; the saliva is then increased. The gums are sensitive to the touch and bleed readily. The inflammatory symptoms may be restricted to the immediate neighborhood of the teeth, which is dark red and superficially ulcerated, or may be the site of small, recurring abscesses. "Stomatitis aphthosa" occasionally develops on the tongue and other parts of the mucous membrane. The two lower middle incisors are especially apt to injure the tongue, and cause small ulcerations in it while nursing or coughing. But all these local symptoms are comparatively rare, and this very fact appears to me to support the view that disturbances of remote organs, which are also absent in most cases, may develop under certain circumstances, especially in very nervous children. Whether this opinion is entertained or not, it is now generally held that every attempt to facilitate the eruption of the teeth, and thus remove the symptoms due to "difficult" dentition, is absolutely useless. I have, in earlier years, performed scarification with sufficient frequency to convince myself of its entire inutility, and it even appears to me that the cicatrix formed may increase the difficulties connected with the penetration of the tooth. As I have stated above, the lower middle incisors appear between the seventh and ninth months, rarely earlier, and are followed in a few weeks by the upper middle incisors. Then the lateral upper and lower incisors make their appearance, usually by the end of the first year. Not very infrequently the upper incisors appear before the lower. The four anterior molars appear between the fifteenth and eighteenth months; in rare cases they develop before the eruption of the lateral incisors. Between the eighteenth and twentieth months the four eye-teeth appear, and the four posterior molars between the twentieth and twenty-fourth months. But this is only true of healthy children. A bad constitution, especially if rachitic, retards teething, so that the first incisors appear only at the end of the first year, and the entire process may extend far into the third year. Occasionally a double formation of teeth occurs; in one of my cases, instead of a single canine tooth, there were two—an anterior almost normal, and a posterior smaller and sharper one.

I herewith close the pathology of early infancy, and will now consider the diseases of childhood in general.

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[1] Centralzeitung f. Kinderheilk., I., p. 190. 1878.

LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

PART III.

DISEASES OF THE NERVOUS SYSTEM.

I. Convulsions of Childhood.

The predisposition of the nervous system of the child to disease does not affect all parts alike. Thus, the spinal cord is much more rarely affected than the brain. The neuroses of sensation are much rarer than those of motion, especially convulsions, which constitute one of the most frequent affections from birth to the end of the third year. Soltmann attributes the great tendency to reflex phenomena in young animals until the tenth day to the absence of reflex inhibitory centres in the brain and spinal cord,[1] but this will not explain the similar tendency in children two or three years old. We must, therefore, rely rather on our own observation, which daily confirms the great liability of children to reflex spasms.

The symptomatology of convulsions or infantile eclampsia is not materially different from that of an epileptic attack. It generally begins with rolling of the eyes upward or to the sides, fixed gaze, and loss of consciousness. Twitching of the facial muscles, occasionally on one side only, then occurs, the jaws are closed by trismus, or drawn across one another laterally by spasm of the pterygoids, giving rise to gnashing of the teeth. Tetanic stiffness of the limbs, interrupted by short twitchings, is rarely absent. The fingers are usually strongly flexed, and are extended with difficulty; the feet are in dorsal flexion or in pes equinus, according as the extensors or flexors are more affected. The trunk-muscles also take part; retroversion or to and fro movements of the head, contraction of the respiratory muscles with pauses in the respiration, alternating with rapid, superficial respiratory movements, hardness of the abdominal muscles, involuntary discharge of urine and fæces, are very frequent though not constant. In a few seconds the face becomes slightly cyanotic around the nose and mouth, and the saliva is forced from the mouth in the form of soap-like foam, not infrequently mixed with blood from the biting of the tongue. These phenomena continue for a few minutes; the twitchings then diminish, the face becomes more quiet and acquires a better color, and only slight twitchings occur now and then. But the attack is often renewed before the child recovers from the coma, and may recur three or four times, unconsciousness persisting in the inter-

[1] Compare, on the other hand, the experiments of Tarchanoff: Centralbl. f. Kinderheilk., II., p. 183. 1879.

vals. Reflex action is occasionally preserved, but in most cases it is entirely absent. The duration of the paroxysm is of great importance. The convulsions, interrupted by short intervals of coma, may continue for hours, and the interference with respiration, the venous stasis in the brain, and the exhaustion of the vital energies, may finally cause death. But recovery may occur although the convulsions have lasted many hours or even for days.

The common form of convulsions, lasting a few minutes, has usually subsided when the physician arrives. The child is then usually found in a comatose condition, which passes imperceptibly into a sound sleep that may last for hours, and from which the child awakes as if nothing had happened. But we should always be on our guard, as the attacks may recur sooner or later.

If you are called to a case of this kind and find the child in convulsions, no time is left to enter carefully into the cause of the attack, and you must immediately begin treatment. The causal indication must give place to the vital, and I know of no remedy which fulfils the latter so certainly as the inhalation of chloroform. If an attack lasts more than five minutes, do not dally with other drugs, but immediately use chloroform. A drachm of chloroform poured on a handkerchief and held in front of the child's nose, so that a stratum of air intervenes, often proves sufficient. After a few respirations the spasmodic irritation subsides and the inhalations may be continued until the complete cessation of the convulsions. As a matter of course, the pulse and respiration must be carefully watched during this time. I have never observed any unpleasant effects, although I have given it in numerous cases, and, in one instance, in a child five months old who had forty attacks in one day. Chloroform was administered at the beginning of each attack; a few inspirations sufficed to relieve the spasms, and on the next day the child was apparently well. I even intrust the attendants with its administration, and have hitherto had no reason to regret my confidence. I do not consider cyanosis of the face, in consequence of the convulsions, a contraindication to its use; it always disappeared as soon as the remedy began to act. Nor has the existence of broncho-pneumonia, during the course of which convulsions developed, deterred me from the employment of chloroform; the spasms soon ceased, while the pulmonary affection pursued its course. However, chloroform is not an absolutely certain remedy against eclampsia. In a few very severe cases I found it practically useless; the cessation of the spasms caused by the inhalations scarcely lasted a few minutes, and the case finally ended fatally from exhaustion. The remedy should not be employed if the child is already in a condition of collapse, with a very small and rapid pulse and cool extremities. But such cases always constitute a very small minority. Compression of the carotids, which I have repeatedly tried,[1] gives such uncertain results that it cannot be taken into serious consideration.

As soon as the convulsions have ceased, the cause of the disease must be taken into consideration. I cannot enter fully into the pathogenesis of epileptiform attacks, but I may remind you that it has been proven experimentally that the attacks may develop in three ways, viz.: by anæmia of the brain from contraction of the smallest cerebral arteries (Kussmaul and Tenner), by unilateral division of the spinal cord or sciatic nerve with subsequent irritation of the corresponding side of the face (Brown-

[1] Beiträge zur Kinderheilk., N. F., p. 97. Berlin, 1878.

Séquard), and by blows upon the head, which give rise to small hemorrhages in the medulla oblongata (Westphal). In my opinion, only the first and third series of experiments can be considered in the pathogenesis of infantile convulsions. Isolated examples are not lacking in which a severe blow on the head has given rise to epileptiform attacks, which have even recurred habitually. On the other hand, anæmia of the brain, in consequence of weakness of the heart in exhaustive diseases or spastic contraction of the small arteries, may be accepted when convulsions occur from reflex irritation or in an attack of fever. But this does not exhaust the pathogenesis of eclampsia. I would remind you that increased tension and even pulsation of the fontanelle may be observed during an attack, symptoms which indicate congestion rather than anæmia of the brain.

But we will now leave this hypothetical domain and turn to the etiological factors determined by the experience of the physician. In the first place, it is important to know whether the convulsions are due to a material lesion of the brain. A unilateral character of the convulsions favors their cerebral origin, if the spasms, upon their recurrence, remain restricted to the same side of the body. But it should not be forgotten that bilateral convulsions may occur with a unilateral lesion of the brain; and, on the other hand, that unilateral spasms are occasionally observed, although no cerebral affection is present. Nevertheless, the presence of unilateral convulsions should always induce us to make a careful examination with regard to the existence of cerebral disease. Many brain diseases—for example, tubercles and tumors—may present for months occasional attacks of eclampsia as the sole symptom.

Attention should next be paid to the osseous system of the child. In my experience the tendency to convulsions is furthered by no other cause to such an extent as by rachitis. Attacks of spasm of the glottis are almost always present in such cases, and may either open the series of spasms or alternate with them; the spasm of the glottis is rarely entirely absent. The cause of this predisposition of rachitic children to spasms is unknown; but, in these cases, we should always expect a repetition of the attacks, though the exciting cause can be discovered in very few instances.

In my opinion rickets plays a much more important etiological part than dentition. Convulsions are rarely observed in teething children who are not rachitic, unless definite reflex causes are demonstrable. The latter may indeed include, as I have remarked above (page 62), the eruption of a tooth under especially unfavorable circumstances; but these cases are extremely rare, and you should always examine for other exciting causes. Among these, irritative conditions of the digestive organs undoubtedly occupy the first place. Convulsive attacks are not infrequent in the dyspeptic conditions of the new-born and infants, and those cases in which eclampsia of the infant occurs soon after emotional excitement of the mother, also belong to this category, as they can only be explained by an injurious influence of the milk on the digestive organs of the child. But overloading of the stomach or intestinal canal may cause severe convulsions in later childhood, until toward the end of second dentition. The following cases will serve as illustrations:

Child, aged three and one-half years. At noon ate heartily of cucumber-salad and plums. Eclampsia in the evening, continu ng about two hours with intervals of coma. Cold fomentations to the head, enemata, emetic after cessation of stupor. Recovery.

Child, aged two years. March 17th, ate heartily of sauerkraut; considerable me-

teorism and unusual drowsiness. Same condition on morning of March 18th; sudden nausea, vomiting, and severe eclampsia at 11 o'clock, continuing until 2 P.M. A few scybala removed by two enemata. At 2.30 o'clock, I found the child unconscious, eyelids forcibly closed, respiration stertorous and irregular, occasional twitchings in the limbs; pulse 120 and very full. Treatment: sinapism to back of neck, cold fomentations to head, four leeches behind the ears, calomel 0.06 every two hours. Six o'clock: consciousness restored, child wanted to eat, slept quietly for past half-hour. Inf. sennæ comp., 50.0. March 19th, free evacuations from bowels; child entirely well.

These cases also indicate the plan of treatment, viz., by emetics and purgatives, ol. ricini, inf. sennæ comp., etc. (P. 6 and 7), which rapidly remove the materia peccans from the stomach and intestines. When the abdomen is markedly distended, it is advisable to give an enema, even during the spasm, of milk and honey (2 to 1), or of cool water, in order to empty the bowels as soon as possible. As a rule, bleeding is not to be recommended. I employed it in the last-mentioned case on account of the long duration of the convulsions (three hours), in which considerable venous stasis of the brain and meninges could be presupposed; I would recommend this plan in analogous cases.

I will not deny the possibility of convulsions being caused by the presence of worms in the digestive canal, but I myself have never observed a case in which they could with certainty be attributed to this cause. I will acknowledge, however, that the use of anthelmintics is advisable, when worms are known to have been present, or may, at least, be suspected.

A febrile condition, which precedes and follows the convulsions, is significant in determining the etiology. Fever may also be present in the cases of dyspeptic convulsions just described; but you should never, under these circumstances, neglect the examination of other organs, acute affections of which begin not infrequently, in childhood, with fever and severe convulsions. In the first rank I may mention primary pneumonia, then pleurisy and enteritis. I may here remark that the diagnosis of a pneumonia beginning in this manner may be impossible at first, because examination of the chest reveals nothing abnormal at this early stage, and it may be doubtful for a few days whether we do not have to deal with an acute inflammatory affection of the brain. The cause of the convulsions in these cases is not very clear. We might attribute them either to reflex irritation starting in the lungs, pleura or intestines, or to the high fever, which is sufficient to produce convulsions in irritable children. I saw repeated eclamptic attacks occur on the first day of simple angina tonsillaris attended with high fever, in two children aged respectively six and eight years; with the subsidence of the fever on the next day, the convulsions ceased. The convulsions which occasionally, though in my experience rarely, occur in the initial stage of acute infectious diseases (measles, small-pox, scarlatina) probably belong in this febrile category, though it is conceivable that the infectious material in the blood may aid in producing them. In all such cases the convulsions can only be treated symptomatically by the application of ice-bags to the head, cool baths at 22 to 20° R., enemata and mild purgatives.

Uræmia and intermittent fever are included among the acute diseases which may begin with violent convulsions. As a rule, only the first paroxysm of intermittent fever runs this course, and may appear like simple eclampsia, until the later and ordinary paroxysms clear up the mistake. Much more rarely the first or second paroxysm of this convulsive form of

intermittent fever presents a complete picture of pernicious intermittent, as shown in the following case:[1]

A healthy girl, aged nine years, complained on Friday at 10 A.M. of double vision, and then of cold hands; soon followed by psychical disturbance. The child did not recognize those around her, and, about 10 o'clock, had a convulsive attack. This continued, alternating with coma, for about an hour, and was followed by sleep, after which the patient appeared well, except slight headache. As no other cause could be found, I thought of intermittent fever, as the family lived near the canal, where malarial diseases were not infrequent. Nothing noticeable on the next day; on the afternoon of the day after, at 4 o'clock, the attack was renewed. The child suddenly failed to recognize those around, the hands were cold, and she complained of dizziness and diplopia in the lucid intervals. At 5 o'clock a severe epileptiform attack began and continued with undiminished severity until 6 o'clock, when I found her cyanotic, pulse small and frequent; I gave a hypodermic injection of morph. acet. 0.01, and soon afterward administered chloroform. A few whiffs sufficed to stop the convulsions, and a quiet sleep, lasting ten hours, ensued, from which the child awoke well. I immediately ordered sulphate of quinia 0.3 every three hours (1.5 on the first day), on the second day 0.18 every two hours, and 0.12 on the following day, so that about 6.0 quinine were given in the first week. No further attack occurred; at noon of the following Tuesday the child suffered from headache and vertigo, and began to tremble; this condition only lasted about twenty minutes. Since then the patient has remained perfectly well.

Psychical causes may also give rise to convulsions, especially sudden fright, more rarely fear. The attacks produced in this manner sometimes recur at a later period, and these cases always give rise to the fear that the affection may develop into epilepsy, as experience teaches that the latter very often begins in childhood. The diagnostic criteria of confirmed epilepsy, viz., diminished mental energy, loss of memory, change of character, are absent in the beginning of the affection in children (except in congenital atrophy of the brain accompanied by epileptic attacks), and can therefore not be utilized in the differentiation of transitory eclampsia from beginning epilepsy. Among the cases of true epilepsy which I found develop in childhood, the following are worthy of mention:

A girl, aged thirteen, had an eclamptic attack during infancy, which recurred in the third and twelfth years. She learned to speak when five years old. Since the age of seven, she has peculiar spasms, consisting of a feeling of strangulation in the larynx, jerky, rapid expirations, with fixed gaze and slight confusion in the head. The attack ends, after a few seconds, with palpitation. Intelligence and memory feeble; frequent pain in neck; no menstrual molimina. The attacks are said to have ceased, for a time, after profuse epistaxis. Soon after complete epileptic paroxysms occurred, preceded by an aura consisting of vomiting and the spasms in the neck. The latter, which had existed for six years, must therefore be regarded as aborted epileptic attacks.

A girl, aged twelve, had suffered from epilepsy for five years. The aura consisted of tinnitus aurium, which roused her from sleep. Only nocturnal attacks.

In a boy, aged fourteen, who had been epileptic for a number of years the aura consisted of nictitation of both lids and nodding movements of the head. This aura had existed as an independent disease before the development of the epilepsy.

A child, aged three years, whose brother was idiotic, had suffered for some months from epilepsy; the aura consisted in the child standing suddenly still with fixed gaze, apparently blind, running in one direction and then losing consciousness.

I consider it unnecessary to enter fully into a description of epilepsy. The cases described especially show the various kinds of aura, which may exist for years as an apparently independent affection. I would therefore advise you in all cases in which nervous symptoms of this kind are present in otherwise healthy children, not to regard the matter too

[1] Berl. Klin. Wochenschr. No. 26. 1873.

lightly, but to keep the prodromata of epilepsy in mind. In one portion of my cases I also observed delirium, not alone after the attacks, but also in the intervals, occasionally the so-called "somnambulistic" phenomena, such as rising from bed at night, crouching under a table, climbing on furniture, while in a half-sleep, consciousness being entirely or partially lost, irrepressible tendency to run around the room or sing aloud. Occasionally the delirium attains such a high grade, that it may be termed "ecstasy." These phenomena are not peculiar to epilepsy of childhood, but are also observed in adults.

I will finally add a few remarks on treatment, as those made previously (page 68) referred only to those cases in which a causal indication was present. Unfortunately there are many cases in which a definite cause cannot be found, especially when the convulsions occur in rachitic children, with or without spasm of the glottis. In my opinion the chief element here is the treatment of the rachitis, but the convulsions may recur so frequently that they constitute the chief ailment, at least for the time-being, and demand therapeutic consideration. I must confess, however, that our art can boast of no very great success under these circumstances. A great many useless drugs have been employed for a long time; bromide of potassium and hydrate of chloral, which have been extensively used in recent times, appear to possess greater importance. I am far from attributing to them any specific action, and I am not wanting in cases in which they were of little or no avail; but they are always worth a trial. I prescribe bromide of potassium, 0.3–1.0 t. i. d., according to the age of the child (P. 8); chloral hydrate, 1.0–2.0 to 100.0 internally, or 0.2–0.5 at a dose in enema (P. 9). Even during the period of childhood, these doses possess no hypnotic effect. When there is great restlessness, insomnia, and rapidly recurring convulsions, it may be necessary to give chloral in full doses (1.0), or even morphine.

II. Spasm of the Glottis.

Spasm of the glottis is the most frequent of those spasmodic affections of childhood which are confined to a circumscribed nervous distribution, but exhibit a tendency to become general at any moment. It is more frequent in boys than in girls, and is confined almost exclusively to the period between the sixth and twenty-fourth months. I have hardly ever observed it at a later age, but have seen cases as early as the fifth or sixth week. The disease is popularly known as "inward spasms."

In fact many characteristics of the affection can be seen in a healthy child who, in the midst of a severe crying spell, suddenly "holds the breath," *i.e.*, lies with the head thrown back, face dark red, somewhat cyanotic, respiration interrupted, and rigid limbs. The excessive crying and the excitement appear to cause spasm of certain respiratory muscles, which disappears, as a rule, after a few seconds. Under abnormal conditions, the attacks may occur during complete quiet, and even on awaking from sleep; but even then it is favored by every respiratory exertion, viz., crying, drinking, eating, as well as by psychical influences, anger and fright.

The simplest form of the attack consists of a momentary cessation of respiration, followed by a few "crowing" or whistling inspirations. There are numerous gradations between this and the most severe forms. The

sudden cessation of respiration is common to all; the child usually throws himself backward, the face is pale and somewhat bluish around the mouth and wings of the nose, the arms and legs are often extended, the fingers bent into the palm, and the toes occasionally flexed. The return of respiration is shown by labored, at first feebly, then more loudly, whistling respirations, with which the attack terminates after a duration of two or more seconds. The "crowing" is due to the passage of air through the still narrowed glottis; so long as the spasm remains at its height, no respiration occurs.

Those cases are most to be dreaded in which the apnœa continues beyond the usual period, and no whistling tone is heard; the complete closure of the glottis may then prove rapidly fatal from asphyxia. A child may suffer for weeks from mild attacks which scarcely arouse any apprehension, when suddenly an attack occurs resulting in immediate death. You must therefore be on your guard in practice, and should inform the family, even in mild cases, of the possibility of a fatal issue.

The further extension of the convulsive affection must not be overlooked. Although confined, in the milder forms, to the arytenoid muscles, *i.e.*, to the distribution of the recurrent laryngeal nerve, the spastic irritation is often propagated to other parts of the respiratory system (thoracic muscles, diaphragm), giving rise to peculiar irregularities in the respiratory rhythm—for example, rapidly following inspirations without noticeable expiration. The ocular nerves may also take part, and the frequently observed contractions of the muscles of the fingers and toes testify to the spread of the irritation to other paths. I have even observed, during a few attacks, trismus-like contractions of the masseter and temporal muscles, and loss of sensibility and consciousness are then alone wanting to stamp the attack as eclamptic. So far as I have been able to judge, I believe indeed that a very short loss of consciousness occurs during severe spasm of the glottis. This explains the fact that spasm of the glottis very often alternates with eclampsia, or the former opens the scene and soon passes into general convulsions. Occasionally I observed a continuation of the contraction of the fingers and toes during the intervals of the spasms. The combination of spasm of the glottis and eclampsia is so frequent that I always prepare the parents, in every case of spasmus glottidis, for the possibility of the sudden appearance of general convulsions.

There is such a manifest relation between spasmus glottidis and rachitis, that I always examine the cranial bones, epiphyses of the ribs and long bones, and rarely fail to find rachitic changes. Even in children three or four months old, who suffered from spasmus glottidis, I have repeatedly found the cranial sutures open, the surrounding parts soft and compressible, and the epiphyses of the ribs distinctly swollen. According to my experience, at least two-thirds of the cases are rachitic, and this explains the family predisposition to spasm of the glottis, which is occasionally observed. In exceptional instances, the rachitis was confined to the cranial bones, whose ossification was then considerably retarded. When Elsässer wrote his book on "The Soft Occiput," he was misled, by the softness and partial erosion of the cranial bones, into attributing spasmus glottidis to this osseous affection, assuming that when the children were lying down the brain was not sufficiently protected against pressure by the softened bones. I have examined hundreds of cases for "craniotabes," and very rarely found them as described by Elsässer. At any rate they must be regarded as rachitic, and from this standpoint alone can we judge

their connection with spasm of the glottis. The enormous frequency with which these attacks may occur in rachitic children is almost incredible: twenty or even thirty attacks may occur in one day. If this condition continues for weeks and months, with alternating periods of improvement and exacerbation, fatal exhaustion may occur. In other cases, as I have already stated, death may occur suddenly from apnœa, in consequence of closure of the glottis. In these cases, the previously mentioned aspiration and rolling of the tongue against the hard palate has also been blamed as the cause of apnœa. I will not deny that the forcible inspirations at the close of the spasm, may render this process possible. I have myself observed a case of this kind, in which the apnœa was relieved by forcibly pulling the tongue forward. On account of such cases, aspiration of the tongue has been regarded as the cause of the apnœal symptoms in spasm of the glottis, a view which I consider entirely unjustified, as I have often found the tongue, under such conditions, in its normal position.

In a third series of cases, death is due to a severe and prolonged eclamptic attack or its consequences. Post-mortem examination, in a number of cases, showed venous hyperæmia of the pia mater and usually of the brain-substance, and œdema of the pia mater with serous effusion in the ventricles in a few instances. But I regard these appearances as the results of the venous stasis occurring during eclampsia, since they were most marked when the affection was complicated by whooping-cough. This complication was observed not infrequently; this, as a matter of course, is only accidental, and, in my experience, furnishes an unfavorable prognosis.

All attempts fail to explain the connection between rachitis and spasm of the glottis. Poorly nourished children are especially affected, but well-developed ones are not by any means spared. When the predisposition is present, spasmus glottidis occurs spontaneously or from reflex irritations, among which the eruption of the teeth plays a part which cannot be denied. This is also true of abnormalities of digestion, constipation or diarrhœa; the influence of colds and of catarrh of the upper respiratory passages must be regarded as prominent among these causes. This is shown by the predominance of the disease in the cold seasons; I have observed by far the largest proportion of cases from January to April inclusive. I therefore warn mothers against exposing predisposed children against the inhalation of cold air. When catarrh of the larynx and trachea is also present, the crowing inspirations acquire a rough, hoarse sound.

All these causes may also produce spasm of the glottis in non-rachitic children, but these cases are much rarer than when this complication is present. All other views with regard to the etiology of the affection are either hypothetical or entirely false, especially the theory that the disease is due to enlargement of the thymus gland (asthma thymicum).

The prospects with which you enter upon the treatment of spasm of the glottis are not very favorable, though the majority of cases finally recover completely, even after the disease has lasted for months. This favorable result is mainly secured, in my opinion, by improvement of the disordered general condition, *i.e.*, the rachitic disposition, and I generally direct my attention to this point, unless the too frequent recurrence of the attacks necessitates treatment. With reference to the latter, I can only repeat what I have said on page 70 regarding eclampsia. Neither bromide of potassium or hydrate of chloral gave reliable results. Although the success in the beginning is occasionally astonishing, we must

always be prepared for a relapse. I have seen no good effects from the use of zinc. In a few cases, musk appeared to act as a sedative and diminished the frequency of the attacks, in others it was entirely useless. When it becomes necessary to put a stop, as rapidly as possible, to the enormous frequency of the spasms and the consequent exhaustion of the child, I employ morphine without any hesitation (P. 10). As soon as the patient becomes quiet and drowsy, its administration should be stopped in order to prevent toxic effects.

The therapeutic consideration of the reflex irritants occupies the first place, if symptomatic treatment is not immediately necessary; protection from cold air, treatment of any catarrh which may be present, purgatives in constipation, anti-dyspeptic remedies in dyspeptic diarrhœa. Scarification of the gums in dental irritation is absolutely useless. But above all I would recommend treatment of the underlying predisposition by anti-rachitic remedies, viz., pure, warm air, bran and salt baths, iron and cod-liver oil.

III. Idiopathic Contractures.

You will remember that contractions of the fingers and toes are very often observed during spasm of the glottis, and occasionally continues during the intervals. They may also arise independently of spasmus glottidis, and spread to other muscles. They occur under the same conditions as eclampsia, and not infrequently alternate with the latter and with spasm of the glottis. Usually the fingers and toes are flexed, more rarely extended, and occasionally flexion occurs at the wrist, foot or elbow-joints; the contractions appear to be painful.

When they persisted for many hours or even days, I occasionally observed œdema or cyanotic discoloration of the dorsal surfaces of the hand and foot, probably due to pressure of the muscles upon the intermuscular veins. I only observed true ecchymoses in one case. At first, the contractions were paroxysmal, but later they became more or less continuous; complete relaxation occurred during sleep, although Bouchut often noticed the reverse. When the contractures occur unilaterally, it must always arouse a suspicion of a cerebral lesion, especially of cerebral tubercles. Exceptionally, unilateral contractures may develop idiopathically as the result of reflex irritation:

A child, aged eleven months; first tooth appeared three weeks ago, rapidly followed by three others. Examination showed contracture of right lower limb at the knee- and hip-joints, the limb assuming exaggerated position of morbus coxæ. Attempts at extension were painful. According to the mother this contracture developed before the eruption of each tooth and disappeared when this was completed. Condition unchanged at end of three weeks.

The reflex irritation may also be situated in the urinary organs, as in the following example:

Child, aged five months, said to have cried from birth before each act of micturition; first examined on October 10, 1861. Had an attack of eclampsia two weeks ago, followed by another a week later. Since the first attack the toes have remained in plantar flexion, after the second one the fingers and knee-joints were similarly affected; attempts at extension very difficult. Throat and neck muscles rigid with impaired mobility of head. Uric acid concretions have been found in the diapers for past three weeks. Purpura spots in different parts of the skin appeared immediately after the

convulsions. October 17th, after lukewarm bran baths and discharge of three calculi, the contractures diminished considerably, but repeated twitchings occurred in the limbs. Œdema of lower lids, left leg and foot, fresh purpura spots. November 21st, child perfectly well. Treatment consisted of bran baths and small doses of iron.

It is evident from this history that eclamptic attacks and contractures have the same significance, the difference between them being due to the loss of consciousness in the former and its preservation in the latter. I therefore regarded contractures as an abortive form of eclampsia; this opinion is also in accordance with the fact that contractures, like eclampsia, are occasionally intermittent.

This affection is classed with tetany by many authors, but in my opinion it is well to separate the two diseases entirely. I have never been able to detect the symptom regarded by Trousseau as characteristic of tetany, viz., production of the contractures by pressure upon the artery supplying the affected limb, in idiopathic contractures of children.

I will here make a few remarks with regard to the rarest convulsive symptom of childhood, viz., tremor; I have observed it almost solely in paralysis or contracture of the limbs due to cerebral tuberculosis, basilar meningitis and other diseases of the brain. In one case I observed general tremor with a favorable termination:

A child, aged fifteen months, previously healthy, presented February 5, 1879; had had pneumonia a month previously. For two weeks has had continuous tremor of hands, feet and head, which was usually drawn slightly backward. The child cried frequently, and the cry was not continuous but bleating in character. The child has forgotten how to stand, but could hold toys in the trembling hands. Complete euphonia and normal function of all the organs. February 18th, the tremor has diminished, and on the 20th has entirely disappeared. Treatment consisted of administration of hydrate of chloral (1.0 : 120).

It is impossible to give the cause of the tremor in this rare case. The otherwise normal condition of the child led me to attribute the affection to dental irritation, and although no perforation of teeth occurred during the period of observation, I still adhere to this view. It is conceivable that the upward growth of a tooth temporarily presses upon and irritates the alveolar nerves, and produces reflex spasms, but its further growth relieves the nerve from pressure, even before the tooth has come through. This view of the causation of the tremor forced itself upon me on comparing the case with others in which similar, though more marked and restricted, movements resulted from this source of irritation. I refer to

IV. Nodding Spasm, Spasmus Nutans.

My first observations of this disease were published in 1851.[1] About the same time a couple of similar cases were described by Faber and Ebert. Among the cases observed by me since that time, I publish the following:

A child, aged nine months. For several weeks almost continuous nodding movements of the head with slight rotation to the right; complete cessation during sleep. Nystagmus of right eye, the inward movement being most marked. After a few

[1] Romberg and Henoch: Klinische Wahrnehmungen u. Beobachtungen, S. 57. Berlin, 1851.

weeks, subsidence of the nodding movements in consequence of the eruption of a tooth; nystagmus continues.

Child, aged one year. Same symptoms as in previous case, except that converging strabismus of right eye is substituted for the nystagmus. Reappearance of affection after a pause, following the eruption of two teeth. Spontaneous recovery in two weeks.

Child, aged nine months, rachitic, had suffered previously from eclampsia and spasm of the glottis; normal development of teeth (two incisors). Nodding movements not confined to head, but affected the entire upper part of body, occurred in paroxysms several times daily, and were so severe that the head was occasionally bent almost to the knee. Diminution of intensity and frequency of attacks after lapse of two weeks. Further course unknown.

These cases show that the characteristic movements are hardly ever confined to the sterno-cleido-mastoid muscles, but also involve the rotators of the head. In almost all cases they were combined with spasmodic movements of the ocular muscles (usually nystagmus, rarely strabismus or rolling movements), generally in both eyes, more rarely on the side toward which the head was rotated. The movements are usually continuous, more rarely paroxysmal; they cease during sleep. By grasping the head or attracting the attention, the movements can be stopped momentarily, as a rule, but the nystagmus becomes more marked or makes its appearance, if it has not been present previously.

The disappearance of the symptoms, in some cases, after the eruption of a tooth, shows that dentition constituted the reflex irritant. In the other cases, this causation is probable, though not undoubted. The almost constant combination with spastic affections of the ocular muscles is interesting as indicating the close relation of the roots of origin of the spinal accessory and upper spinal nerves, which supply the throat and neck muscles, with those of the motor oculi communis, supplying the ocular muscles.

The reflex form of spasmus nutans just described must be differentiated from a far more dangerous variety, which is undoubtedly due to central disease. Even the first descriptions by English authors (Newnham, Willshire) refer to cases in which disordered intelligence and epilepsy were combined with nodding movements, not only of the head, but also of the entire upper part of the body. The latter either occurred in paroxysms (fifty to one hundred movements per minute) or continuously, but with less severity. The termination was always fatal. I would also include in this category the not infrequent cases in which weak-minded or idiotic children often fall forward, the arms being drawn away from the body at the same time, and the eyeballs slightly distorted. A third variety is usually described under the head of chorea magna, to which I shall refer at a later period.

The treatment of the reflex form is purely expectant.

I may mention incidentally that I have repeatedly observed more or less continuous rocking movements of the upper part of the body in small children as an expression of onanistic irritation, but these movements are voluntary, and should not be mistaken for true spasmus nutans. The other partial spasms of childhood are exactly similar to those of adult life; but I wish to say a few words with regard to a very rare form, which I have not observed in adults, viz., laughing spasms. In the three cases which I observed they were undoubtedly due to intestinal irritation; the first two cases occurred in the children of two sisters. The following is the history of one of these cases:

Child, aged four weeks, moderate diarrhœa for a week, a few days ago sudden spasm of face and trunk muscles, together with loud laughter. The paroxysms lasted about five minutes, and occurred three or four times a day. Infus. ipecac. and tinct. theb. removed the symptoms in a week.

V. St. Vitus' Dance, Chorea Minor.

This disease is the most frequent neurosis affecting children from the second dentition to the age of puberty. In fully developed cases, the entire body of the child is in continuous motion, the limbs being almost always most affected. The arms and hands are scarcely kept quiet for a moment, the shoulders are raised and depressed, the head drawn down to one side and more or less rotated. The eyes are alternately opened and closed, the forehead wrinkled and rapidly smoothed again, the angles of the mouth drawn to one or the other side. The lower limbs may be still able to carry the body, but their movements are often so severe as to impede motion and the children frequently stumble and fall. In more severe cases, even sitting is rendered impossible. If we direct that the tongue be protruded, this is often done with great suddenness and then as rapidly restored; even when it can be protruded for a few seconds, distinct twitching of its muscular bundles is noticeable. The speech becomes indistinct and may be entirely abolished, but recovery from this condition always takes place.

Numerous deviations from this symptomatology may be observed. Very often the twitchings are comparatively feeble during the whole course of the disease, and certain muscular regions may entirely escape. There are numerous gradations between this mild form and the most severe cases, in which almost all the muscles of the body are in constant motion, and throw the body in various directions, and may thus produce contusions in many places. I have seen such children covered with black and blue spots, and even thrown out of bed. Deglutition may also be interfered with. But even in the most severe cases, the ocular muscles, with the exception of the orbicularis palpebrærum, are almost always spared.

In very many cases, the movements are much more severe on one-half of the body, or may even be entirely confined to this side (hemichorea). This restriction is either manifested only at the beginning of the disease or continues throughout its course. The tongue, however, presents distinct movements on both sides in hemichorea, and this is readily explained by the numerous anastomoses of its muscular fibres. In general, I do not attach any more serious significance to unilateral than to bilateral chorea.

The movements are rarely uniform, but vary in intensity from day to day and from hour to hour. They are always increased on volitional effort. The children are, therefore, incapable of any occupation which requires the use of the fingers, such as writing, sewing, etc. In severe cases, even an attempt to speak or to sit, causes a great increase in their severity. Many patients are unable to eat alone and must be fed. Embarrassment or the consciousness of being watched often intensifies the movements, while others control themselves better under these circumstances. Quiet sleep almost always induces complete cessation, and in only a few cases have I observed the movements continue mildly during sleep. If the sleep is not quiet and profound, the movements may continue, and this fact furnishes the therapeutic indication to cause the children to ob-

tain quiet nights. It is remarkable that fatigue is always absent despite the continuance of violent movements during the entire day. In a few severe cases, in which thermometric measurements could be taken, no rise of temperature could be detected.

All other symptoms which have been described, I regard neither as characteristic, nor indeed as certain; this includes the tenderness on pressure of some of the spinous processes of the cervical vertebræ, and the possibility of intensifying the movements by pressure on certain nerves, such as the brachial plexus or crural nerve. Most of the children are perfectly well, apart from the movements, and though some of them are pale and weak, this is by no means constant. Complaint is made at times of weakness of one or the other arm, but I have never seen a paralytic condition. I have occasionally observed psychical changes; the children become irritable and violent, but a true psychical disorder (usually characterized by ecstatic delirium) is very rarely observed.

The disease almost always runs a tedious course, extending over many weeks or even several months. As a rule, it begins very gradually with uncertain movements of one hand, or distortion of the facial muscles; children at school are often punished in consequence by inexperienced teachers. After a period of six or seven weeks the movements reach their height and then gradually diminish so that a few months may elapse before complete recovery. In general, cases of slow development and moderate intensity have a tendency to a more chronic course, while I have seen the severest forms, which began abruptly, terminate favorably in four to five weeks. A fatal termination ensues in very rare cases, which almost always run an extremely acute course; the patients usually sink into a comatose condition developing after the most violent spasmodic movements combined with delirium. Among a couple of hundred cases of chorea, I have only seen a fatal result in two instances.

An anæmic girl, aged ten years, had suffered from chorea for ten weeks; violent, continuous movements which compelled her to remain in bed. Complete cessation during sleep. For past three weeks, sensorium clouded, great apathy, impossibility to sit up, diminution of sight and hearing, paralytic dysphagia, so that she was fed through the œsophageal tube. In the last few days, the choreic movements are only moderate. Pulse extremely small, 50 to 60 per minute; nothing abnormal discoverable in the heart. Constipation, very great emaciation. Death in collapse at the end of a week. Autopsy not allowed.

I may mention here that no characteristic changes have been found in the central organs in fatal cases of chorea.

With the exception of those just mentioned, I have met with no incurable cases of chorea in childhood; in one case occurring in a blind boy, nine years old, the disease, according to the statements of the relatives, had existed for a year, although in a very mild form, and the final termination of the affection was unknown to me. Its confusion with other conditions has had a good deal to do, in my opinion, with the assumption of its incurability in certain cases. But the disease is distinguished by an unusual tendency to relapse, and I would therefore advise you to prepare the parents, in every case, for its recurrence; the relapse may be as severe and prolonged as the first attack, but, as a rule, it is milder and runs a more rapid course. The interval between the primary attack and the relapse varied from three months to two years in the cases observed by me. I have also repeatedly observed a number of relapses in one case.

I may remark here in passing that the relation of chorea to rheumatism, which will soon be discussed, does not always come into consideration with reference to the development of the relapse. The reason of this tendency to relapse, which also belongs to other nervous diseases, especially convulsive ones, is unknown and will remain so, so long as we possess no knowledge with reference to the character and seat of the affection.

Although we are still lacking in this respect, there is no dearth of hypotheses and also of experimental interpretations. At the first glance, there is an evident difference between choreic movements and other convulsive diseases, for example, eclampsia or tetanus. While the latter present rigid contractures or spasms occurring in jerks as if produced by electrical shocks, you observe in chorea only such movements as are also performed in the normal condition, flexion and extension, adduction and abduction, pronation and supination, but these movements are performed involuntarily and in great haste. As Romberg has emphasized, they are combined or co-ordinated muscular actions, which increase in intensity as soon as the patient desires to set a group of muscles in action for a definite purpose, and this inability to prevent a group of associated movements constitutes one of the chief features of chorea. But it has not yet been proven that the centre for co-ordination is the site of the disease, and authors are even at variance as to whether chorea originates in the brain or spinal cord. Earlier experiments prove that decapitated animals can perform combined movements,[1] and the later experiments of Chauveau, Legros and Onimus[2] favor the location of the disease in the spinal cord, in the nerve-cells of the posterior horns or in the fibres connecting them with the motor cells; but the occasional combination with psychical disturbances (delirium, ecstasy, etc.) testifies also to the implication of the brain. I have maintained for years in my clinic that chorea, like epilepsy, is not a morbid entity, but a symptom-complex, and that it would be better to confine the term "chorea" to the distinct neurosis which is, with few exceptions, peculiar to childhood, but in other cases to speak only of "choreiform movements," which may develop under these or those circumstances. These undoubtedly include diseases of the central organs, especially the brain. In some cases of tuberculosis I observed, in combination with hemiplegia or partial paralysis of an arm, almost continuous automatic movements of these parts, which were very similar to those of chorea. This category includes athetosis and postparalytic "chorea," which occasionally occurs in hemiplegic and anæsthetic limbs and whose site was placed by Charcot in the corona radiata (?).

Chronic chorea, which may last many years, is occasionally observed in adults, rarely in children, and at the autopsy various changes have been found in the brain and spinal cord.

In the very large majority of cases, however, a material lesion of the central organs can be excluded, and we must interpret the disease as a "neurosis" which is probably due to a condition of irritation in the centres of co-ordination. The causes of this irritation are entirely unknown in many cases. Among thirty-six cases, in eleven I could discover no cause whatever, despite the most careful investigation. Among the other cases, anæmia was found a few times with pallor of the skin and mucous membranes, venous murmurs in the neck, and general weakness. Fright

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[1] Romberg: Lehrb. d. Nervenkrankh., I., S. 509.
[2] Robin's Journal, S. 403. 1870.

or terror appears to have led repeatedly to the development of chorea. Rheumatism in its various forms must be undoubtedly characterized as one of the most frequent causes. I have had repeated opportunity of convincing myself of the frequency of rheumatic chorea. It develops most frequently in acute articular rheumatism during the period of diminution or convalescence. Occasionally an alternation of both affections is noticed, as in Roger's case, in which six attacks of acute rheumatism and five of chorea occurred. Chorea follows, with almost equal frequency, mild rheumatic conditions, wandering pains with slight swelling of a few joints, which only last a few days and are accompanied by hardly any fever, or simply pains in the back, calves, or in various joints without any swelling or fever. I have repeatedly noticed that the reappearance of these rheumatic affections during the course of chorea intensified the movements which had been previously diminishing. There are even cases in which a very localized rheumatic affection—for instance, caput obstipum—gives rise to chorea. More rarely chorea opens the scene, and the rheumatism appears later. Chorea may thus be the first manifestation of the rheumatic affection, or, at least, readily causes a relapse, when such a disorder develops in a child who was previously choreic. That valvular disease of the heart, either at the mitral or aortic orifices, is often found under these circumstances, is readily explained by the known relation of rheumatism to the endocardium. I have therefore made it a duty to examine the heart in every case of chorea, and have often found valvular disease and its results, which had not manifested themselves by any subjective symptoms, not even by palpitation. But we must be on our guard against mistaking anæmic heart-murmurs for organic ones, as these children are not infrequently anæmic. The view that the chorea, in such cases, is always the result of the cardiac affection (either by reflex action or by embolic processes occurring in the large ganglia of the brain) I consider erroneous from the fact that I have not infrequently observed cases of rheumatic chorea, in which the heart was entirely normal, but on the other hand, I know from experience that in cases in which valvular lesions are present and rheumatic affections are denied by the relatives, these disorders have often been overlooked on account of their mild character. It is also possible that chorea may run its course as a manifestation of rheumatism with endocarditis, although no pains or swellings of the joints are present (Botrel, Sée, Roger). At all events we must acknowledge that embolic processes in the corpus stritaum and its vicinity would have entirely different results and would scarcely terminate so rapidly in complete recovery, as is observed in almost all such cases of chorea. In my opinion the cardiac affection has nothing to do with chorea; both are due to the same cause, viz., rheumatism, which appears to act, in some unexplained manner, upon the centre of co-ordination.

Chorea is observed much more rarely as a sequel of acute infectious diseases. In one of my cases it developed two weeks after the termination of an attack of diphtheria, in two other children in connection with scarlet fever. In opposition to Bouchut, however, I regard the appearance of chorea as a sequel of scarlatina as rare, nor have I been able to corroborate his statement that chorea, under these conditions, may only last from six to eight hours. Two of my cases accompanied the scarlatina, and were therefore not sequelæ. In one girl, who had previously suffered from chorea, a relapse occurred at the height of an attack of scarlatina, and, in the second case, important complications developed.

Boy, aged three; had suffered, in the first week of scarlatina, from painful swelling of the joints of the feet, knees, and many of the fingers. Soon after severe chorea developed, which had lasted for a week and a half when I first saw the child. Examination showed loud, systolic murmur at apex of heart, high fever with exacerbations at night. At end of third week hemorrhagic nephritis with fatal issue.

I have observed no cases in childhood of chorea due to reflex causes, like chorea gravidarum.

The effect of intercurrent diseases upon chorea varies. Febrile affections do not by any means always shorten its course, as some maintain.

The spontaneous recovery of chorea after a duration, on the average, of about three months, is apt to cloud our judgment of the remedies employed. At a certain time all remedies employed appear to prove successful, because the disease is terminating spontaneously; there is no remedy which will certainly shorten the course of chorea. Arsenic, which I have employed since the beginning of my practice, still holds the first rank in my estimation, but even this drug does not show any constant effects. I have often seen the disease continue for months despite its constant use, while, in the majority of cases, a diminution in the violence of the movements soon becomes noticeable, and may terminate in a comparatively short time (five to six weeks). According to my own experience, arsenic is suitable in all cases of chorea, if there is no contraindication on account of gastric or intestinal disturbances; it appeared to me to be especially advisable in anæmic individuals. I give two or three drops of Fowler's solution (P. 11) three times a day about an hour after meals; when administered in this manner I rarely found nausea or diarrhœa develop, necessitating a discontinuance of the remedy. The majority of the children tolerated the arsenic well for many weeks, and I consider all fears and warnings in this respect (nomina sunt odiosa) as nursery tales. I have had no experience in the employment of hypodermic injections of arsenic (one part Fowler's solution, two parts aq. destil.), but would not hesitate to resort to them if the stomach did not tolerate the remedy. An addition of opium (about 1.0 tinct. theb. in the mixture recommended above) appeared to me to increase the effect in a few very severe cases.

If violent movements continue at night on account of insomnia, I would advise the administration of chloral hydrate 0.5 to 1.0 at night. But chloral is only useful in beginning treatment, and must give way to arsenic, after quiet and sleep have been secured.

If the patients are not anæmic, the use of purgatives has appeared to me to be advantageous. I give purgatives in the beginning for a couple of days, and afterward discontinue the arsenic once a week for twenty-four hours, giving castor-oil or compound infusion of senna instead. I have observed no good results from the many other remedies recommended, not even from large doses of bromide of potassium or of strychnine, which I have frequently tried, according to Trousseau's plan, internally as well as hypodermically (0.002 to 0.003 daily). Nor can I recommend the application of the ether-spray to the spinal column, sulphur-baths, or the constant galvanic current.

Mental exertion should be avoided as much as possible during the continuance of the chorea, and attendance at school should therefore be prohibited. I have never observed a transmission of the disease to other children, but the possibility of its occurrence from imitation, especially in schools and institutions, is maintained by others. In very severe cases the children must be held in bed, surrounded by pillows in order to pre-

vent contusions. Nourishing diet, pure air, cold-water friction in the morning, if not distasteful to the children (lukewarm baths, in the other event), and finally preparations of iron (P. 12), are to be strongly recommended as after-treatment.

This appears to me the most appropriate place to refer to a disease which is often mistaken for chorea by physicians, but which is very different from it by its rarer occurrence and its symptoms. I had already called this affection "chorea electrica"[1] in an earlier article, and Hennig[2] also appears to have had analogous cases in mind under this title. In this form you never observe the sudden co-ordinate movements, increased by voluntary muscular action, which constitute chorea; the patients are entirely quiet, and lightning-like twitchings occur from time to time, perhaps every five minutes or even more frequently, especially in the muscles of the neck and shoulders, but also in other parts, and which bear the greatest resemblance to those produced by a weak induced current; as a rule they are feeble and subside so rapidly that occasionally very careful attention is requisite to detect them. I observed the affection in boys as well as in girls between the ages of nine and fifteen years. When the body is naked, the rapid twitchings of the individual muscles can be distinctly seen and felt, and the protruded tongue shows, in some cases, vermicular movements similar to those of ordinary chorea. Speech is not affected, and writing, sewing, etc., are undisturbed, except when these actions are interrupted by a twitching of the arm. One half of the body is occasionally more severely affected than the other, and in one case the movements were entirely confined to the right side. In one instance, the twitchings continued during sleep, though more feebly and rarely; in the remainder, there was complete cessation. In a boy, aged eleven, the twitchings of the head, by which the face was thrown upward and to the left, were combined at times with nictitation of both eyes and twitching of the left ear, and it was ascertained that, a year previously, this boy had suffered for a few weeks from nictitating spasm of the eyes.

In one case it was ascertained that epileptic convulsions had been present until two years ago, and after their disappearance the twitchings in question developed. In two others, these had been preceded by shooting pains in the limbs and acute articular rheumatism, and, in a girl of fourteen years, by violent crying spells. Repeatedly, however, I was unable to obtain any previous history of significance, and was then first led to regard the affection as the result of habit. I believe that these spasms, like many other neuroses—for example, chorea and epilepsy—only represent the form in which various direct or reflex irritative conditions of the nervous centres manifest themselves. Very surprising combinations of spasmodic symptoms may thus develop.

A boy, aged ten, had suffered for seven years from the following symptoms. The left half of the body presented almost continuous choreiform movements, and, in addition, the arm occasionally twitched by jerks, as in epileptiform attacks. The left side of the face had been previously affected, but was so no longer; lower limb more quiet than formerly. Complete cessation during sleep.

We here find a combination of true choreic movements with clonic twitchings, the etiology of which is entirely in the dark. This was unfortunately the rule in the cases of chorea observed by me, and treatment was

[1] Beiträge z. Kinderheilk., N. F. S., 113.
[2] Lehrb. d. Krankh. d. Kindes, 3 Aufl., S. 343. 1864.

therefore merely empirical. In one case, bromide of potassium had a decided effect; in all the others the remedies employed (arsenic, atropia, hypodermic injections of strychnia, ext. calabar) proved useless. I would chiefly advise the persistent employment of the galvanic current, as this was undoubtedly serviceable in a few cases, and once indeed produced complete recovery, though I cannot vouch for its persistence.

VI. Hysterical Affections of Childhood.

The age of childhood is by no means exempt from hysterical affections. I cannot, indeed, maintain with certainty the identity of hysteria in adults with that in children, but I know of no name which will better designate the, in part, wonderful symptoms in question; I can present, as an excuse, the fact that we know almost nothing concerning the nature of hysteria in adults, and that we must be satisfied in regarding it as a complex of the most varied motor, sensory, psychical, and even trophic, symptoms. The starting-point and the intimate connection between the symptoms are entirely unknown.

Similar symptoms are observed in children, in boys as well as in girls, though more frequently among the latter. I expect that the following description will be opposed from many quarters, because it groups together a series of diseased conditions which are usually treated of separately, such as chorea magna, catalepsy, etc. I will acknowledge that this may be wrong, but it may be claimed in my defence that transitions from one form to the other, and combinations, not infrequently occur in practice, so that we may be embarrassed as to the name to apply to any individual case. It is impossible to give a complete general description of the hysterical conditions of childhood, on account of the great variety of the symptoms and their numerous changes. I will therefore only attempt to concisely discuss certain categories of such cases.

The first category includes those cases in which the psychical symptoms, viz., complete or incomplete loss of consciousness, hallucinations, delirium, predominate. Almost all the symptoms described under the name catalepsy or eklipsis, belong to this class. Consciousness is suddenly lost, the children remain standing or sitting with a fixed stare; occasionally they fall if not supported; more rarely they are able to go around in a semi-conscious state, as if in a dream, at times muttering unintelligible words. In other cases the eyes are closed, the expression of the face unchanged, the color pale, but the normal character of the pulse and the unchanged temperature distinguish this condition from fainting. After a few seconds, at the most several minutes, everything is over and the patient is entirely restored. Many are unaware that they have had an attack; others remember the beginning of it or had only lost consciousness in part, but were unable to speak, so that they saw and heard everything going on around them as if they were half asleep. After the attack they usually continue their interrupted occupation as if nothing had happened. In exceptional instances I noticed during the attack increase of muscular tonus, which is known in the catalepsy of adults as "waxen flexibility of the limbs," the latter remaining in any position in which they are placed. The spells almost always occur very irregularly, occasionally five to six times or even more in a day, at other times every few days or weeks. It is especially painful to the physician that he can never be certain that the attacks will not degenerate into epilepsy,

though, as a rule, this does not occur. I have frequently had the opportunity, in private practice, of convincing myself of a finally favorable termination, though many months of alternate improvement and exacerbation may elapse. I usually give a favorable prognosis, if no hereditary tendency to epilepsy is present.

Even the combination with convulsive symptoms should not disquiet us too much. In a few cases, in which the attacks described above occurred repeatedly during the day, delirium with slight twitchings of various parts of the body were often observed at night, many of the children sitting upright in bed without being conscious of their condition. The following case shows that such a complication may also occur during the day:

A boy, aged nine, of perfectly healthy family, was suddenly seized with vertigo in August, 1865. In January, April, and August, 1866, he had attacks of the following character. He suddenly complained of vertigo, which was so severe, at times, that the patient fell down; the gaze became fixed, the head very hot, and delirium developed, which appeared to be always induced by the same hallucinations. On all sides the boy saw large closets and armed men about to attack him; there were slight twitchings in the hands. An attack of this kind lasted two or three days, not continuously, but interrupted by intervals in which the patient was more quiet, though consciousness was not perfectly restored. The cessation of the attack occurred suddenly, and the patient then stated forthwith that all was now over. With the exception of headache, he was perfectly well in the interval. December 23d he had another paroxysm which ushered in an attack of measles beginning on the 26th. No attacks have been observed since that time, the headaches have long since disappeared, and the patient is now a perfectly healthy officer.

A girl, aged seven, presented at the dispensary, April 19, 1873, of healthy family; fell on the head about a year ago, and was unconscious for a little while. Since then she has suffered from attacks, during which she suddenly sat or stood still, with a fixed stare or the eyes slightly rolled upward. The attacks lasted a few minutes, and occurred one or more times a day. Occasionally she could walk a few steps during the attack. Pale complexion, otherwise healthy. Further course unknown. The etiology (fall on the head) rendered this case suspicious with regard to the development of epilepsy.

The second category includes those cases in which convulsive symptoms predominate; they are either confined to a definite region, especially to the vocal organs, or may more or less affect all the muscles of the body.

Anna H——, aged nine, healthy family; had three convulsive seizures (?) in her fourth year. During the past three weeks, she suddenly emits, at irregular intervals (from five to fifteen minutes), a tone, which may be compared to the roaring of a wild animal. Complete cessation during sleep. If she attempts to cough, the roaring noise occurs instead; otherwise perfectly healthy. Arsenic, chloral, and bromide of potassium entirely useless. Recovery from application of galvanic current after a few sittings.

The use of electricity also produced rapid recovery in a boy, aged eight, who had suffered for some weeks from such severe crying spasms, that the cries could be heard on the street.

Spasm of the muscles of phonation was alone present in these and similar cases, but in other children they either precede or accompany the spasms described by me as chorea electrica (page 81). In other cases they are combined with paralytic symptoms, and the following case shows the sudden transition of nervous symptoms from one extreme to the other, which is so often observed in hysterical women:

A boy, aged ten; since the age of three years, he has suffered from attacks of quick, forcible twitching of the upper part of the body, attended with a forward

movement and shaking of the head. These attacks, which may occur every few minutes or at longer intervals, are always combined with a purring or clucking sound (spasm of muscles of phonation). Embarrassment increases the frequency and intensity of the attacks, while exercise and playing in the open air cause them to cease almost entirely. Complete cessation during sleep. The continuous employment of the galvanic current for more than a year finally produced unexpected improvement.

Marie S——, aged eleven, suffered from dyspepsia since January, 1878. At the end of February, 1879, attacks of eructation began, were very often repeated during a period of three weeks, sometimes lasting an entire day; they suddenly ceased in the middle of March, and were replaced by the following condition: the delicate, pale, emaciated child lay on the sofa, with an expression of pain, and at every expiration produced a half-whimpering, half-croaking tone, but without shedding a tear. This spasm of phonation disappeared very rarely after a few eructations, and during such intervals the features immediately assumed a cheerful expression, from which it could be inferred that there was a combination of the vocal spasm with a similar condition of the mimetic muscles. Treatment proved ineffectual. March 26th the dyspepsia suddenly ceased, the tongue became clear, the appetite excellent, while the condition, in other respects, remained unchanged. Inhalations of chloroform caused the spasms of phonation to cease, but they reappeared within eight to ten minutes. In the beginning of April the tone suddenly changed its character: it became converted into a dull groan, and the features also lost their crying expression. But speech was difficult and the child could only be made to utter a few words; in addition, paralytic weakness developed in all of the muscles, rendering it impossible for the child to hold the head or to walk a single step, the legs moving like those of an ataxic at every attempt. The child manifested a striking persistence in cutting paper dolls with the scissors all day long. April 18th the child suddenly acquired the power of speaking in a whisper, could walk a little and hold the head upright. The spasm of phonation gradually grew weaker and disappeared by May 1st, speech became loud and distinct, walking much improved, and recovery soon became complete. A milder relapse, occurring after the lapse of a few months, also terminated favorably.

The third category, in which the spasms occur paroxysmally as coordinated movements (jumping, climbing, running, etc.), either at indefinite intervals or according to a definite type, are the most surprising, and therefore readily regarded as simulated. As a rule, there are also certain psychical changes during the attacks, consisting of great excitement, hallucinations, delirium, while during the intervals a change is observed in the character, viz., great irritability, unusual cheerfulness, or more often a tendency to cry. These latter symptoms may be absent, and the child may be entirely well in the intervals. These cases are usually termed chorea magna. The name "St. Vitus' Dance" was first applied to an epidemic prevailing in Suabia at the close of the fourteenth century, characterized by dancing paroxysms combined with ecstatic symptoms, for which pilgrimage to a chapel near Ulm consecrated to St. Vitus was recommended as a curative measure.

The following is the history of the most marked case of chorea magna which has come under my notice:

Louise A——, healthy until eleven years old. At this time (Nov., 1845), frequent gnashing of the teeth during sleep, weakness of the legs, and headache; soon became unable to walk. Twitchings in the limbs, and in March, 1846, complete opisthotonos, combined with psychical disturbance, occurred every morning. This soon underwent the following change. Between 12 and 1 o'clock P.M., a feeling of dread and desire to be undressed, soon followed by rapid, panting respirations alternating with low moaning, the arms twitching slightly at the same time. Suddenly the patient appears melancholy, rolls the eyes or stares into vacancy, then jumps out of bed, walks forward vigorously and falls in a crouching position. She then jumps up lightly, walks around, and picks up various articles, which she either grasps obstinately or throws violently to the floor. She then crouches down and lies still, with an irregular pulse, as if she has fainted. Occasionally she goes to the door or window, rattles at it with enormous force, stamps on the floor, attemps to climb up the walls, claps her hands, and finally falls on the bed, on which she throws herself around incessantly. After this prelimi-

nary stage the real attack begins. The patient suddenly stands upright in bed, and jumps up four times with great violence, so that the father, a strong laborer, is scarcely able to hold her. She then sinks down exhausted, but soon leaves the bed, and runs around the room six or seven times from left to right. Occasionally she brushes against articles in her path, and then, although running rapidly, calls upon her father to remove them. Always stopping at the same place, she turns around four or five times and then sinks into her father s arms. Immediately afterward she again breaks loose and goes through exactly the same performance, repeating it not less than thirteen and never more than twenty times. These paroxysms recurred daily from 2 to 6 P.M., from March until the beginning of September. After this they only occurred on alternate days. During the paroxysm the patient recognized persons and things; hearing was extremely acute; speech was occasionally absent, though not always. Piercing, shrieking, laughing and crying spells were often observed. Hallucinations were not lacking: the patient would suddenly start, grasp the air, and speak with imaginary persons, especially with her dead mother. She always knew how many "rounds" she still had to go through, and she also stated correctly, at a certain period, that the attacks would occur, in future, only on alternate days. The force impelling the patient was supposed by her to be situated outside of her body; she often said, "It pulls me around so fast that I can hardly keep up with it." She arose an hour after the termination of the attack, and could walk a considerable distance without feeling tired. She was usually quiet at night; after September she was generally confined to bed during the day in the intervals between the attacks, on account of weakness of the legs, and normal mobility did not return until 7 P.M.

This condition lasted until November 22d. On this day the attack began at 1.30 o'clock, and consisted of only one "round," followed by paroxysms of opisthotonos. The attacks now become shorter, and entirely disappeared in the second half of December. She suffered from opisthotonos on alternate mornings, and from mild choreiform movements every night between 8 and 10 o'clock. She became cheerful and mischievous. In the summer of 1847 all the symptoms disappeared, but reappeared in the autumn, and gave place, in the winter, to ordinary chorea. In February, 1848, the opisthotonos recurred on alternate days, and a tender spot was found on the scalp, compression of which gave rise to opisthotonos. The political disturbances of 1848 had an unfavorable effect; in consequence of the storming of the armory, she lost her speech and her mental powers began to suffer. She could laugh loud, and moved her tongue freely. In June the first signs of menstruation appeared, but were not fully developed until December. Gradual diminution of the spastic symptoms now occurred, but the last traces of the disease did not disappear until July, 1850. I have often seen the patient since then and satisfied myself of her complete mental and bodily recovery.

The unusual feature of this case consists in its long duration, as it lasted five years from the first attack of opisthotonos to complete recovery. You here find the most varied manifestations of changed nervous action, viz.: psychical disturbances, hallucinations and delirium, jumping and running paroxysms, opisthotonos, choreic movements, partial hyperæsthesia of the scalp, and a sort of clairvoyance by which the patient was enabled to foretell the number of "rounds" and their change of type. Under these circumstances, as a matter of course, I and all other observers long suspected simulation; but careful observation disproved this idea completely. It is absolutely impossible that the child's powers would have proved sufficient for this form of simulation, especially during the period from March to September, 1846. I consider this enormous functional activity of the muscles as an essential characteristic of this wonderful affection, and have also found it in other cases.

An anæmic girl, aged thirteen, was entirely well during the forenoon. Paroxysms occurred daily between 3 and 6 P.M., in which spasmus nutans played the chief part, combined with entirely changed mental condition. Nodding and rocking movements of the head and trunk occurred forty to fifty times a minute, and so continuously that the possibility of such muscular action was almost incredible. The disease lasted at least four weeks, and various hysterical symptoms remained, viz.: great weakness, globus hystericus, hyperæsthesia of the scalp, etc. Complete recovery occurred on the appearance of the menses.

A healthy boy, aged twelve; the disease began with intense hyperæsthesia of the entire anterior wall of the chest. At the end of four weeks the hyperæsthesia suddenly disappeared and gave way to severe paroxysms of spasmodic cough, in which the long-drawn inspirations were accompanied by a whistling sound (spasmus glottidis). During these attacks, which were accompanied by a dread of suffocation and occurred a couple of times daily at irregular intervals, the boy jumped up so violently that he could be restrained only with difficulty. Of all the remedies employed, injections of morphine alone produced any improvement. Euphoria in the intervals, with the exception of unusual irritability of temper. Sudden disappearance of all the symptoms at the end of six weeks.

This case is distinguished by the fact that it began with a sensory neurosis, which I have never observed in any other cases. It is also noteworthy that the hyperæsthesia was bilateral and not confined to the distribution of one or more nerves. It forms a connecting-link to the fourth category, in which sensory and trophic disturbances play the principal part, while the motor symptoms are entirely in the background. As only three cases of this kind have come under my notice, I regard this category as the most infrequent.

G. K——, aged six and one-half years, examined May 2, 1878. Healthy child, had measles four weeks ago. Two weeks ago another boy fell upon the patient's abdomen while scuffling. A week later pains began in the abdomen, and continually increased in severity, so that the child cried aloud and tossed from one side of the bed to the other. The crying and rolling around gradually became so prominent that the pains subsided into the background. The frequency of the paroxysms increased daily, and were interrupted only by short intervals of complete euphoria. Temperature, 38° to 38.5°; pulse frequent, tongue coated, fœtor oris; urine abundant, dark-colored, normal; evacuations regular; anorexia. Nothing abnormal was discoverable in the abdomen; intense hyperæsthesia of the skin of the abdomen and the entire anterior surface of the thorax, so that severe pains were produced by picking up a fold of skin. Treatment: lukewarm bran-baths, muriatic acid, morphine at night. May 3d, diminution of the frequency and intensity of the paroxysms; scarcely any urine passed in last twenty-four hours, except in evacuations from the bowels. Hyperæsthesia unchanged, and is now also present in the face in the distribution of the first branch of both trigeminal nerves. May 4th, rapid diminution of hyperæsthesia and attacks of pain, abundant discharge of urine and fæces, tongue clean, appetite, no fever. May 8th, complete recovery.

A girl, aged twelve, who had recently menstruated, suffered for two weeks from severe attacks of cardialgia, which occurred daily, continued for hours, and were accompanied by constant crying, which set the whole house in an uproar. Normal in other respects, except intense nervous irritability. Morphine rapidly produced relief.

A girl, aged eleven, prematurely developed, but had not yet menstruated. In September, 1878, I was consulted with regard to frequent attacks of headache, combined toward evening with vomiting. I saw her again in February, 1879. For the past ten days, hæmatemesis has occurred on alternate nights at 8.30 o'clock, about half a cup of blackish red blood mixed with considerable mucus being discharged. The attack lasts about half an hour, and never occurs in the day. In the last four days the hæmatemesis occurred every night. The evacuations from the bowels never contained blood; the food was readily digested without giving rise to any gastric pains. All the remedies employed proved useless.

I presumed that I had to deal either with a case of simulation or hysteria. There was no known cause for the former, and examination of the teeth, pharynx, tongue, etc., revealed nothing which could be regarded as the source of the vomited blood; the attending physician had also watched the attacks, and became convinced that they were not simulated. I was confirmed in the theory of hysteria by the fact that, on February 12th, hæmatemesis occurred in the day-time, after emotional excitement. The child was therefore allowed to leave the bed, and to ride out daily, medicines were discontinued, and the advice given to make light of the entire matter. I learned from the father about the middle of May, that no attack had occurred since my last visit. The child remained well during the summer, though traces of hæmatemesis showed themselves very rarely after excitement. In October the same symptoms returned at night, though not as regularly as before. The ergotine injections ordered

by the physician had a purely moral effect, since, at a later period, the mere threat of repeating them, when signs of hæmatemesis showed themselves, caused immediate and complete euphoria.

This is the only case in which I have observed hæmatemesis as an accompaniment of hysterical affections, but examples have been occasionally reported by other authors. The interpretation of this process is difficult and remains purely hypothetical. When I remember the sudden redness of the face in consequence of emotional excitement, and the case of an epileptic child whose attacks always began with an aura consisting of marked redness of the whole integument, I think that I may accept the view that hyperæmia and hemorrhage into the stomach may occur as the result of irritation of the dilator nerves of the vessels of this organ. The periodical appearance of the hæmatemesis in one case is not surprising, as the convulsive symptoms also appeared periodically in some cases of chorea magna previously mentioned. The affection was probably connected with the development of menstruation.

The observations reported above will suffice to furnish a picture of the various forms of these remarkable conditions. All their modifications have, indeed, not been exhausted, and I could relate to you many variations in the symptoms, the explanation of which is as yet impossible. Their incomprehensible and wonderful character naturally rouses a suspicion of simulation, and we cannot be too careful in this respect during the age of childhood. I have occasionally met with such cases, and, among others, one of a girl aged twelve years, who had suffered for two years from frequent cataleptic attacks (of late, four or five attacks daily), but who did not have a single attack from the moment in which she was admitted to the Charité until her discharge, a period of at least two weeks. But I can give the assurance that, in the cases mentioned above, the suspicion of simulation may be absolutely excluded, and this is also true of many analogous cases reported by other authors. The complete cessation of the attacks in the child just mentioned during her stay in the hospital can be so much the less regarded as a proof of simulation, as it is known that radical changes in the surroundings not infrequently produce a temporary or even permanent improvement in these "nervous conditions."

The resemblance to the hysteria of adults is much more striking at times, as, for example, in the following case:

A girl, aged eleven; completely blind since her second year, when she had suffered keratitis and atrophia bulbi on both sides. Healthy until two years ago, when she over-exerted herself at school. Soon had attacks of headache and vomiting, so that she was compelled to leave school. Then devoted a great deal of time to study of music. For some months has complained of sudden darting pains in the forehead, vertigo, alternating with severe colic-pains around the umbilicus and attacks of rapid, dyspnœal respiration. Her mental characteristics did not correspond to her age; she was unusually talkative and detailed in the description of her disease. She always repeated the last words of any remark made by her mother; slept twelve hours continuously without any trace of nervous symptoms; otherwise, complete euphoria and no signs of development of puberty. Further course unknown.

I also had the opportunity, on a few occasions, of observing hysterical paralysis of the lower limbs in young girls of eleven to thirteen years of age. At times it had been preceded by severe crying spasms lasting for weeks, after whose disappearance the paralysis had developed in the same manner as in adults. While lying down and sitting the limbs

could be moved almost as well as in the normal condition, and the sensibility and functions of the sphincters were intact; but, on attempting to stand or walk, their powers left them and the patients fell to the floor, unless supported. The suspicion of a disease of the spinal cord could be immediately rejected, and, in fact, the paralysis disappeared in a few weeks, to give place to other nervous symptoms.

The etiological relations of these outwardly different, but in their nature closely connected conditions, are no less obscure than their pathogenesis. I have been unable to find any definite causes in a single case. In general, the female sex and the development of puberty predispose to their occurrence, and all these affections, especially chorea magna, have been brought into intimate relationship with the latter. But, inasmuch as boys and younger children of nine to eleven years are not by any means exempt from the conditions described, other etiological factors must also be active. These were sought in irritation of the genitalia, and onanism was regarded on many sides as the main cause of these nervous disorders. I will not deny that constant practice of this vice, when there is a strongly marked "nervous predisposition," may be etiologically important; but, from its wide-spread practice, the cases in question should be observed much more frequently than is really the case. It is, however, very justifiable in practice to keep this cause in mind. You will hardly believe that many children practise onanism in the second year, or even earlier, either by manipulation or by rubbing the thighs against one another. At this age the evil can be abated most readily by carefully watching the child; but this is more difficult in older children, who occasionally use every moment, in which they are unwatched, to practise the vice. Among other cases I remember that of a girl eight years old, who, when she hesitated using her hand, produced intense excitement by rubbing her genitals upon the edge of a chair in which she was sitting, her cheeks glowing, eyes glistening, and respirations growing rapid. But the diagnosis is not always so easy, and the most careful observation of the children, especially before falling asleep, is necessary to catch them in the act. A couple of stains in the underclothing are by no means sufficient testimony for a positive diagnosis. I have endeavored to satisfy myself with regard to this point in all cases of hysterical affections and chorea magna, but have been unable, in a single case, to convince myself that they were due to masturbation. However, you will do well to bear this factor in mind and to repress the vice, whenever it is discovered, since although it may not be the real cause of the disease, the irritation of the nervous system, to which it gives rise, may prepare the way for its development and retard recovery. How serious such irritation may become, is shown by the following case:

Charles A——, aged seven, entered hospital January 8, 1873; has masturbated since his fifth year. Continually increasing weakness, nocturnal incontinence, insomnia, inability to walk during last two weeks. When supported, he complains of vertigo, and manifests distinct ataxia on attempting to walk. Considerable increase of these symptoms on closing the eyes. Movements of the legs normal in bed, though somewhat feeble. Sensibility intact, but cutaneous reflexes from soles of feet are less vigorous and slower than normal. Urine and fæces originally passed involuntarily. Treatment.—Daily lukewarm bath, lasting ten minutes, with cold douche to the head and back, patient strictly watched, and every attempt at onanism prevented. January 23d, decided improvement, and complete recovery had occurred by middle of February.

The very rapid recovery in this case shows that the disorder was a functional one, and that continued irritation of the genitals in children may

give rise to paresis of the lower limbs with atactic symptoms, diminution of the muscular sense and diminished energy of the sphincters, as in the hysterical paralyses of women which are produced by diseased conditions of the sexual organs, or by general causes which depress the nervous system. This category also includes the cases of paresis and ataxia of the lower limbs, occasionally observed in children with severe phimosis and consequent genital irritation, and which are relieved by circumcision.

The majority of children who presented one or the other form of hysterical conditions were of delicate constitution, lean, more or less anæmic; only a minority showed normal conditions of nutrition. Some error in education could almost always be detected. Children who are brought up with unusual care and tenderness, around whom, so to speak, the whole household turns, who are surrounded by extremely indulgent individuals, yielding to all their whims, are especially subject to these wonderful affections.

You have already learned, from the cases reported above, that medicinal treatment promises no results under such conditions. I know of no remedy which has really done any service, with the exception of morphine (best used in the form of hypodermic injections 0.005 to 0.01 at a dose), from which, in a few cases, I have observed palliative effects in the removal of severe spastic symptoms. Inhalation of chloroform, in crying and other vocal spasms, had very temporary effects. In many cases—for example, running and jumping paroxysms—these remedies cannot be employed at all during the attack, or only with great difficulty, or they produce no effect. The attack must then be allowed to subside spontaneously, and we need only take care that the patient does not injure himself by his movements. Nor are we able to diminish the duration of the disease as a whole by the use of drugs. From the frequency of anæmia in these cases it is always best to treat the children with small doses of iron or arsenic, because these remedies, when administered in small doses for a long time, have a decidedly favorable effect on anæmic constitutions. I also strongly recommend lukewarm sedative baths with soap or bolus alba (50.0 to 100.0 to a bath), continued as long as possible (half an hour), good food and fresh air. In spasms of phonation the galvanic current should be tried, and occasionally produces rapid recovery, while in other cases it is either useless or aggravates the symptoms. Fortunately, we can always relieve the relatives with regard to the termination of the affection, and I would even maintain that a favorable termination can be prognosticated so much the more certainly, the more remarkable and incomprehensible the symptoms are. Cases of so-called chorea magna, spasms of phonation, and hysterical paralyses, may always be regarded as the most favorable, whereas there may be some doubts in the cataleptic variety with regard to its transformation into epilepsy. After recovery has occurred, it is well to continue the tonic treatment, and, if practicable, to resort to ferruginous baths, or lukewarm, indifferent thermal baths in the mountains or the country. Among the latter I would especially recommend Schlangenbad in the Taunus, Landek in Silesia, Johannisbad in Bohemia; among the former, Schwalbach, Pyrmont, Driburg, Flinsberg, Sarasp, and St. Moritz.

I do not doubt that a recurrence of these affections may be prevented and their duration shortened by measures of this kind. If the affection, however, proves very obstinate, it only remains to remove the child from the usual surroundings of home to new scenes, either in a hospital or a strange family. Mere change of locality is insufficient, unless the child

is at the same time deprived of the society of its mother or usual attendant. As a matter of course, attendance at school must be prohibited during the course of the disease, and mental overwork must also be carefully guarded against, even after recovery. In girls at the period of puberty the beginning menses necessitate especial quiet and care.

VII. Night-Terrors, Pavor Nocturnus.

This condition is often a source of anxiety to the parents. In the midst of a deep sleep, especially a few hours after falling asleep, the children suddenly start up, cry out aloud and continuously, grasp in the air with their hands or sit in bed with a fixed stare and anxious expression of countenance, muttering unintelligible or with difficulty intelligible words. Many tremble in all their limbs, throw themselves terrified into the arms of the frightened mother or nurse, without clearly recognizing them, and it becomes difficult to soothe them. After a short interval the scene is repeated, not infrequently several times in succession, so that half an hour or more may elapse before complete quiet ensues and the exhausted child again falls soundly asleep. As a rule, the remaining part of the night is passed in quiet repose, and on awaking the child knows nothing of the events of the night, and does not even remember the physician who was seated before the bed during the attack. Attacks of this kind are repeated at irregular intervals, sometimes every night, sometimes a few times a week, or even more infrequently. Two attacks rarely occur in one night. The children present no symptoms during the day which can be brought into any relationship with the nocturnal paroxysms. The duration of this condition, which causes great anxiety among the relatives, is entirely indefinite; in some cases only a few paroxysms occur, in other children they are repeated for many weeks or even months, but finally disappear without leaving any bad consequences.

Although I treat of this affection immediately after hysterical conditions, I do not do this for the reason that I assume any relationship between the two; on the contrary, I regard them as entirely distinct. Even the age of the patients favors this view, as pavor nocturnus occurs almost exclusively in young children near the period of second dentition, while hysterical conditions usually develop after this period. Nor can any change be noticed in the psychical character, which plays an important part in the latter. The entire affection is restricted to the nocturnal paroxysms described above, and it has always appeared to me as if the children were roused from sleep by a terrifying dream, whose effects still continued in the half-waking condition. That dream-visions and hallucinations play a part in the process is evident from the fact that they are often distinctly mentioned by the children; I have heard them beg that the chains be taken away, animals driven away, etc. It also happens occasionally that they want to jump out of bed in order to escape their fears. A boy, aged four years, who had been very much frightened by a bee, had an attack of pavor during the following night, in which he was constantly talking of a "fish" which was threatening him. This was repeated for a few nights, until the child was afraid to enter his bedroom, and always wanted to be in the open air. The more active the child's imagination and the more it is excited by the ghost-stories of nurses, the more readily will the nocturnal terrors develop; this constitutes a warning which should be taken to heart by the relatives.

I do not agree with the opinion entertained by West, that nocturnal terrors are usually caused by disturbances of digestion. I could rarely, with any certainty, detect dyspeptic affections whose removal rapidly relieved the affection. The majority of cases presented no disorders of the digestive organs, and, in my inability to detect any cause, I was forced to confine myself to the prohibition of any stimulation of the youthful imagination by the recital of tales at night, and to the administration, before retiring, of a dose of bromide of potassium (0.5 to 1.0), which appeared to me to exercise a sedative action. I have not used morphine or chloral, but would not hesitate to employ these remedies in very severe cases.

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VIII. Peripheral Paralyses.

Among the nerves which are most frequently affected by peripheral paralysis, the facial nerve occupies one of the most prominent places in children, as it does in adults. Paralysis of the facial nerve not infrequently occurs in infancy immediately after birth, the mouth being drawn toward the healthy side in crying, and the eye being often kept open on the paralyzed side; this depends upon whether the cause of the paralysis affected the labial and palpebral branches at the same time, or spared the latter. This cause is the pressure of the forceps during delivery, which occasionally leaves a small ecchymosis in the region of the parotid in such cases. The distortion of the mouth usually causes great fright in the minds of the midwife and the parents of the new-born, as it is regarded as a sign of apoplexy. But you may quiet them with the assurance that it will probably disappear within a few weeks, as soon as the extravasation of blood is absorbed, or the nerve has recovered from the effects of the compression. I say it will "probably" disappear, as a favorable termination cannot be predicted with certainty. In some cases the pressure of the forceps appears to have been so severe and continuous that degenerative processes (fatty degeneration of the nerve-fibres) develop in the facial nerve, which never recover, but produce paralysis persisting during life. I observed a case of this kind in a girl thirteen years of age, and Parrot and Troisier[1] have furnished the anatomical proof.

Congenital paralysis of the facial nerve not due to the pressure of the forceps, is much more rare. I observed this once in a boy ten years of age, who was born without artificial aid, and had paralysis of the left facial nerve immediately after birth. All the branches, including those to the left side of the velum palati, were paralyzed, and hearing was lost in the left ear, although no disease had ever affected the latter. Protracted treatment with galvanism remained entirely ineffectual. Similar congenital cases are reported here and there in literature, but their anatomical causes have not been cleared up sufficiently. The unilateral facial paralyses occurring in later childhood correspond, in general, to that already known to you. But I would call your attention to the fact that here, even more than in adults, it is necessary to cause movement of the features in order to detect the appearances. During repose you see no noticeable change in the child's face; during crying and laughing the

[1] Note sur l'Anat. Path. de la Paralysie Faciale des Nouveaux-Nés. Arch. de Toxologie. Août, 1876.

asymmetry of both halves of the face immediately appears. Inspection of the velum palati is often attended with especial difficulties, and we must frequently be satisfied with a rapid glance. The causes are also similar to those of facial paralysis of adults. A rheumatic origin is more often accepted than proven; but cases are not rare in which the action of a draught of cold air, especially when the skin is perspiring, can be distinctly proven as the cause. I have more often seen cicatrices of abscesses or enlargements of glands behind and under the ear, in the region of the stylo-mastoid foramen, give rise to paralysis by pressure upon the emerging branches of the facial nerve.

Child, aged two; complete paralysis of all the facial branches of the left facial nerve. A deep, sinuous abscess, starting from the lymphatic glands in the vicinity of the stylo-mastoid foramen. Upon opening this, considerable swelling and infiltration of the connective tissue remained. From February 25, 1861, applications of tincture of iodine; March 7th, marked diminution in the size of the tumor, paralysis unchanged. Applications continued, and iodine (0.05) internally with iodide of potassium (1.2, aq. dest. 90, syr. simp. 30—one teaspoonful four times a day. Complete recovery in the beginning of April.

But the most frequent cause of facial paralysis in childhood is caries of the petrous portion of the temporal bone, which destroys the trunk of the nerve in the Fallopian canal. The numerous cases of this kind which I have observed all agree in the circumstance that all the facial branches of the nerve were paralyzed, while unilateral paralysis of the velum palati was not always present; in a number of these cases the uvula was perfectly vertical and the movements of the velum palati were uniform on both sides. It could therefore be inferred that, in these cases, the destruction of the Fallopian canal occurred on this side of the exit of the nervus petrosus superficialis major. Deafness of the affected ear is detected with difficulty, or not at all, in small children; but more direct testimony is furnished by the otorrhœa which is always present, and sometimes combined with hemorrhage, and with which small or larger sequestra of bone, or the clean auditory bones, are not infrequently discharged from the auditory meatus; also the sensitive swelling of the temporal bone behind the ear, where the redness and fistulous openings indicate the destructive process within. This cause of paralysis is also occasionally found at a very early age. I have seen it begin in the fifth month and continue for years, until death finally resulted from complications, especially tuberculosis of the brain or other organs, meningitis or thrombosis of the sinuses. The longer the paralysis continues, the more atrophic do the facial muscles become, and I have seen them shrunken into thin, brownish yellow bands. In the cases observed by me, the autopsy always showed very extensive carious destruction of the petrous portion of the temporal bone, which occasionally extended to the dura mater. But even when a carious cavity was found immediately beneath this membrane, the latter was intact, or, at the most, of a dark color, so that a perforation of the caries into the cranial cavity could not be thought of. At the autopsy a long sequestrum could occasionally be removed from the external auditory meatus, after which, when the external ear was removed, we could look into a large cavity comprising the largest part of the petrous portion of the temporal bone. The abscesses and fistulæ situated behind the concha always communicated with the carious bone.

Almost all children in whom I observed this form of paralysis were

at the same time tuberculous, and died sooner or later. The caries was more rarely due to a simple, neglected otitis media, especially as a sequel of scarlet fever, and I therefore advise you to watch carefully all otorrhœas remaining after recovery from scarlatina.

The rarer peripheral paralyses of other cerebral nerves present even less characteristic features in childhood than facial paralysis, and this is also true of paralysis of the spinal nerves due to local causes; among these I shall enter into the consideration of only one which develops during delivery. The pressure may be so severe, not alone upon the facial nerve, but also upon the brachial plexus, as to produce paralysis of one or more groups of muscles of the arm.

Roger[1] mentions a case of this kind in which the facial nerve and an arm were paralyzed immediately after birth; the imprint of the forceps above the clavicle was still distinctly visible, and after death, which occurred soon afterward, extravasations of blood were found in the vicinity of the stylo-mastoid foramen and the brachial plexus. The same effect which follows pressure by the forceps may also be due to other obstetrical operations, especially difficult extraction, forcible dragging upon the arm, luxation or fracture being occasionally observed at the same time. The hæmatoma of the sterno-cleido-mastoid muscle, which has been previously described, (page 19), may also be present under such circumstances. This "congenital" or really "artificial" paralysis of the upper extremity may, like that of the facial nerve, either rapidly disappear, or, if the cause of the paralysis has led to degenerative processes in the nerves of the arm, may continue for many years—even during life, and be combined with sensory disturbances. For example, in a child aged five years, I observed at the same time anæsthesia of the ulnar side of the forearm. The arm assumes various positions from the contraction of the antagonists, according to the localization of the paralysis in the various muscles; on account of the preponderance of the pectoral, subscapular and latissimus dorsi muscles over the paralyzed infraspinatus, the arm is usually rolled inward, the hand being in marked pronation. The faradic excitability of the paralyzed muscles rapidly disappears and atrophy of the affected limb soon occurs; even the bones may take part in the atrophy, so that the scapula and bones of the arm become considerably shortened and the entire limb looks withered. Success can be looked for only from treatment during the first period of the disease. The persistent employment of electricity can be of service only so long as the nerves have not undergone fatty degeneration and the muscles still react.

Excessive stretching of the plexus brachialis may lead to the development, in later childhood as in adults, of paralysis, or, at least, paresis of the upper limb, which may last for weeks and months. For example, I observed paresis of the left arm in a little girl, who had sustained a severe dragging of the arm backward and outward in putting on her cloak. The power of moving the limb, especially upward and outward, was extremely restricted, and several weeks elapsed before the functions of the deltoid were completely restored, after the persistent use of stimulating frictions, and finally of electricity. If the cause is not evident, such cases may give rise to great anxiety, as not alone the parents, but also the conscientious physician, cannot rid themselves of the suspicion of a cerebral origin of the paralysis until distinct improvement occurs. This is also true of the paresis and paralyses of the lower limbs, which occasionally

[1] Journ. f. Kinderkrankh., S. 405. 1864.

persist for some days after severe attacks of eclampsia. It is then impossible to determine at first whether we have to deal with a temporary disturbance of motion or a cerebral affection, since, as we shall soon see, very grave diseases of the brain, especially tuberculosis, not infrequently begin by suddenly developing convulsions followed by paralyses, which disappear after some time, then return unexpectedly, or manifest their true nature by the onset of fatal tubercular meningitis. I therefore advise you to be very careful in the diagnosis of all cases of partial paralysis, whose peripheral causation is not placed beyond a doubt, and not to leave out of sight the possibility of a central affection, although no further symptoms may be present. As a matter of course we must also think, under such circumstances, of the possibility of an injury of the affected joints, of luxation or subluxation of the shoulder or elbow, and even of fracture of the bones. I would not refer to this, were it not for the fact that I have seen a few cases in which these surgical affections were regarded as pareses by careless physicians. The opposite occurs occasionally in the lower limb, where a dragging of the leg or a slight halt is erroneously regarded as beginning coxitis, although it is only due to injury of the muscles from a fall, and soon disappears when rest is maintained.

IX. Spinal Paralysis of Childhood.

This disease, which was formerly described as "essential paralysis," deserves special attention on account of its relative frequency and the serious consequences which it may entail for the remainder of life. The majority of cases occur from the age of one and one-half to four years. The parents relate that the child has been unable to move an arm or a leg for a few weeks or months. Upon examination you will find, in a part of the cases, the affected limb entirely motionless. The limb is flaccid, like that of a doll, so that it can be thrown hither and thither without any resistance. The sensibility is almost always completely intact. In other cases the paralysis is already diminishing, and certain movements can still be performed. In all other respects the child is usually in perfect health. The beginning of the affection is almost always described in the same manner, and the following case will serve as an example:

A girl, aged four, was brought to me on July 20, 1874. She had been suddenly seized in September, 1873, with a high fever, the temperature rising to 41°. The child complained of headache and was drowsy; no other local symptoms. Disappearance of the fever in two days; on attempting to rise, paralysis was discovered in both lower limbs and the right arm. After the lapse of three or four days, the power of the lower limbs was restored, but the arm remained paralyzed.

This is the usual course. In the midst of perfect health, the children are affected with fever, usually with very high temperature, complain of headache, if they are old enough, and are somewhat somnolent; more rarely they lie in a comatose, semi-unconscious condition, from which they can with difficulty be roused, or they even present spasms and contractures. More rarely the scene is opened by a convulsive seizure. This condition subsides after a few days—at the most a week—and the parents now find that one or more limbs can no longer be moved. Either both legs and an arm are affected, or an upper and a lower limb on different sides, more rarely the arm and leg on the same side, still more rarely both arms or

both legs. Occasionally the paralysis is limited from the beginning to one limb. But the characteristic feature is that the paralysis reaches its acme at the start or, at least, within the first twenty-four or forty-eight hours. In exceptional cases it was stated that the paralysis continued to increase in the first few weeks after its development. The restoration of motion often occurs very rapidly; after a few days or a week one or the other limb is again movable, or individual groups of muscles of a limb can be moved while others remain absolutely paralyzed. After a few weeks the paralysis is often confined to an arm or a leg. The condition then remains unchanged for many months or years, and may even persist during the whole life.

After the paralysis has lasted for some weeks or months, a series of characteristic symptoms appear and render the diagnosis unquestionable. These symptoms are increasing atrophy of the paralyzed limbs, diminution of temperature and electrical irritability, especially the faradic, which disappears before the galvanic. The paralyzed limb diminishes in circumference, the deltoid and shoulder muscles disappear in particular, so that we can readily enter between the acromion process and the head of the humerus, and the shoulder, especially when seen from behind, appears markedly flattened in comparison with the healthy one. The arm and forearm also atrophy, all the muscles are flabby and thin, the ligaments of the joints strikingly lax, and it must be borne in mind that in very stout children the atrophy of the muscles may appear smaller than it really is, on account of the overlying fat. The hand readily recognizes the lower temperature of the paralyzed limb, when compared with the healthy one, and by suitable thermometers it is found that the diminution may reach 1° C. The reaction of the parts to electricity is also very characteristic. The reaction, especially to faradism, disappears almost as rapidly as in peripheral paralysis, while the galvanic current is still active or even produces increased reaction. As early as the fifth day from the beginning of the paralysis, more often after the lapse of a week, some of the muscles react very feebly to the faradic current—others not at all; this is a grave sign, as the latter are apt to remain paralyzed for the remainder of life.

In addition to the atrophy of the muscles, retardation in the growth of the bones is also observed, the limb becoming shorter than the healthy one. This inhibition of osseous development does not always keep pace with the degree and extent of the paralysis and muscular atrophy; the latter may be very marked and the limb scarcely shortened, while, in other cases in which the paralysis and atrophy are very limited, the growth of the bones may be considerably interfered with, a fact which Charcot attempts to make responsible for the direct influence of the central disease on the nutrition of the osseous system.

If recovery from the paralysis does not occur within ten to twelve months, there is very little hope that this will ever take place. About this time a new series of symptoms usually develop. As the paralysis and atrophy do not affect all the muscles of a limb uniformly, the antagonists of the affected ones will produce deformities by their contraction, consisting of pes equinus in the great majority of cases, but also appearing as pes varus, club-hand, and other abnormal positions of the upper or lower limbs. The explanation of the deformities by the tonus of the antagonists was universally accepted until recently, and even now has many adherents. Volkmann attributes the deformities to the effects of the position of the limbs and their own weight, while others (Hitzig)

regard it as the result of retraction of connective tissue in the affected muscles.

Recent anatomical investigations have shown that the spinal cord is the real seat of the disease. Autopsies made during or soon after the initial fever are still lacking; almost all the anatomical examinations date from the later stages of the disease. But all the observations prove beyond a doubt that we have to deal with an inflammatory (according to Charcot an "irritative") process in the anterior horns of the gray matter of the cord, and which may extend into the anterior portions of the lateral columns. According to the situation of the paralysis, circumscribed myelitic foci are found in the upper or lower parts of the spinal cord, especially in the cervical and lumbar enlargements. In the relatively recent cases (two and six months respectively) described by Roger and Damaschino,[1] these foci had a soft consistence, reddish color, and under the microscope showed an increase of capillaries, thickening of the walls of the vessels, with profuse formation of nuclei and very numerous granular corpuscles. The multipolar cells of the anterior horns and the motor roots of the nerves were atrophic, and slight sclerosis of the anterior and lateral white columns was noticeable. The older the affection, the more marked is the atrophy of the multipolar ganglion-cells, combined with sclerosis of the anterior gray horns and atrophy of the emerging motor roots; in very old cases there may be diffuse atrophy of the anterior horns and the antero-lateral columns, with disappearance of the large ganglion-cells and profuse development of corpora amylacea (Charcot, Leyden[2]).

At an early period of the disease, a large proportion of the primitive bundles of the paralyzed muscles appear to undergo simple atrophy without any fatty degeneration. The accumulation of fat within the sarcolemma occurs at a later period in the place of the disappearing primitive bundles; it also develops between the fibres, and sometimes to such an extent that the atrophy of the muscles is masked thereby, their dimensions appearing normal or even increased. But the development of fat is by no means constant; it may be present in some muscles, absent in others, and the interstitial connective tissue may then be more or less hyperplastic. The macroscopic appearance of the muscles varies accordingly; they may be either thin, pale red, yellowish, or large, and then appear to be converted into fat; if general emaciation occurs, this fat also disappears. The nerve-roots and trunks of the paralyzed parts were not infrequently found atrophied and appeared thin and gray, while in other cases the thickening of the sheath and increase of interstitial connective tissue and fat conceal the atrophy.

In view of the facts just mentioned, there can be no doubt that spinal paralysis of childhood is due to a disseminated myelitic process which especially involves the gray substance of the anterior horns, particularly in the cervical and lumbar enlargements.[3] The process may extend in time to the antero-lateral columns, and, in a few cases, even the posterior horns were found affected, thus explaining the fact that sensory disturbances (anæsthesia, spontaneous pains) are exceptionally observed. The sphincters of the bladder and rectum are only affected in exceptional instances. In one case I observed paralysis of the muscles of the neck, but this disappeared at the end of a week.

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[1] Gaz. Méd., 1871. [2] Klinik d. Rueckenmarkskrankh. Berlin, 1875.
[3] Kussmaul therefore proposed to call the disease poliomyelitis acuta anterior.

All authors deny any implication of the brain. Leyden[1] expressly states that the facial and hypoglossal nerves and the ocular muscles are never implicated, and that in only one case did he find a small sclerotic patch in the medulla oblongata which produced no symptoms during life. The following observation therefore appears so much more important:

Bertha M——, aged two and a half, presented May 1, 1876. Three weeks ago she had sudden fever, with vomiting and somnolence, continuing two days. On the second day, weakness of the right hand, and paralysis of the entire limb on the following day. After the third day the child felt well, but paralysis was present in the right arm and a part of the left facial nerve; the latter had not entirely disappeared when I saw the case. The left facial muscles reacted normally to faradism, those of the arm showed diminished reaction. The facial paralysis recovered without treatment by the middle of May; considerable improvement in the arm ensued from the persistent use of electricity; the atrophy remained unchanged.

The characteristics of infantile spinal paralysis were clearly marked in this case, and the implication of the facial nerve constitutes a hitherto undescribed exception. If we consider that other affections of the spinal cord are not infrequently combined with analogous changes in the brain, we do not know why this may not happen in infantile paralysis; the occurrence of stupor and convulsions in the initial stage seems to indicate that the brain is more often implicated than we suppose.

The symptoms of infantile spinal paralysis are so characteristic that it is scarcely possible to mistake it for any other form of paralysis. The initial febrile stage, the sudden paralysis, which is hardly ever progressive, but always retrogressive, the almost constant integrity of sensation and of the sphincters, the rapid disappearance of the faradic excitability of the muscles, the early atrophy and diminution of temperature, finally the deformities—all these symptoms are not found associated in any other disease. But the question nevertheless presents itself whether all cases having the clinical characteristics of infantile spinal paralysis are caused by the disseminated myelitic patches described above. In fact, it cannot be denied that peripheral paralysis may run a very similar clinical course to the disease under consideration. The former may soon be followed by atrophy of the muscles and diminution of faradic muscular excitability, but the febrile initial stage, which is often combined with cerebral symptoms, is always absent in such cases. Many years ago Kennedy called attention to paralyses which occur suddenly in perfectly healthy children, without any prodromata. The children sometimes go to bed feeling well, and wake up in the morning paralyzed in an upper or lower limb; the paralysis usually disappears after a shorter or longer period (so-called temporary paralysis), but it may also pursue the same course as spinal paralysis. Search was usually made in such cases for local causes, and when none were found, it was supposed, usually without justification, to be due to pressure of the head of the bone on the nerves of the arm during sleep, or to a cold or a reflex irritation from dentition. Under such circumstances and in the absence of febrile prodromal symptoms, it remains an open question whether we must accept the view of a myelitic process or of a peripheral cause; this is true only of paralysis of a single limb, as there can be no doubt of the myelitic origin in cases of widespread paralysis.

Concerning the causes of infantile spinal paralysis we know almost

[1] L. c., II., p. 555.

nothing. The symptoms were occasionally observed after acute diseases, for example, scarlatina, measles, small-pox, typhoid fever, or pneumonia; in the majority of these cases recovery occurs, but atrophy may develop at a later period, and it must be left undecided at present whether the anatomical conditions in these cases correspond exactly with those found in infantile spinal paralysis.

In the majority of cases the physician is summoned only after the disease has lasted a few weeks. If we see the case in the acute initial stage, we will naturally be unable to determine that a spinal paralysis will result. Under these circumstances we should apply an ice-bag to the head, a few leeches behind the ears, or on the temples in very severe cases, and give purgatives internally, such as calomel (0.03 to 0.05 every three hours) or inf. sennæ comp., etc. If the paralysis has already developed, internal treatment is useless, and only the earliest possible and continuous use of electricity can further the recovery of the paralysis and prevent atrophy. Although some maintain that electricity does not have much effect, or that all hope must be abandoned if a successful result has not been achieved at the end of a year, this view is opposed by the successes obtained by Duchenne and others, even after the lapse of this period. We can only give the advice to be persistent in its use. Electrical treatment may be begun a few weeks after the inception of the disease. The galvanic current is recommended during the first stage, the faradic being too irritating and painful, and in addition the reaction to the latter may be diminished or lost. In the later stages the faradic current is as suitable, if not more so, than the galvanic, as the object then is to irritate the undegenerated muscular fibres by a vigorous stimulus and to increase their nutrition. I repeat that the treatment, in obstinate cases, must be continued for years before it is abandoned as hopeless. Gymnastic exercise is especially advisable in combination with electricity. In the later stages orthopedics furnishes important adjuvants in the form of apparatus and operations (tenotomy), which endeavor, on the one hand, to prevent deformity and aid the atrophic muscles, and, on the other hand, to relieve the contracture of the antagonists. The apparatus, as well as the kind of gymnastics, must be adapted to each individual case.

Although the return of electrical excitability is a favorable sign, experience teaches that the reaction to both currents is occasionally absent, although the first traces of voluntary motion become noticeable, and we must then continue so much more persistently with the use of electricity. It is also advisable to give the children the benefits of forest and mountain air, and salt and ferruginous baths, which irritate the sensory nerves of the skin by their large amount of carbonic acid, and then act upon motion in a reflex manner. If the case is of long standing—if the ganglion-cells have become atrophic, and the muscles shrunken and fatty, no measures will prove of any avail.

Infantile spinal paralysis is the only disease of the spinal cord which affects the period of childhood with an especial predilection and under certain characteristic symptoms. Among other spinal affections, the paraplegia resulting from spondylitis alone plays a prominent part in childhood on account of its frequency, but is not distinguished in any way from the similar affection in adults. I shall not discuss it here, as the causative vertebral affection is considered in all surgical works. Other diseases of the spinal cord, viz., inflammatory processes, hemorrhages, even tumors of various kinds, may occur in children, though much more rarely than in adults; but their symptoms are then the same as in

later life. There are two diseases which have recently excited interest with reference to the age of childhood, viz.: multiple sclerosis and so-called "spastic spinal paralysis." The former has been demonstrated in children, though rarely, by post-mortem examination. The latter, as is well known, is very little more than a symptom-complex, which does not correspond to any definite anatomical change. Such cases, which are characterized by chronic paresis or paralysis of the lower limbs (dating even from infancy) with contracture of individual groups of muscles, have been repeatedly observed by me in children. In the attempt to stand or walk, the application of the sole of the foot to the floor immediately produced rigid contracture of the calf-muscles with pes equinus position of the foot, or there was also contraction of the adductors of the thighs, by means of which they were almost drawn across one another and every movement rendered impossible; but all these cases escaped further observation and were anatomically incomplete. The frequently observed complication with slight mental development, even with idiocy, permits the conclusion that similar symptoms may start from the brain, and I will soon mention a case to you in which the autopsy disclosed very marked structural changes of the cerebral cortex. It is hardly necessary for me to remind you that secondary degeneration starting, under such circumstances, from the affected part of the brain, may extend into the spinal cord.

X. Pseudohypertrophy of the Muscles.

This remarkable disease always develops during childhood, but may be protracted into youth or adult life. When fully developed, this condition presents a very characteristic appearance. While the muscles of the calves and thighs, especially the former, present an unusual volume and striking firmness, the muscles of the chest, arms and shoulders are atrophic, but not uniformly, since, on more careful examination, nodular thickenings are found here and there in the deltoids, and perhaps also in the triceps. The recti abdominis, glutei, and the muscles of the loins and back, are also often enlarged, though not to the same extent as those of the lower limbs. The gait is very peculiar. The patients have a waddling walk, with legs well separated, and the foot (which is in a position of pes equinus) only touches the ground with the tip; the lordosis of the lumbar vertebræ is much more marked than in the normal condition. If you let the patient lie on the floor and then rise, you will notice that, in so doing, "he climbs up on himself," as it is customary to say. He first places himself in such a position as will enable him to use his hands as levers in rising, and finally effects this by first supporting the hands firmly on the legs, then on the thighs, and thus lifts the trunk upward. I have observed only six cases of this disease, but in none was this peculiar manner of rising absent. All the movements are coarse and awkward, and become weaker the more the disease progresses. The adipose tissue, especially in the lower limbs, may still be well preserved, but disappears after the final development of a marasmic condition. The atrophic muscles of the upper part of the body often show fibrillary twitchings like those in progressive muscular atrophy of adults. The integument of the lower limbs not infrequently presents a marbled appearance and cool temperature, as the result of venous stasis. Many of these patients are mentally weak and have labored speech, and, in a few cases, increase in the size of the tongue is said to have been observed (Chvostek).

The development of this disease begins in the middle of childhood, and in a few cases it is expressly stated that there had been awkwardness in the movements from early childhood. Usually the majority of patients are first seen at a more advanced stage, when they are seven to ten years old, or even later. In one patient, aged ten, Demme observed a slow pulse (44 to 60) and a not inconsiderable but inconstant amount of sugar in the urine. If the general health is undisturbed, the disease may continue for ten to twenty years, and a period of quiescence may develop. If the patients do not succumb to an accidental complication, the increasing atrophy and weakness of the respiratory muscles, or a marasmic condition, usually put an end to life.

The anatomical process in the muscles is very like that of infantile spinal paralysis and progressive muscular atrophy. There is diminution in the size of the muscular fibrillæ, which are replaced by interstitial development of fat and fibrous tissue (atrophia musculorum adiposa) in the apparently hypertrophic parts (calves and thighs). These compensations can also be partially found sometimes in the atrophic muscles of the upper parts of the body (deltoid, etc.) in the form of separate nodules; a few hypertrophic primitive bundles may also be present. It has hitherto not been determined whether the atrophy is due to the pressure of a primary connective-tissue proliferation between the bundles of muscular fibres, or to some other cause. The changes occasionally found in the spinal cord cannot be regarded either as constant or essential.

The electrical contractility diminishes in the atrophic as well as the thickened muscles, and corresponds to the progressive atrophy of the fibrillæ; the sensibility of the skin remains intact.

It is noteworthy that, with few exceptions, all cases occur in boys, and occasionally in several children of the same family. Apart from this inexplicable predisposition, all other causes mentioned are uncertain. Treatment is entirely useless.

XI. Paralysis from Hemorrhage.

Much more frequent than spinal paralyses in children are those due to cerebral disease, the general characteristics of which, viz., hemiplegia and the preservation of the electrical contractility of the paralyzed muscles, are the same as in adult life. Atrophy of the muscles may indeed occur in this form of paralysis, but it develops with extreme slowness, never attains the high grade it does in infantile spinal paralysis, and appears to depend upon the disuse of the muscles. Contracture from the predominance of the non-paralyzed antagonists, more frequently caused by central irritative conditions, likewise tremor and automatic movements are frequent accompaniments.

In many cases the development of hemiplegia occurs suddenly in the midst of apparently perfect health, and we are then inclined to attribute it to a cerebral hemorrhage or embolic process. Both conditions occur with comparative rarity during childhood, and suddenly developing hemiplegias, despite their apoplectiform symptomatology, are much more often the manifestation of a cerebral affection which has already existed for some time, especially tuberculosis.

The rarity of cerebral hemorrhage in childhood must be attributed to the fact that the most frequent cause of the hemorrhage in later life, viz., fatty degeneration of the small cerebral arteries and the formation of

small aneurisms upon them, almost never occurs at this age. The most experienced physicians in this department admit that they have seen very few cases of cerebral hemorrhage which were recognizable clinically. I have often seen small, capillary hemorrhages as a result of tubercles of the brain, tubercular meningitis, thrombosis of the sinuses, and other diseases; but I have hitherto been unable to obtain an autopsy which would confirm the diagnosis of larger cerebral hemorrhages in children. In the following case the diagnosis of cerebral hemorrhage is probable, though not positive:

A boy, aged seven, suddenly became hemiplegic on the right side, consciousness being retained. The lower extremity improved more rapidly than the upper, in which the rigid contraction of the flexors of the fingers gave the hand a claw-like shape and rendered it almost incapable of function. Dipping the hand in warm water relieved the contracture, and the extensors then acted quite freely. Aphasia was present in the beginning; at the end of ten months it had disappeared in so far that the boy could speak a few words. The protruded tongue distinctly showed a deviation to the paralyzed side. Health normal in other respects.

In a child, aged three, suffering from very severe whooping-cough, a very severe paroxysm was followed by convulsions and unconsciousness, lasting nine hours and leaving left hemiplegia. This continued for several weeks; the arm and leg were flaccid and immovable, the facial muscles intact. In view of the fact that, in whooping-cough, hemorrhages may occur into the connective tissue of the eyelids, conjunctiva, from the nose, and even from the ears, we can infer almost with certainty that the case just described was one of cerebral hemorrhage.

Purpura hæmorrhagica will also give rise, in rare cases, to apoplexy in children. Mauthner reports a case with autopsy; I have had only one case, and in this an autopsy could not be obtained.

A child, aged seven; four years ago he had scarlatina followed by dropsy. During the past year, had morbus maculosus with repeated hemorrhages from the mouth, nose, ears, eyes, intestines, and kidneys; great weakness, loss of appetite; spleen not enlarged. Sudden development of severe convulsions and unconsciousness, soon followed by left hemiplegia with paralysis of the facial nerve. Death the same night. Autopsy refused.

Whether the extravasation, concerning whose occurrence there can be very little doubt, took place into the brain-substance or into the meninges, must remain undecided.

In the following case, also, in which aphasia was present, the assumption of a circumscribed cerebral hemorrhage appears to me to be undoubted.

May 29, 1878, I was summoned to a three-year-old boy who had suffered from intermittent fever for ten weeks. Two weeks ago, on the day following a fall upon the head, the last attack of intermittent fever had occurred. During the hot stage he was taken into a railroad car, and there became affected with eclampsia, which lasted almost seven hours. On coming out of the unconscious condition, marked interference with speech was noticed, which passed into complete aphasia at the end of twenty-four hours. Headache and increased temperature of the head were present at first, but soon disappeared under the use of ice-fomentations and calomel. Complete euphoria with the exception of the aphasia; no paralysis. After a few days the ability to speak gradually improved, and recovery was complete at the end of two weeks.

If we consider the combination, in this case, of various circumstances favorable to cerebral hyperæmia, the previous concussion of the brain, and the exciting ride in the railroad during the hot stage of an attack of

intermittent fever, we are readily led to accept the view of a hemorrhage (probably in the neighborhood of the third left frontal convolution) caused by the severe congestion. The absence of paralysis does not negative this view, as cases are not lacking in which very limited paralysis was due to small hemorrhages, which were demonstrated on autopsy. That the supposed cerebral hemorrhage began in this, as in other cases, with severe eclampsia, is so much the less surprising, as convulsions occur much more frequently in childhood than in adult life in connection with hemorrhages into the brain. The previously mentioned small extravasations, which are found in the form of red spots or patches as large as a pea in the tissue of the pia mater or the cortex, present no other symptom than convulsions during life. This is true of the capillary hemorrhages in the brain or pia mater, which are observed in asphyxiated new-born and in the first weeks of life, as well as those which are not infrequently found as the result of grave constitutional diseases (typhoid fever, diphtheria, scarlatina, etc.) or local cerebral affections (especially tuberculosis of the brain and tubercular meningitis). None of these hemorrhages can be diagnosed with certainty, because their symptoms cannot be separated from those of the primary disease, and are often entirely absent. In tubercular meningitis I have frequently found no inconsiderable extravasations into the pia mater and sometimes into the substance of the brain —for example, in the commissures of the third ventricle, without any corresponding change in the ordinary symptoms. The rarer cases of larger extravasations, which develop in older children with sudden hemiplegia, present no notable differences from the apoplexy of adults, either anatomically or clinically; this is also true of the hemorrhages which occasionally occur from traumatic causes in the space between the dura mater and arachnoid (apoplexia meningea). It may be here mentioned that the disease described by French writers as "hemorrhagies dans la cavité de l'arachnoïde" is not a hemorrhage, according to our present views, but a pachymeningitis, *i.e.*, an inflammation of the inner surface of the dura mater, accompanied by small hemorrhages.

Cerebral paralysis may suddenly develop from embolic processes in childhood as in adult life. Although more infrequent in the former, a number of cases have been reported in which clots from the left heart, or even from the pulmonary veins, were carried by the current of blood into the carotid and its branches, especially the middle cerebral, and gave rise to more or less extensive softening in the parts supplied by this vessel. The same diagnostic difficulties are present as in later life, and the differentiation between embolism and hemorrhage is only approximately possible when we are unable, by examination of the heart, to obtain any basis for diagnosis (endocarditis, valvular lesions). If nothing abnormal is found in the heart, the possibility of embolism is not excluded, as the thrombus, from which the embolus is formed, may be situated in the pulmonary veins and pass from them into the left heart and aorta. A case of this kind was observed in my clinic in August, 1877. It occurred in a boy aged two and one-half years, suffering from chronic pneumonia and cheesy degeneration of the bronchial glands, in whom right hemiplegia combined with contracture suddenly developed. After death an embolus was found in the middle cerebral artery, with extensive softening of the corresponding hemisphere of the brain, the embolus being derived from the main branch of the right pulmonary vein, which was filled by a thrombus.

The sudden development of hemiplegia may also be due to diseases

of the brain, which have either been entirely latent for some time or have manifested themselves by other cerebral symptoms, especially by convulsive seizures. Among these diseases the following one undoubtedly occupies the first place:

XII. Tuberculosis of the Brain.

Of all the chronic cerebral affections of childhood this is undoubtedly the most frequent, and is indeed so predominant that we will rarely be mistaken in making a diagnosis of cerebral tuberculosis when chronic cerebral symptoms are present. This affection may occur at a very early age, and among twelve cases under my observation there were ten between the ages of nine months and two years.

The diagnosis of cerebral tuberculosis is aided by a peculiar complex of symptoms and conditions. The children are hardly ever entirely well, but bear evidences of scrofula or tuberculosis, eczematous eruptions, inflammations of the eyes, otorrhœa, swelling of the lymphatic glands, osteomyelitic enlargement of the phalanges of the fingers and toes, or other bones, and especially caries of the petrous portion of the temporal bone. These conditions may no longer be present when the cerebral symptoms develop; it is sufficient that the children have previously suffered from them, or even that the brothers or sisters have died from "disease of the lungs or glands." These data render the diagnosis much easier; we will very rarely find, on careful examination, that the child has been entirely free from scrofulous manifestations.

In a number of these cases an epileptiform attack suddenly occurs, which may be repeated at irregular intervals. In children during first dentition or in rachitic patients, it is hardly possible to distinguish these convulsions from the more harmless ones which we have previously discussed (page 67), and careful attention must therefore be paid to the symptoms occurring in the intervals, which may continue even for months. Every cerebral symptom during these intervals is important in the diagnosis. Even little children, but especially the older ones, often complain of headache, which occurs in paroxysms like migraine, is not infrequently combined with vomiting, and compels the children to lie quiet or to support the head with the hand. In others, strabismus develops usually in one eye, and is often unnoticed among the poor, or attributed to a bad habit. Suddenly, after one of the convulsive attacks referred to, or occasionally independently, paralysis of one limb occurs, or hemiplegia, with or without implication of the facial and ocular nerves. As in all central paralyses of the facial nerve, only a few branches, especially those to the lips, are paralyzed, while the paralysis of the motor oculi communis is manifested by diverging strabismus and dilatation of the pupil, that of the abducens, by internal strabismus and inability to move the eyeball outward. These paralyses may disappear after a few days or weeks, and the inexperienced physician is then very strongly inclined to regard them as residua of the epileptiform attack, until the scene is repeated and perhaps has a rapidly fatal issue.

Martha M——,[1] aged two, rachitic and scrofulous; repeated convulsions, cannot hold the head up, fretful disposition. June 29, 1864, convulsion limited to the left side of the body, which was paralyzed immediately afterward. Cerebral nerves and sensibility normal. I diagnosed tuberculosis of the right hemisphere, with congestion around

[1] Beitr. zur Kinderheilk , N. F., S. 64.

it. July 1st, marked improvement. July 8th, paralysis disappeared. July 26th, severe convulsions on the left side, followed by coma. Repetition the of attacks on October 16th, February, 1865, and a very severe one on March 30th, with fatal termination.

Autopsy.—Severe congestion of the pia mater, especially on the left side. A yellowish gray tubercle, as large as a pea, and surrounded by a thin capsule of connective tissue, was found in the posterior lobe of the right hemisphere. No tubercular meningitis. Miliary tuberculosis of the pleuræ and cheesy enlargement of the bronchial glands.

I again call your attention to the unilateral character of the convulsions, which has already been referred to (page 67), and which justifies us the more in diagnosing a serious affection of the opposite hemisphere, when, as happened in the above case, the ensuing paralysis occurs on the same side as the convulsions. This case furnishes an illustration of solitary tubercle; but you should not for this reason be led to believe that unilateral convulsions and hemiplegia can occur only when the tubercles are solitary or confined to one half of the brain. The following case shows that hemiplegia may develop in a tubercular affection of both hemispheres:

O. A——, two and one-half years of age, admitted October 24, 1876. Had a convulsive attack a year ago. Four days ago had sudden hemiplegia of the left side, soon followed by tubercular meningitis and death. Autopsy.—Numerous adhesions between the dura and pia mater. A number of tubercles, from the size of a hazel-nut to that of a walnut, in the cortex of the hemispheres (six in the right, four in the left), and an equally large one in the posterior part of the left half of the cerebellum. Tubercular meningitis.

We here touch upon an important point in the pathology of cerebral tuberculosis, viz., its latency. As in this case the tubercles of the left hemisphere were not manifested by any symptoms during life, so also much more extensive tuberculosis of the brain may be entirely latent during life, and be revealed only on autopsy. Indeed, I am inclined to believe from my experience that multiple tubercles have a greater tendency to latency than solitary ones. The following case will serve as an example:

Child, aged fourteen months. Caries of the petrous portion of the right temporal bone, paralysis of the right facial nerve, enlargement of the glands. No cerebral symptoms. Phthisis. Death. The autopsy showed a tubercle as large as a walnut at the surface of the right frontal lobe, another larger one in the occipital lobe, a third one near the base of the brain; also multiple, large tubercles on the surface of the left hemisphere; the left lobe of the cerebellum almost entirely converted into a soft, cheesy mass.

In this and other similar cases, tuberculosis and cheesy degeneration of other organs were far advanced, and the latency of cerebral tubercles occurs most frequently under such conditions. I therefore still adhere to the opinion formulated by me in 1868,[1] that in children who suffer from extensive tubercular degeneration of the lymphatic glands, lungs, abdominal organs or bones, and who perish with symptoms of tubercular meningitis of a normal, more frequently abnormal, course, we may also diagnose, with probability, tuberculosis of the cerebrum or cerebellum, though the latter may never have been manifested by any definite symptoms. The probability is so much greater if the petrous portion of the temporal bone is carious.

The occurrence of cerebral tuberculosis, with repeated epileptiform attacks followed by hemiplegia, is only one of the forms under which the

[1] Beiträge, N. F., 69.

disease is manifested. In another series of cases it begins gradually with unilateral paresis, which increases and is often combined with tremor or contracture of one or both limbs. After the lapse of months or even years, during which the condition shows many variations, violent convulsions or tubercular meningitis form the fatal termination. The following case will serve as an example[1]:

W. J——, aged two; cough for six months, emaciated, rachitic, admitted April 3, 1875. Constant tremor, at times twitching, of the right arm and side of the face; no paralysis, sensibility normal; consolidation at apices of the lungs. After a few days, increase of tremor, the head and right lower limb also becoming affected. The thoracic and abdominal muscles and the cremaster on the right side manifest repeated twitchings at short intervals. Slight paresis of the right arm. April 6th, continuous contracture of the right thumb. April 7th, nystagmus of the right eye. Death in high fever and collapse. Autopsy.—Œdema of the pia mater, especially over convexity of the left hemisphere, in which numerous miliary tubercles are embedded. A yellow tubercle, as large as a hazel-nut, with moderate softening around it, is found immediately in front of the middle of the fissure of Rolando. Phthisis pulmonum, etc.

The duration of the disease may vary greatly, so that, in some instances, many months and even years elapse before the fatal termination, while in others the first symptoms are observed a comparatively short period before death, so that we must then suppose the disease to have been latent until its last stage.

Cerebral tubercles appear most frequently in the form of grayish, cheesy nodules, from the size of a pea to that of a hazel-nut, rounded or nodular in shape, and generally found in the gray matter, the cortex, large basal ganglia, pons Varolii and cerebellum, though the white substance, the corpora quadrigemina, etc., are not exempt. Tubercles of the cortex, which are situated immediately beneath the arachnoid and pia mater, can scarcely be distinguished from those developing in the meninges and then spreading into the cortex. In both cases the arachnoid and dura mater situated over the tubercles are more or less adherent to one another, so that in drawing off the dura mater a part of the tubercle may remain adherent to it. The tubercles may attain the size of a walnut or even larger, and then, as a rule, they are no longer uniformly cheesy on section, but contain fissures and cavities, which are filled with a curdy fluid. In one child I found a tubercle as large as a hen's egg on the outer surface of the right optic thalamus, in other cases a diffuse cheesy degeneration of the cortex, or cheesy metamorphosis of an entire hemisphere of the cerebellum. Calcification of cerebral tubercles is not frequent, and I have observed only two cases.

Careful examination clearly shows that the large tubercles have developed from an aggregation of smaller nodules. Their internal structure, apart from the fissures already mentioned, is partly firm and homogeneous, partly granular and friable. The external layer is often formed of a narrow, grayish white, translucent zone, in which numerous miliary nodules can be demonstrated, by whose confluence the larger nodules appear to have developed, although this is due in part to a chronic cheesy encephalitis. Smaller tubercles are not infrequently encapsulated in a thin layer of connective tissue, while the larger ones are usually more diffuse and are embedded in very vascular, moist, softened brain-tissue. The number of cerebral tubercles varies greatly; most rarely only one is found (solitary tubercle), usually several in various parts of the brain,

[1] Charité-Annalen, Jahrg. IV., 492 ff.

occasionally even a dozen or more. In the majority of cases, tubercular meningitis is also present, associated with an accumulation of serum in the ventricles. I have repeatedly observed that the collection of miliary tubercles in the pia mater was most marked in the immediate vicinity of the cheesy nodules, especially at the convexity. It is usually, though not always, accompanied by more or less advanced tuberculosis and cheesy degeneration of other organs.

The question whether we are able, from the symptoms, to diagnose the situation of the tubercles in this or that portion of the brain, does not strictly belong here, as the conditions are the same as in adults. I therefore refer you to an article published by me in the fourth report of the *Charité-Annalen*, from which it appears that, despite recent experimental investigations, the regional diagnosis of tubercles still has an extremely feeble basis. However, I know of three cases in which a solitary tubercle of one frontal lobe produced irritative or paralytic symptoms on the opposite side of the body, and we may infer that these symptoms *can* be produced by the exclusive affection of the parts mentioned. I purposely say "*can*," as it does not follow necessarily. I have often seen the same symptoms, hemiplegia and contractures, in cases in which the autopsy showed that these portions of the cortex were entirely intact, while various other parts of the cerebrum or cerebellum were the site of the tubercles. I therefore advise you to be very cautious in making a regional diagnosis. It would be useless to enter more fully here into certain cases of solitary tubercles which have been employed to draw conclusions with regard to the functions of this or that part of the brain, as we would constantly encounter serious inconsistencies. In my opinion their solution can only be effected by an exact knowledge of the course of the pyramidal track, since motor disturbances will arise as soon as a part of these fibres are affected by an irritating or paralyzing lesion ; it is immaterial whether this lesion affects the fibres as they start from the cortex or in any other part of their course.

An approximate regional diagnosis seems to me most possible with reference to the pons Varolii and corpora quadrigemina. The simultaneous paralysis of one or more motor oculi, optic, facial, or abducens nerves, which either constitutes the chief feature of the symptoms or at least precedes the hemiplegia, throws a heavy weight into the scale in favor of this regional diagnosis.

It now remains to say a few words with regard to a not infrequent sequence of cerebral tuberculosis, viz., chronic hydrocephalus. Experience teaches that tubercles situated in the middle lobe of the cerebellum, or between it and the tentorium, are especially apt to produce effusion into the ventricles from pressure upon the vena magna Galeni and its chief branches. This may be manifested during life by an increase in the size of the head, even after the sutures have closed. My first case of this kind occurred in a girl three years old, in whom the symptoms of cerebral tuberculosis were followed by enlargement of the head with diminution of intelligence and double amblyopia, due to neuro-retinitis with marked swelling of the papilla and tortuosity of the veins. In the absence of an autopsy, it is undecided whether the compression of the veins was due to a tubercle or to another variety of tumor. The following case, observed in my clinic, is a significant one :

C. G——, aged three, formerly healthy. Gradual enlargement of the head for the past six months, associated with slowly increasing right hemiplegia. The latter not so

marked now as formerly; pertussis during the past seven weeks. Admitted to the clinic, January 4, 1879. Circumference of the head, 54 cm.; fontanelles wide, tense, and elastic; eyes prominent; somnolence. Diffuse bronchial catarrh, fever increasing in severity, death on January 15th. Autopsy.—Very marked chronic hydrocephalus ventriculorum, with compression of the brain-substance, flattening of the convolutions, dilatation of the skull. The left hemisphere of the cerebellum converted almost entirely into a homogeneous, yellowish white cheesy mass, surrounded by a narrow zone of normal brain-tissue.

This tubercular mass had undoubtedly been latent for a considerable time before it produced hemiparesis and gave rise to stasis by pressure upon the veins. It is not, therefore, absolutely necessary that the nodule be situated in the middle in the direction of the vena magna, since a tumor situated to the right or left may lead to stasis in the neighboring veins by the lateral pressure. But it is questionable whether the mechanical interpretation of chronic hydrocephalus as a consequence of compression of the veins is alone correct in all such cases, or whether an irritative condition of the pia mater, conveyed by the tela choroidea to the ependyma ventriculorum, cannot also be adduced as a cause of the serous accumulation.

As a matter of course, there can be no effective treatment of cerebral tuberculosis. But the possibility of spontaneous recovery, especially of a solitary tubercle by encapsulation or calcification, must be conceded, and you should therefore endeavor to further this as much as possible by tonic treatment (iodide of iron, cod-liver oil, salt-water baths, fresh air, nutritious food). Temporary improvement should not lead us to assume a successful termination; the case is entirely hopeless as soon as the first positive signs of tubercular meningitis develop. Epileptiform attacks, with or without febrile symptoms, which suddenly develop during the course of the disease and are followed by coma or partial paralysis, are suspicious because meningitis not infrequently begins with these symptoms under such circumstances; but it should be remembered that they may also be due to a sudden hyperæmia—a circumscribed encephalitis in the immediate vicinity of the tubercles; leeches to the head, ice-applications, and purgative remedies (P. 7), should then be ordered. The threatening symptoms sometimes subside under this treatment until, after a certain lapse of time, a new attack or terminal tubercular meningitis puts an end to life.

XIII. Tumors of the Brain.

I shall discuss this subject very briefly, since the tumors of the brain are similar in all respects to those of later life. Various forms of sarcoma occur most frequently, growing either in the substance of the brain, especially in the pons Varolii and its vicinity, or developing from the cranial bones and exerting pressure on the brain.

A. G——, aged six, admitted to the hospital July 16, 1874.[1] Severe headache for some months, especially in the left forehead; double amaurosis for the past six weeks. Examination showed incomplete left ptosis, complete immobility of the left eye, pupil wide and destitute of reaction. Right eye movable normally, pupil dilated. Neuroretinitis on both sides. Occasional pain in the left nares, with gray purulent secretion. July 24th, child was attacked with severe scarlatina; death on August 2d. Autopsy.—A myxo-sarcoma, half as large as a fist, growing from the bones of the middle fossa,

[1] Charité-Annalen, Jahrg. 1, 561.

filled this completely, perforated the lamina cribrosa, and grew into the upper part of the left nares; it surrounded the optic chiasm and all the ocular nerves of the left side. Brain and meninges normal.

The occurrence of gummy tumors in the brain during childhood is occasionally mentioned, and indeed there is no reason known for the exemption of childhood from this manifestation of syphilis. But I would call your attention to the fact that the differentiation of these tumors from tubercles is often very difficult—even the microscope may leave us in the lurch—so that many cerebral tubercles may have been mistaken for gummata, and vice versa. In such cases a cheesy condition of other organs, especially the lungs and bronchial glands, decides in favor of tuberculosis. If other signs of syphilis are not present at the same time, and the entire absence of tubercles in other organs is determined by a very careful post-mortem examination, I would be cautious in making an anatomical diagnosis of cerebral gummata in children, on account of the enormous preponderance of tubercles at this age. I have observed, to my knowledge, only one positive case, which was reported ante (page 46).

Other varieties of tumor (glioma, medullary sarcoma, echinococci, cysticerci), which occasionally occur in the brains of children, present no more characteristic features than the localized encephalitic processes which terminate in softening of the brain-substance or in abscess-formation. The latter is found not very rarely at this age, since a frequent cause, viz., traumatism, is more common at this period than in later life. In addition we must also take into consideration the relatively greater frequency of caries of the petrous portion of the temporal bone, whose tendency to produce cerebral abscess is well determined. My personal experience only refers to the previously mentioned combination of cerebral tubercles with the osseous affection in question. On the other hand, I observed, in a scrofulous girl twelve years of age, a colossal abscess of the brain, implicating almost the entire anterior lobe of the right hemisphere, in connection with caries of the lamina cribrosa of the ethmoid bone. In this case severe paroxysms of neuralgia in the region of the right supra-orbital nerve had existed for weeks, and could only be alleviated by injections of morphine, while the intervals were almost entirely free from morbid symptoms, and only pressure upon the upper rim of the orbit, especially toward the nasal side, gave rise to pain. Severe epileptiform attacks, coma and hemiplegia suddenly developed, and terminated fatally after the lapse of a few days. You thus see that diseases of the nares (chronic rhinitis) in children, especially if they are scrofulous, must be treated with no less care than those of the ear, whose dangerous terminations have long been recognized.

XIV. Atrophic Cerebral Paralysis.

Like infantile spinal paralysis, the cerebral form may also be prolonged into a late period of life, and then come under the observation of the physician; but this occurs much more frequently in childhood, during the first years of life. The children in question present the symptoms of a more or less complete hemiplegia, with or without implication of the facial and other cerebral nerves. The movements of the upper limb are usually more affected than those of the lower, which can often be used in walking, but is dragged after. The paralysis is either congenital, or de-

velops shortly after birth, about the age of three to six months—usually after a febrile and comatose preliminary stage, and more or less severe convulsions, similar to the rare hemiplegic form of infantile paralysis. This similarity increases with time, as contracture and atrophy of the paralyzed parts gradually develop, so that they finally appear not alone more emaciated and flabby than the healthy parts, but also shorter and withered. It is distinguished from infantile spinal paralysis by the hemiplegic symptoms, and by the long continuance of the electrical contractility of the paralyzed muscles, which disappears only after the atrophy has become extreme, *i.e.*, when normal muscular tissue is no longer present. But this rarely occurs, the atrophy of the limbs in the cerebral form develops only slowly, after the lapse of years, and hardly ever attains the intensity which is so often present in infantile paralysis. Sensory disorders are rarely observed, but the development of speech and the intelligence usually suffer more or less, and all gradations between slight dulness and idiocy may be present. The symptomatology is very often completed by the association of epileptiform attacks. As already mentioned, these children may attain an age of twenty years or more, but they usually die earlier, either in a convulsive seizure, coma, or as the result of an accidental complication.

The incurability of this condition is founded upon its anatomical relations; there is atrophy or complete absence of certain parts of the brain —for example, a portion of the convolutions of one hemisphere, a half or an entire lobe, the basal ganglia, etc., which are then compensated by an accumulation of serum, and often by thickening of the cranial bones. I described one of the most exquisite cases of this kind in my inaugural dissertation.[1]

A girl, aged nineteen, born healthy; convulsions at the age of three months, followed by right hemiplegia. Atrophy of the paralyzed limbs; sensation normal; cerebral nerves unaffected. Flexion of fingers. Mental condition almost idiotic. Death from phthisis. Autopsy.—Left half of the skull one-half inch thinner than the right, left frontal bone thickened. Middle and upper part of the left hemisphere entirely absent, and replaced by a cyst filled with serum, extending to the lateral ventricle, which is markedly dilated and filled with serum. Corpus striatum and optic thalamus half the normal size. This atrophy is continued in a crossed direction, so that the optic tract, eminentia mammillaris, crus cerebri, and pons of the left side, and the pyramid of the right side, are much diminished in size.

In all these cases we find atrophy of the pyramidal fibres, due to regressive metamorphosis (degeneration into granular corpuscles); this starts from the atrophic portion of the brain and crosses over into the opposite half of the spinal cord. But we are still deficient in a clear insight into the nature of the disease proper. Cases like the one just reported may be congenital, and they must be due to an encephalitic or hemorrhagic process, occurring either in fœtal life or a few months after birth, and completely destroying the affected portion of the brain. In consequence of a reactive inflammation of the immediate neighborhood, a cyst-like encapsulation of the destroyed portions of brain occurs after a lapse of time; the latter gradually become fatty and are absorbed, until finally the cyst contains more or less clear serum. In other cases more or less different changes are observed, but their development may be interpreted in the same manner.

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[1] Henoch: De atrophia cerebri. Berolini, 1842.

E. R——, aged twelve, admitted to the hospital, January 8, 1879; has had, at irregular intervals, repeated epileptiform attacks since early childhood; has had right hemiplegia as long as she can remember. The seizures observed in the hospital were epileptic in character, affecting chiefly the paralyzed limbs, the head, eyes, and right facial nerve. The paralyzed arm could be used very little, was moderately atrophied, and slightly flexed at the elbow. Death occurred from phthisis. Autopsy.—Pia mater very œdematous over the convexity of both hemispheres; convolutions of left side very small and narrow, sulci very deep. Pia mater thickened and removed with difficulty from the sulci, between the second and third frontal convolutions and the central fissure. These extremely small gyri showed a depression and rusty brown color, evidently due to previous hemorrhages. Right side of the brain normal. The depression referred to is filled with serous fluid, which is covered by the arachnoid.

In this case a primary failure of development (smallness of the convolutions of the left convexity) appears to have become associated later with a hemorrhagic exudative affection of the meninges. Atrophy and depression of the frontal convolutions, replaced gradually by serum, were produced by the pressure of the exudation. This case also furnishes evidence of the fact, which has been mentioned previously (page 106), that affections of the convolutions in question may produce irritative and paralytic symptoms of the opposite half of the body.

Bilateral atrophy of the cerebral substance, which may give rise to morbid symptoms on both sides of the body, are much more infrequent than the cases previously described.

A boy[1] aged six; admitted to hospital, July 20, 1874; had measles when six months old. Soon after had "spasms" frequently repeated for a week, then more infrequent, and finally they occurred only rarely. The present affection is said to have developed immediately after the first attack of spasms. True paralysis was not noticeable, but a wide-spread rigidity of the muscles. In the prone position both lower limbs were rigid, with slight flexion at the knee; flexion and extension were performed with difficulty, on account of tension of the flexors and extensors. The upper limbs, especially the right, were flexed at the elbow; extension proved very difficult, and could not be performed at all by the patient. As soon as the boy was placed on his feet and asked to walk, rigid contraction of the calf-muscles immediately occurred, with pes equinus position of the feet and strong dorsal flexion of the toes, so that standing and walking were absolutely impossible. Slight contracture of the joints of the hands and fingers; choreiform movements on grasping objects. Speech stuttering, labored, understood with difficulty; mental activity very feeble. Death from diphtheria. Autopsy.—Two and one-half centimetres' shortening of the right arm from the axilla to the styloid process of the radius, and atrophy of the muscles. Calvarium shows slight asymmetry, the right parietal bone being more arched and larger than the left, and its diameter (from before and toward the left to behind and toward the right) larger than the corresponding one on the opposite side. Dura mater normal. Pia mater thickened over the frontal lobes on both sides of the incisura magna, opaque, and raised like a vesicle by clear fluid, after the removal of which the corresponding portion of the brain appeared depressed. The first, and in part the second, frontal convolutions on both sides were atrophic, the gyri scarcely one-third as broad as the normal ones, very soft, and of a uniform grayish red color on section. The adjacent part of the white matter was similarly constituted and atrophic. The third frontal convolution very slightly affected; insula normal. Corpus callosum, fornix, and septum lucidum considerably atrophied. Lateral ventricles markedly dilated and filled with serum, especially the anterior horns, which were larger than the lateral ventricles and posterior horns combined. Ependyma very thick, firm, finely granular.

Many cases of "spastic spinal paralysis," upon which an autopsy has not been obtained, may be due to such bilateral deficiencies of brain-substance, especially those which are associated with weak or entirely absent intelligence (page 99).

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[1] Charité-Annalen, Jahrg. I., S. 567.

Microscopic examination of the atrophic gyri in these cases showed a sclerotic process, *i.e.*, destruction and finally disappearance of the true nerve-elements, in whose stead there was interstitial hyperplasia of the neuroglia, and granular corpuscles were also present with more or less numerous corpora amylacea. Hæmatoidin crystals also often furnished evidence of the original hemorrhagic process. These sclerotic atrophies therefore appear to represent the terminations of encephalitic-hemorrhagic processes occurring in fœtal life or early infancy, with which an exudative inflammation may be associated afterward as a compressing factor. The very rare cases of cerebral sclerosis in children, affecting not only the cortex, but other parts of the brain, also belong to this category. The cases mentioned show that the prognosis must be absolutely bad and treatment entirely useless. If we desire to do something, nothing remains but the employment of electricity, which may, perhaps, stop the muscular atrophy better than in infantile spinal paralysis. Friction of the limbs, stimulating baths, and gymnastics, are also in place.

XV. Chronic Hydrocephalus.

The only certain indication of this disease is the more or less rapidly increasing enlargement of the head, which is caused by the growing pressure of fluid filling the ventricles. Slight grades of hydrocephalus, in which enlargement of the head is absent, cannot be diagnosed. More than one hundred grammes of serum may be found in the dilated ventricles of children who have died from various cachectic, especially tubercular affections, although no signs of this condition have been present during life. These cases will not be discussed.

On the other hand, an unusually large size of the head should not lead us forthwith to a diagnosis of hydrocephalus. Children have often been brought to me who had been declared hydrocephalic by the parents, because the head was large, the fontanelles and sutures not closed; and yet I could soon assure the parents that their fears were unfounded—that not hydrocephalus, but a rachitic skull, was present. I acknowledge that the diagnosis is occasionally difficult, if we take the size and impeded ossification of the skull alone into consideration; but a careful examination of the intelligence, the movements, and the glance, will soon enable us to decide. The diagnosis will be doubtful only for a time in those cases in which rachitis is combined with hydrocephalus.

The majority of children suffering from chronic hydrocephalus come under medical observation during the first six months of life, as the constant increase in the size of the head, which does not keep pace with the growth of the rest of the body, soon attracts attention. At first the increase in size is not very marked, so that we may be tempted to deny it altogether; but it can soon be recognized by measurement, with a leathern centimetre measure, of: 1st, the circumference of the head[1] (the glabella and occipital protuberance being taken as the median points); 2d, the transverse diameter (from one mastoid process over the vertex to the other process); and 3d, the antero-posterior diameter (from the root of the nose,

[1] In the new-born the circumference measures, on the average, thirty-nine to forty centimetres; from six to twelve months about forty or forty-five centimetres, and then gradually increases to fifty centimetres, which it reaches about the twelfth year (Steffen).

over the vertex, to the occipital protuberance). An increase of one centimetre or more may thus be shown every week or two. Most hydrocephalic skulls are characterized by marked prominence of the frontal bones and lateral protrusion of the parietal bones, which is especially distinct when viewed from above. I have exceptionally observed a dolichocephalic form—*i.e.*, an elongation of the long diameter, with lateral flattening of the skull—in combination with enormous hydrocephalus. The subcutaneous veins are almost always found dilated into bluish cords. Palpation of the skull always shows cessation of ossification; all the fontanelles, especially the large ones, are widely opened, the sutures gaping so that the fibrous membrane stretched between the bones is forced outward by the pressure of the fluid contents, and is felt to be elastic and more or less distinctly fluctuating.

Occasionally, but only in very marked congenital or early developed cases in which the formation of bone was still extremely scanty, I could feel scattered osseous nuclei in the midst of this fluctuating membrane, which connected the cranial bones with one another; and in one of these cases the membrane immediately above the squamous process of the occipital bone, was protruded into a round diverticulum as large as a walnut, which, as was shown by an exploratory puncture, was filled with fluid, and therefore regarded as a form of meningocele. The opinion that external hydrocephalus—*i.e.*, an accumulation of fluid beneath the dura mater—was also present in this case, was confirmed upon autopsy.

On account of the increase in the size of the head, it gradually becomes so heavy that the children are unable to hold it erect; without assistance it follows the law of gravity, and totters from one side to another. The large size of the head contrasts with the small face, which becomes smaller and almost triangular on account of the increasing emaciation. A striking feature is the peculiar fixed stare, or the direction of the eyeballs downward, so that the iris is half-covered by the lower lid, and a large part of the upper section of the sclera is constantly visible. The former view, that this position of the eyes (which is, however, by no means constant) was due to downward pressure of the orbital plate of the frontal bone, is untenable, because exophthalmus could alone be produced by such a cause. It is evidently due to partial paralysis of the motor oculi communis, viz., of those branches which supply the superior rectus, the rectus inferior thus becoming predominant. Paralysis of other branches of this nerve frequently occurs, giving rise to diverging strabismus or other abnormal positions of the eye, or to more or less marked ptosis. The gaze and position of the globes are rarely entirely normal. The ophthalmoscope usually reveals atrophy of the optic disc from pressure and dilatation of the retinal veins, on account of the retardation of the return flow of blood in the cavernous sinus, brought about by the compression. The development of the intelligence is deficient in the majority of cases. The children are very apathetic, appear neither to see nor hear distinctly, do not know those around them, and not infrequently present a picture of complete idiocy, with the saliva running from the half-open mouth, and macerating the integument of the lower lip and chin. But this is not always true, and the degree of intelligence present in well-developed cases of hydrocephalus is sometimes astonishing. I observed a child, aged a year and a half, with very marked hydrocephalus, who recognized those around him, said "papa" and "mamma," and followed with the eye all objects held before him. Even a few weeks before death, which occurred in violent convulsions, sight was perfectly preserved, the child

spoke as well as before, and recognized its mother. I have repeatedly seen similar cases, and they contain a warning not to attach too much importance, in the diagnosis of chronic hydrocephalus, to an entire abeyance of intelligence.

As a rule, the mobility of the upper limbs is not much affected, but we often notice that the children, on attempting to grasp an object, make unsuitable movements which bear a remote resemblance to those of chorea. Paraplegia is almost always present; the legs are either completely paralyzed, or are, at least, unable to carry the body; the children are often unable even to sit without support. But there are also exceptions to this rule, and the literature contains cases in which the mobility of the lower limbs was almost entirely preserved. The disease is often associated with convulsive seizures of various kinds—spasmus glottidis, rolling of the eyes, twitching of the body with a tendency to fall forward ; finally, general epileptiform attacks or contractures. All the animal functions—respiration, circulation, and digestion—may be normal for years, but nutrition suffers considerably, and the children finally fall into an atrophic condition, which shows the size of the head so much more markedly on account of the contrast with the rest of the body. Although the majority of these children die in a few years from atrophy and marasmus, or in a convulsive seizure, the prognosis of the duration of the disease should be made with caution. Apparently desperate cases not infrequently reach the age of five to six years, or more; and cases are not lacking in which the disease was prolonged into the period of youth, or even later.

Post-mortem examination shows more or less marked attenuation of the cranial bones, due to the pressure of the distended brain; this can often be detected by palpation during life. In a child, nine months old, whose intelligence was not appreciably affected, and who showed no signs of paralysis, I found this attenuation and disappearance of the diploë so marked as to cause transparency, so that the color and the blood-vessels of the dura mater could be distinctly seen through the bones. The fontanelles and sutures gape widely; the diastasis of the latter is closed by fibrous membranes of a finger's breadth or even broader, containing osseous nuclei. The cerebrum consists of two more or less flaccid, flapping sacs, and the enormously dilated ventricles, filled with serous fluid; these are surrounded by a condensed mass of brain, occasionally only a few centimetres in thickness. The fluid averages one hundred and fifty to two hundred grains in amount, but may measure one litre or more; it contains no albumen, or but a very small quantity. The boundaries of the white and gray matter can still be distinctly recognized in the surrounding rind, into which the substance of the hemispheres has been compressed. The gyri, as well as the basal ganglia, are flattened by the pressure. The third and fourth ventricles are also often dilated and filled with fluid; and I even saw the fifth ventricle, on two occasions, take part in the dropsical dilatation. The central portions (corpus callosum, fornix, etc.) are almost always unusually firm, if the brain is examined fresh. The ependyma generally presents a finely granular surface, and seems to be dusted with extremely small, gray, translucent granules, which appear under the microscope as hyperplasia of the ependyma. In rare cases flakes of fibrinous exudation are found occluding the foramen of Monro, thus preventing communication between the ventricles. These changes naturally vary very much in intensity; the dilatation of the ventricles and the thickness of the compressed masses of brain are especially variable. The following case will serve as an example of an extreme grade:

A. P——, aged five months, admitted March 26, 1877, with chronic hydrocephalus. Circumference of head, 45 ctm.; long diameter, 25 ctm.; transverse diameter, 27 ctm. Eyeballs directed downward. All functions—nursing, etc.—performed normally. April 3d, collapse, broncho-pneumonia; April 7th, death. Autopsy.—The cranial cavity is entirely filled with fluid, an elongated lump found in its lowermost part constituting the remains of the brain. On close examination it is found that the hemispheres have almost entirely disappeared. Under the normal dura mater appear, in spots, plates and bands as thin as paper, covered with something looking like the pia mater—these constituting the sole remains of the hemispheres. The shapeless mass situated at the bottom of the cranial cavity consists of the remains of the basal ganglia, to which the cerebellum and spinal cord are attached in a normal manner. These parts and the cerebral nerves and vessels are entirely intact.

Although the compression of the hemispheres had progressed to their almost complete disappearance, all functions were performed in a normal manner, and the entire conduct of the child did not differ in any respect from that of a healthy infant of similar age. There was no question of the persistence of a psycho-motor centre, and the case therefore furnishes a clinical proof of the theory that all actions of the new-born must be regarded as involuntary (reflex, automatic).

We are not altogether clear with regard to the pathogenesis of chronic hydrocephalus. There is no doubt that it is sometimes congenital, and it may then constitute a serious obstacle to delivery. In these cases we sometimes find various kinds of failure of development; absence of the corpus callosum, fornix, etc.; also spina bifida, club-foot, club-hand, etc. Much more frequently, however, the children are apparently healthy at birth, and only after the lapse of a few months does the unusual increase in the size of the skull attract attention. To what is this due? The peculiar granular, hyperplastic condition of the ependyma, which may even be removed, at times, in tough strips from the walls of the ventricles, favors the theory of a chronic inflammatory condition of the ependyma, which begins either in fœtal life or soon after birth, and occurs so gradually that the dilatation of the head by the constantly increasing pressure of the ventricular fluid constitutes the first sign of the disease. This theory will not explain all cases of chronic hydrocephalus, as the granular condition of the ependyma may be absent, and with it all reason for supposing an irritative process within the ventricles. Causes of compression (tumors), which were referred to above (page 106), are found in only the smallest proportion of cases—most rarely in the congenital or early developed cases, and it then only remains for us to take advantage of the unsatisfactory assumption of a fault in development, an excessive secretion of cerebro-spinal fluid. The adherents of the inflammatory theory rely upon the not frequent cases of hydrocephalus developing in older children from the second half of the first year, after previous meningitic symptoms. I have also observed a few cases of this kind which alone possess any validity as proof when the autopsy shows that the ventricles were the site of the fluid, and also shows the change in the ependyma. If this is not done, we remain in doubt whether we really have to deal with hydrops ventriculorum or with a collection of serum between the meninges (hydrocephalus meningealis s. externus).

French authors have described this hydrocephalus meningealis as the second stage of hemorrhage into the arachnoid sac. According to our present views this is not a true hemorrhage, but an inflammatory process upon the inner surface of the dura mater (pachymeningitis) associated with extravasation of blood, which occurs not very rarely in children in a moderate degree and extent, though not as often as in old people. I have

found, under different conditions, more or less thick, fibrinous, hemorrhagic deposits upon the inner surface of the dura mater, with a larger or smaller collection of reddish serum between it and the arachnoid, although no distinct symptom-complex during life led to a suspicion of such an affection; experience teaches that all symptoms may be absent even in very severe cases.[1] But I have never observed a case of pachymeningitis upon the post-mortem table, which corresponded to the observations of the French authors, *i.e.*, in which the fluid had produced hydrocephalus by pressure upon the cranium. Whether the following case must be regarded as a "pachymeningitic" hydrocephalus cannot be determined, since recovery occurred.[2]

P. W——, aged three years and two months, presented February 14, 1861; previously healthy. Pains in the head and neck for the past eight weeks, tendency to retroversion of the head, irregular fever at night, emaciation. Examination showed: impossibility to hold the head erect, retroversio capitis, pain in neck on pressure and motion, frontal pains; inability to stand or walk, but no paralysis. Moderate fever in the afternoon; pulse, 96°–100.° Slight improvement until February 19th. February 20th, an exacerbation, with vomiting; severe pain in the neck and forehead, and marked retroversion of the head. Slight improvement under quinine, but vomiting, grinding of teeth during sleep, and incontinence of urine, made their appearance. March 22d, the fever and attacks of pain had entirely disappeared; the head could be moved more freely, but was enlarged, and examination showed diastasis of the parietal bones. These symptoms increased daily, so that on March 26th the child wore his father's hat. The sagittal suture gaped, the mother remarking that it had already been closed in the second year of life; feeble pulsation over the former fontanelle. Intelligence normal, the right arm weaker than the left. April 16th, diameter of the head unchanged, but more freely movable; right arm again readily movable; general condition unimpaired. June 11th, every trace of the disease had disappeared, except the enlargement of the head. The sutures showed beginning ossification. I again saw the child in May, 1863, and he was then perfectly healthy; sutures ossified.

There is no doubt in this case that an accumulation of serum occurred in the cranial cavity, but I must leave it undecided whether it occurred into the ventricles or between the dura mater and arachnoid. But it appears to me to be a very important fact that, as late as the third year, and after complete ossification of the sutures and fontanelles, a strong intracranial pressure was capable of separating them. I also observed this symptom, but confined to the coronal suture, in a seven-year-old boy whose hydrocephalus, associated with considerable increase of the size of the head, had developed two years previously, after a fall. Perhaps this very circumstance, by which the brain is relieved from a part of the pressure, must be regarded as favorable, inasmuch as it prevents the development of severe cerebral symptoms. Our case, moreover, shows that absorption of a very large accumulation of fluid and final recovery is possible, if the cause of the affection is of a "meningitic" character, since we cannot conceive in what manner the empty space could be filled which would be left after absorption of fluid collected in the ventricles.

Under such conditions the treatment in the beginning of the affection should consist of local bleeding, by means of a few leeches applied behind the ears or upon the forehead, ice-fomentations to the head, and purgatives, preferably calomel (0.015–0.03 a few times a day). At a later period, when the inflammatory stage has passed and we wish to initiate the absorption of the fluid, I would advise the continued use of mercurials in small doses, inunctions of unguent. cinerit. on the head and neck (1.0

[1] Moses: Jahrb. f. Kinderheilk., VI. 1873.
[2] Beitr. zur Kinderheilk., N. F., S. 28.

every day), and a blister to the neck, to the application of which a great fear is now unjustly entertained. But the antiphlogistic method is applicable only in those cases in which the primary inflammatory stage has either been directly observed or can, at least, be proven to have been present. On the other hand, I always regard internal chronic hydrocephalus as incurable, as soon as the head has increased in size to any considerable extent. At least I have achieved no results whatever, either with mercurial inunctions, iodide of potassium, or application of tincture of iodine to the head, nor can I promise any results from compression of the skull with strips of adhesive plaster, or from puncture through the fontanelles (to one side of the median line). When these methods of treatment have proven successful (this has happened in very rare cases) the case must have been one of external hydrocephalus. You may satisfy your desire for an operation, as the danger of the production of meningitis is not very great; but it is well to renounce, from the start, the hope of a radical success.

This appears to me to be the most suitable opportunity to say a few words with regard to acute hydrocephalus. By far the largest majority of cases described under this head are of inflammatory origin and due to tubercular meningitis. With the exception of these cases, there are very few in which a rapid effusion occurs into the ventricles or between the meninges. Many children, indeed, present upon autopsy serous effusion, with moderate dilatation of the ventricles, which, to judge from the symptoms, could only have developed within a very short period, and thus acute hydrocephalus is especially presented by children suffering from miliary tuberculosis, Bright's disease, and scarlatinous dropsy. But such cases cannot be diagnosticated with certainty, as the same symptoms may be produced by a not infrequently occurring œdema of the pia mater and brain, without any effusion into the ventricles. Coma, convulsions, fatal termination in a few hours or days, are not sufficient to form a new affection, as has been attempted by Gaelis. It is better to acknowledge our ignorance, and to admit that we may suspect acute serous effusions in the central organ, whether into the ventricles, between the meninges, into the pia mater or the brain-substance, according to the circumstances under which the patient dies, but can never diagnose this condition with certainty from the cerebral symptoms just mentioned.

XVI. Hyperæmia of the Brain.—Thrombosis of the Sinuses.

Autopsies prove that the amount of blood in the brain of children varies greatly, and that there may be every possible gradation, from very slight filling of the vessels of the pia mater, and a pale, anæmic color of the gray matter, to the finest injection of the vascular net-work, and numerous puncta vasculosa on section; but the attempt to connect these various vascular conditions with distinct symptoms is almost always fruitless. We must always remember that the hyperæmia found on post-mortem examination may be the result, as well as the cause, of fatal cerebral symptoms—for example, very severe and prolonged convulsions. Even purely reflex spasms must finally lead to passive congestion of the cerebral veins on account of the interference with respiration, and this may finally terminate in œdema of the pia mater or brain, and in serous effusion into the ventricles or arachnoid cavity.

Hyperæmia of the brain and its membranes may be due to increased

circulation in the arteries or to stasis of venous blood. The first form may be looked for in hypertrophy of the left ventricle and in the preliminary stage of inflammatory processes (meningitis). In addition, local foci of irritation (tubercles or tumors) appear to be able to produce "meningitic" symptoms (fever, vomiting, somnolence, convulsions) by exciting hyperæmia in their immediate neighborhood, which rapidly disappear either spontaneously or under antiphlogistic treatment, and, when often repeated, may lead to inflammatory softening or to encapsulating proliferation of connective tissue. Thus far we stand upon the firm basis of pathological anatomy. But we not infrequently meet with cases which can hardly be explained in any other manner than by arterial congestion of the brain, though its mode of development is not always clear and the anatomical confirmation is lacking. Among the causes which here come into consideration, traumatism is most frequent. Immediately after a fall on the head, the child may be dazed or entirely unconscious, but this is not constant. In three of my cases, the children were perfectly well immediately after the fall, and only after a few hours or days did the symptoms develop, viz., continuous headache, apathy, somnolence, yawning, change of color, restlessness at night, anorexia, repeated vomiting, and fever, the pulse being 140 to 160 per minute, but regular. One of these children suffered at the same time from nocturnal terrors. I will acknowledge that these symptoms may be due to a febrile gastric disturbance as well as to congestion of the brain, but their rapid development after an injury to the skull and, above all, the surprisingly rapid effects of antiphlogistic treatment, in my opinion render the diagnosis certain. The application of a few leeches behind the ears, whose bites were not allowed to bleed after removal, were sufficient to ameliorate the symptoms considerably. The dread of bleeding, which has gained ground at the present time, is not justifiable here. We can withdraw the blood directly from the cranial cavity through the emissaria Santorini, and we should not hesitate to do this, because the neglect of these prodromal symptoms may lead to true meningitis. At the same time an ice-bag should be constantly applied to the head, and calomel or inf. sennæ comp. and syrup. spin. cerv. (P. 7) given internally, in order to produce free evacuations. Under this treatment complete recovery occurred within thirty-six to forty-eight hours.

If we take into consideration that the symptoms of cerebral congestion after a fall on the head develop in only a relatively small number of children, we should suppose that individual disposition plays a part in the dilatation of the blood-vessels, in addition to the intensity of the concussion. In fact my cases occurred in children who had, shortly before, passed through an attack of whooping-cough or chronic pneumonia, or came from a tuberculous family. The formation of the skull must also be taken into consideration, since small children, with membranous fontanelles and sutures, appear to escape the evil effects of concussion more readily than older ones, whose cranial bones are entirely ossified.

In a smaller number of cases the symptoms of congestion develop without any recognizable traumatic cause, especially in children during the period of first dentition. These symptoms consist of fever, somnolence, frequent starts, inability to hold the hot head erect, tension and pulsation of the fontanelle, perhaps vomiting. I merely mention this without being able to prove the dependence of these symptoms upon dentition, but I would also remind you that severe congestion of the buccal mucous membrane, erythema and papules on the face, and catarrh of the con-

junctivæ and bronchi are often observed at the same time. Purgatives (small doses of calomel) and cold fomentations to the head usually suffice to remove these symptoms in a few days. But this does not always happen; the symptoms may gradually grow worse and assume a meningitic character by the development of spasms, retroversion of the head, and coma.

Finally, excessive mental exertion must also be mentioned as a cause of cerebral congestion in children. Although symptoms of another character develop more frequently under such circumstances, examples are not lacking in which symptoms of hyperæmia have developed after unusual mental effort. I have previously described the case of a boy, aged nine, who, after such a cause, suffered not alone from violent headache and photophobia, but also from dizziness, anorexia, nausea, sighing, constipation, pain in the neck, intermittent pulse, and staggering while walking. Emetics and quinine proved useless, but rapid improvement occurred after the application of five leeches and an ice-bag to the head, and the administration of purgatives.

The second form of cerebral hyperæmia, produced by mechanical stasis in the venous system of the central organ, is better understood anatomically than active congestion. Heart disease, with dilatation of the right ventricle, compression of the large venous trunks by swollen glands in the chest or neck, but especially thrombosis of the sinuses of the dura mater, may gradually produce this form of hyperæmia, and great weakness of the heart from exhausting diseases may produce it in a more acute form. In cases of the latter kind, anæmia is usually regarded during life as the cause of the symptoms. The weakened heart is indeed unable to force the normal amount of blood into the cerebral arteries, but the consequent slowness of circulation produces venous stasis, which finally leads to œdema of the pia mater and serous transudation into the ventricles. The symptom-complex, described by Marshall Hall, under the term "hydrencephaloid," is composed of the symptoms of arterial anæmia and venous congestion combined, and is characterized chiefly by increasing apathy and somnolence, half-closed lids, flattening or depression of the large fontanelle, opacity of the cornea by flakes of mucus or from dryness of the tissues, great weakness of the pulse and depression of temperature, especially in the extremities—symptoms which are due only in part to venous congestion of the brain, and also to heart-failure and general collapse. Persistent diarrhœa or very acute attacks of cholera infantum are especially apt to produce this symptom-complex.

In the treatment of such cases, our chief aim must be to stimulate the action of the heart. Repeated doses of wine (a spoonful of Hungarian, port, or sherry wine every one to two hours), warm baths (27° to 28° R.), with cold fomentations to the head, or a douche of cold water, are advisable. As a matter of course, the cause of the collapse (usually diarrhœa) must be appropriately treated. In many cases this has ceased when the cerebral symptoms first become noticeable, and cardiac stimulants may be administered at once. Among these camphor occupies the first rank (according to age, 0.03–0.05, every two hours, in powder or emulsion, P. 14). If this drug, or the use of wine, does not maintain the heart's energy, no effect can be expected from other remedies; musk and ammonia are almost useless. Milk, bouillon, and the yolk of egg stirred in wine, should be given at short intervals. Despite all our efforts, however, a large number of these children perish. Under these circumstances the retardation of the venous circulation not infrequently leads to complete stasis and coagulation of the blood in the large sinuses of the dura

mater (marantic thrombosis). The longitudinal sinus is most frequently, the other sinuses are more rarely, filled with more or less pale, firm thrombi, which can be traced for a greater or less distance into the veins emptying into them; they increase the venous stasis in the brain and pia, as well as the danger of serous transudation. The same effect is produced by every thrombosis of the sinuses, whether due to compression or to the spread of inflammation from the adjacent cranial bones. The petrosal and transverse sinuses are especially subject to the influence of the petrous portion of the temporal bone, when carious, and their thrombi sometimes extend far into the jugular veins. But this process may also develop without any visible change on the free surface of the dura mater. I have repeatedly observed that caries of the cranial bones, especially the petrous portion of the temporal bone, may attain a wide extent, even to immediately beneath the dura mater, without affecting the latter. It remains intact and glistening for a long time, and the adjacent petrosal sinus may nevertheless become the site of a thrombus, which may probably be explained by the detachment or projection of small thrombi from the osseous veins. The thrombosis of the sinuses occasionally observed in consequence of profusely suppurating eczematous eruptions of the scalp, is also explained in a similar manner (continuation of the thrombosis through the emissaria Santorini).

Great efforts have been made to render the diagnosis of thrombosis of the sinuses possible. In the diagnosis of thrombosis of the transverse sinus, or the beginning of the internal jugular, Gerhart and Huguenin attach special importance to the fact that the external jugular on this side appears more empty than on the opposite side, while in thrombosis of the cavernous sinus the stasis in the ophthalmic vein is shown by venous congestion of the fundus of the eye, slight exophthalmus and œdema of the lower lid or the entire side of the face. Despite careful examination, I could not satisfy myself of the certainty of these signs. However, these symptoms appear to me to possess more value in diagnosis than the signs of thrombosis of the pulmonary artery mentioned by authors. That this condition may be due to an embolus breaking off from a thrombosis in the sinuses is undoubted; but its diagnosis is so difficult, under these circumstances, that it is only in exceptional instances that it can be determined to be due to sinus thrombosis. Treatment of this affection is entirely useless.

XVII. Tubercular Meningitis.

This disease is one of the most frequent fatal affections of childhood. If positive signs of its presence are observed, the prognosis is always fatal. The incurability of this form of meningitis is indicated by the adjective "tuberculous;" it proves fatal by its combination with tubercles of the pia mater and other organs. It is not a local disease, but affects many important organs; in one word, it is a form of acute miliary tuberculosis.

A description of this disease is difficult, on account of the numerous variations in its course, and, despite the large amount of material at my command, I hardly dare hope to present to you a full account. It appears to me best to describe the ordinary "classical" form of the disease first, and to consider the different varieties later.

In many cases the real outbreak of the disease is preceded by a prodromal stage, which may last for weeks, and even months. The children

become emaciated, without any apparent cause being detected. The general condition is often undisturbed, while in other cases numerous disorders occur—capricious appetite, fatigue, varying moods, irregular febrile disturbance—thus constituting indefinite symptoms which the physician is unable to interpret positively, despite careful examination. These symptoms betoken the slowly progressing development of miliary tubercles in various organs, and the examination should always take into consideration, in such cases, an hereditary tendency to tuberculosis, the demonstration of which always constitutes a point of light in the obscurity of the symptoms. But we must not forget that such an hereditary tendency to tuberculosis is not necessary; that hyperplasia and cheesy degeneration of the bronchial and mesenteric glands may develop, in consequence of long-standing catarrh, whooping-cough, measles, typhoid fever, or repeated diarrhœa, and may finally act as foci of infection for miliary tuberculosis. Cheesy processes in peripheral lymphatic glands, or in the bones (spondylitis, osteomyelitis), may act in a similar manner. The prodromal symptoms mentioned above are not by any means constant; despite careful questioning, I have often been told by the mothers that their children were entirely well until the outbreak of the disease itself—and the blooming, well-nourished appearance of the patients testified to the correctness of these statements.

The outbreak proper of the disease occurs almost suddenly, with complaints of headache, especially in the forehead, and with vomiting, which is generally repeated a number of times during the first few days, and occasionally occurs whenever food or drink is ingested. Definite characteristics, to which I cannot subscribe, have been attributed to this vomiting. I have seen it occur in the upright as well as prone position—at times projectile, at others combined with retching—and can therefore recognize no essential difference in the manner of cerebral vomiting from that of purely gastric vomiting. But it is this very diagnosis which must first be taken into consideration. The symptoms during the first half or whole week are so similar, in many cases, to those of a mild gastric fever, that even experienced physicians, who have seen many of these children die, are not secure against error. The general apathy, the absence of any desire to play, the headache, tendency to support the head and to lie down; the more or less coated tongue, the loss of appetite, with vomiting and constipation; finally, the irregular febrile disturbance—these symptoms are of such an ambiguous nature that we may be doubtful whether we have to deal with beginning meningitis, gastric fever, or even the first stage of typhoid fever. Even the peculiar symptom, that the children are continually picking at the lips and nose, or rubbing the eyes, is common to all these conditions. As long as you are in doubt, be careful about attributing the symptoms to a "spoiled stomach," as the parents will never forgive the physician for the false prognosis.

But the indecision generally lasts only a few days, at least for the experienced physician. At the end of the first week, at the latest, more distinct symptoms arise. Among these I especially include oft-repeated deep sighing, which has hardly ever deceived me, and the characteristic change in the pulse, which becomes slower, irregular, and unequal in the force of the individual beats. Under the circumstances mentioned, I consider the symptom decisive, even though it is observed only temporarily. In scarcely any other disease of childhood is the pulse so variable as in this. Slight movements increase its rapidity by 20 beats or more, while variations in temperature have no effect. The frequency varies from 96

to 120, and may even go down to 80, 72, and less. But, however important this symptom may be, we must always remember that it may also occur in mild gastric affections from reflex irritation of the vagus. The slowness of the pulse is occasionally absent, and only irregularity is observed. But such cases are rare, and, when irregularity and slowness of the pulse appear in combination, you may rest assured of the development of tubercular meningitis. The heavy quality and vibrating character of the pulse (pulsus tardus), upon which stress has been laid by Rilliet and Barthez, I do not regard as characteristic, although I have repeatedly observed it, and more frequently, even, upon the large fontanelle than upon the radial artery. The slowness and irregularity of the pulse continues for several days, until about the middle of the second week, and then gives way to gradually increasing frequency, with regular rhythm. During this period the intensity of the previously described symptoms gradually increases. The headache is rarely so severe that the children cry out and press the hands against the forehead; many complain very little of headache, but of pain in the ears, the neck and abdomen, although examination reveals nothing abnormal in these parts. If headache is present, it is usually increased on coughing. Vertigo appears to be present occasionally, as the children think they are falling, even while sitting or lying down, and beg those around to support them. The apathy and somnolence gradually increase, being occasionally interrupted by restlessness, loud cries, or mild delirium. If the children are roused from this condition, the sensorium is found to be clear. The disappearance of childish obstinacy and indifference to the manipulations of the physician are bad signs, and may even possess diagnostic significance in doubtful cases. There is a noteworthy influence at this time upon certain secretory and trophic disturbances. Suppurating eczemas of the head or other parts not infrequently become dry, profuse secretion of the nasal mucous membrane ceases, and in two cases I observed enlargements of the cervical glands, which had existed for a long time, disappear in a few days under the influence of meningitis.

About the middle of the second week, or perhaps a little earlier, irritative symptoms in some of the cerebral nerves make their appearance in many children, though not in all; these are due to inflammatory irritation at the base of the brain, and consist usually of converging strabismus and grinding of the teeth. Whether the movements of mastication, which begin at this time, are characteristic of this disease and are to be regarded as due to irritation of the motor root of the trigeminus, appears to me to be doubtful. Slight retroversion of the head is occasionally observed at this time. The color of the face changes on account of fugitive redness. The somnolent condition gradually increases to coma, until finally the child lies perfectly unconscious, with half-closed lids, one leg extended, as a rule, the other flexed at the knee, the hands upon the genitals, sighing deeply from time to time, or giving utterance to a piercing cry ("cri hydrencéphalique" of Coindet). About this time the pupils become dilated, one being often larger than the other, and react slowly or not at all to light; the conjunctiva bulbi presents vascular injection and flakes of mucus near the cornea, and gradually opacity of the cornea, especially of that part which is not covered by the half-closed lids, and is thus continually exposed to the air. The reflex sensibility of the skin disappears like that of the conjunctiva. Automatic movements of the hands toward the head, pendulum-like movements of an upper or lower limb, rigid contracture of the muscles of the neck and mastication also

develop. Not infrequently there is rigidity of one side of the body. The constipation, which could with difficulty be hitherto overcome by purgatives, now gives way to involuntary, thin evacuations. The abdomen becomes sunken in the umbilical region, so that it finally looks like a tray with projecting borders of the ribs and iliac crests which allow the vertebral column to be readily felt. The frequency of the pulse begins to increase from about the middle of the second week, and may reach 180, 200, or even more beats; it becomes smaller and is felt with more difficulty. Respiration, whose implication had been previously manifested by the sighing inspiration referred to, usually presents the Cheyne-Stokes phenomenon in the last twenty-four to forty-eight hours, either in its classical or a somewhat different form. Thus, I have seen, after a respiratory pause of a quarter of a minute, a deep, sighing inspiration, followed by two to three superficial respirations, and then another pause. There may be only five to seven respirations per minute, and this fact, in combination with the extreme weakness of the heart, explains the cyanosis of the integument of the face, the visible mucous membranes, and the tips of the fingers and toes, which often develops at this period. In many cases the face appears dark red in the last few days, and the forehead and cheeks are covered by profuse perspiration; but I have rarely observed the eruptions (erythema and papules) mentioned by other authors. Epileptiform convulsions occur very often during the last twenty-four to forty-eight hours; they affect either the entire body or one side, but are occasionally confined to the facial muscles or to feeble twitchings in the limbs. Rigid contractures of the limbs and the muscles of the neck, and tremor, develop in many cases. You will do well to prepare the parents for the occurrence of terminal convulsions, although no spastic symptoms were observed previously. The agonal stage is always unusually long, and not infrequently extends over several days. In the midst of this condition the unconscious child sometimes regains its senses, turns the head toward its mother, and takes nourishment, or even grasps its toys. But the child relapses into its former condition in a few hours, and dies in convulsions or in collapse in two to three weeks, as a rule, from the first appearance of vomiting.

It now remains for us to briefly discuss the febrile conditions of tubercular meningitis. My investigations show that this disease possesses no characteristic temperature-curve, but that marked fluctuations occur during its entire course. The evening temperature is higher in greater or less degree than that in the morning, but exceptionally is somewhat lower. The temperature rarely exceeds 39.0°, and in many cases scarcely attains this height for a single day.[1] In the majority of cases, however, it rap-

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[1] I adduce a few curves as examples:

L. S——, aged one, admitted September 29, 1878:

	Morning.	Evening.
September 29th	38.0°	38.5°
" 30th	37.6°	38.5°
October 1st	37.6°	38.2°
" 2d	38.0°	38.0°
" 3d	37.6°	37.6°
" 4th	38.1°	39.0°
" 5th	38.1°	38.9°
" 6th	38.8°	39.0°
" 7th	40.0°	41.0°
" 8th	41.2°	Death.

H——, aged four, admitted April 6, 1878:

	Morning.	Evening.
April 6th	....	38.5°
" 7th	37.5°	38.0°
" 8th	37.2°	36.8°
" 9th	38.4°	38.5°
" 10th	36.8°	37.5°
" 11th	38.0°	38.1°
" 12th	38.2°	38.6°
" 13th	38.5°	11 P.M., 39.2°
		4 P.M., 39.8°
		6 P.M., 40.3°
		9 P.M., 41.8°—Death.

idly rises, on the day of death or the day before, to 40° and even 42.0°; almost always remains at this height until death, and rarely falls, shortly before the fatal event, to 38°–39°. This sudden rise of temperature cannot be regarded as a simple febrile exacerbation, because the fever plays a subordinate part during the entire course of the disease. Nor can it be attributed to the terminal convulsions or to accidental complications with inflammations of the respiratory organs, as I have seen it absent under such circumstances. I cannot explain this symptom, which occurs not only in tubercular meningitis, but also in adults who die from paralysis of the cerebral functions, except by the assumption of paralysis of the presumed moderating heat-centre, which is situated at the boundary of the brain and spinal cord. If this is paralyzed, the heat of the body, which is no longer moderated, must attain an unusually great height. An abnormally low temperature of 31°–28.0° occurs much more rarely, and must be explained by paralysis of the heat-producing centre.

I have not divided the disease into certain stages, because I regard all attempts at such division fruitless, whether based upon anatomical or clinical principles. Perhaps we could distinguish a stage of irritation and one of paralysis; but even this is not entirely justifiable, as we often see symptoms of irritation—for example, convulsions—during the last stage. If we consider those cases which run an abnormal course, and the numerous varieties, to which I shall soon refer, we will find that the division into stages is illusory, and should be abandoned.

The variations from the normal course are, in fact, so numerous in this disease that the certainty of diagnosis would be seriously endangered, if we should decide merely according to the standard. I have occasionally observed, for a period of ten to twelve days, a series of symptoms very similar to those of typhoid fever; or the little patients gave utterance, day and night, and almost continuously, to a piercing cry, and then suddenly fell into coma. The justly feared initial vomiting may be entirely absent, while in other cases it continues with great severity for nine to ten days, or longer, and associated with such mild cerebral symptoms that they entirely escape the attention of the physician, who sees the child once or twice a day. One of these children sat up in bed whenever I came, apparently taking part in everything around him, and busying himself with picture-books. The eyes were bright, there was no tendency to somnolence, and the obstinate vomiting alone disquieted the parents and physician; but the varying and irregular character of the pulse rendered the diagnosis positive.

When the vomiting is so obstinate, the children often complain of pain in the region of the stomach. Especially in children one or two years old, this persistent vomiting, occurring without any other threatening symptoms, appears to me to deserve careful attention, because it is readily interpreted as dyspeptic in character, until somnolence, strabismus, ptosis, and convulsions suddenly clear up the diagnosis. The obstinate constipation usually present is also an unreliable symptom. I have repeatedly seen cases which began with vomiting and diarrhœa, and were therefore regarded as cholera infantum, until, after a lapse of twenty-four to thirty-six hours, constipation set in, while the vomiting either continued or disappeared. Occasionally I have seen diarrhœa, due to old follicular or tubercular ulcers of the intestines, continue despite the development of meningitis. Instead of the ordinary "boat-belly," more or less marked meteorism is occasionally observed, usually due to a complication with chronic tubercular peritonitis. The rule regarding the

condition of the pulse (moderate acceleration in the first few days, then retardation and irregularity, and finally increasing frequency with regularity of the beats) holds only in the majority of cases. I have already (page 120) called your attention to the varying condition of the pulse, and will add that in three cases, I observed during the last stage, after epileptiform convulsions had occurred, a frequency of only seventy, ninety-two, and ninety-six beats.

According to Legendre, Rilliet, and Barthez, the symptomatology undergoes considerable modification according as the meningitis attacks an apparently healthy child, or one suffering from advanced tuberculosis or phthisis. Only in the first event is the "classical" course, described above, pursued; in the latter the disease is much more acute, like simple meningitis. I have had repeated opportunity of confirming these statements. I have most frequently observed a violent course, beginning with severe epileptiform convulsions, in cases which were complicated with tuberculosis of the cerebral substance, and I have repeatedly been able to diagnose this complication before the autopsy, although I was unacquainted with the previous condition of the child. But exceptions to this rule are not infrequent.

Pathological anatomy is unable to explain these variations in the course of the disease. The post-mortem appearances are apparently the same, whether the disease run a normal or abnormal course, and the differences must therefore consist in finer structural changes, which are demonstrable with difficulty. In support of this view, I will refer to the observations of Rendu,[1] who found thrombosis of the middle cerebral artery in a number of cases, in consequence of the surrounding tubercular inflammation; and also found small spots of softening in their area of distribution, to which the paralyses observed during life could be attributed. In a number of cases which ran an unusually violent course, I found the inflammatory products more marked at the convexity.

In the large majority of cases, however, the affection of the base of the brain is the characteristic feature of the disease. An opaque, greenish gray infiltration of the pia mater is found in the space between the optic chiasm and medulla oblongata, and surrounds the cerebral nerves, which it may thus irritate or paralyze. An œdematous infiltration extends into the vicinity, especially into the Sylvian fissures, and here are found more or less numerous gray or grayish yellow miliary tubercles, as large as a pin's head, or even smaller, which can be seen most distinctly when the pia mater is carefully drawn out of the sulci. The tubercles appear flat and soft, or hard and prominent, according as they are recent or old. Similar and often quite numerous miliary tubercles of the pia mater are found not infrequently in the choroid plexuses of the ventricles, on the convexity and inner surface of the hemispheres, the pia mater being often very opaque from serous infiltration, and strips of grayish yellow, puriform, or a more granular exudation are noticeable along the larger veins. I have very rarely found miliary nodules on the inner surface of the dura mater. The vessels of the pia mater are usually more or less injected, and, upon withdrawing them from the sulci, small particles of softened cortical substance are apt to remain adherent. Here and there are strips of adhesions between the arachnoid and dura mater, or a collection of serum between both membranes, and bloody suffusion

[1] Recherches Clin. et Anat. sur les Paralysies Liées à la Méningite Tuberculeuse. Paris, 1874.

of the pia mater. The brain-tissue is usually anæmic, rarely hyperæmic; the ventricles are considerably dilated by serous fluid; their walls, like the central parts of the brain, often, though not always, softened or converted into a creamy mass floating in the ventricular fluid. I found some small ecchymoses in a few cases, especially around the third ventricle. An accumulation of serum in the ventricles, and dilatation thereof, may, however, be entirely absent. The creamy softening of the parts surrounding the ventricles is then absent, and this appearance must simply be regarded as a post-mortem change due to maceration by the accumulation of serum.

In a small series of cases, the appearances just described are also present, but, despite the most careful examination, no miliary tubercles are found in the pia mater, though they may be wide-spread in other organs. Rilliet and Barthez, nevertheless, regard such cases—and, I believe, with propriety—as tubercular meningitis, because characterized as such by the presence of miliary tubercles in other organs, and by the peculiarity of the inflammatory products. This shows that the latter may develop spontaneously without the irritation of miliary granulations; and, on the other hand, there are also cases of acute tuberculosis, in which inflammation of the pia mater does not occur, despite the presence of miliary tubercles.

In one case alone did I find tubercles confined to the pia mater to the exclusion of all other organs, and only exceptionally has it been restricted in limit—for example: in a boy, aged two and one-half years, with numerous cerebral tubercles and meningitis tuberculosa, who presented merely a few miliary nodules in the right lung. Much more frequently tubercular changes were found in a number of other organs; the most constant was a more or less extensive cheesy degeneration of the bronchial glands, then tuberculosis and cheesy processes in the mesenteric and other lymphatic glands, the brain, lungs, pleura, peritoneum, spleen, liver—even in the epididymis and in the genitalia of little girls. In recent times, the implication of the choroid by tuberculosis has awakened lively interest, because it was supposed at first to constitute an absolutely positive criterion for the diagnosis of tubercular meningitis and acute miliary tuberculosis. Ophthalmoscopic examination was regarded as the most important clinical act, and the discovery of a few grayish white granules in the fundus of the eye was regarded as decisive in doubtful cases. Though this is undoubtedly true, the choroid is by no means constantly involved, and negative appearances in the eye cannot, therefore, be regarded as proof against the existence of meningitis. Tubercles are also found occasionally in the pia mater of the spinal cord; this complication would probably be found more frequently, if the spinal canal were opened at every autopsy. But the assumption that the occurrence of severe convulsions or contracture depends chiefly on the implication of the spinal cord is unfounded, since, in a case in which these symptoms were prominent, the spinal cord was found entirely normal at the autopsy.

I have little to add with regard to the etiology of the disease. Children with a hereditary predisposition to tuberculosis, or those suffering from scrofulous affections, phthisis, or chronic suppuration of bone, are most subject to the affection; but apparently healthy children are not infrequently victims, and it is then often difficult, at the autopsy, to find an infecting cheesy focus in a bronchial or mesenteric gland, as also happens at times in acute miliary tuberculosis without meningitis. The assumption of a traumatic cause—especially a fall on the head, to which the parents are always inclined—is generally mistaken, and is usually due to

an accidental coincidence. But it cannot be denied that concussion of the brain is more apt to produce congestion and its sequences in children with a tuberculous predisposition, than in others (page 117).

All physicians who are conscientious in making a diagnosis will agree with me that they regard every case of tubercular meningitis as lost. The exceptional cases of recovery reported by authors must be accepted with great reserve. However, the possibility of recovery cannot be denied. If we consider that in tuberculous individuals every attack of pleurisy or peritonitis does not prove fatal, that the danger of the disease does not depend upon the miliary granulations of the pia mater (which are not infrequently entirely latent), we can attribute the enormous fatality of the meningitis to only two causes, viz.: coincident acute tuberculosis of numerous other organs, and the local changes undergone by the brain from the softening of the gray matter underneath the pia mater and the increasing pressure of the dilated ventricles. When this has occurred, recovery is no longer possible. But I do not consider it impossible to produce recovery in the beginning, when the miliary tuberculosis is not general, but localized. In former articles[1] I have reported five cases which presented almost all the symptoms of the first period of tubercular meningitis and were cured by energetic antiphlogosis. One of these cases, in a child one and three-fourths years old, terminated fatally in an attack of meningitis three years after the first attack, and after the death of a brother from the same disease—a circumstance which appears to me to support the correctness of the diagnosis. Rilliet and Barthez report two cases in which death occurred from a relapse a couple of years after the recovery of the first attack, and at the autopsy the old and recent tubercles of the pia mater could be clearly distinguished. Although a fatal relapse must be expected sooner or later, even after recovery, this should not induce the physician to remain passive. The fear of antiphlogosis, which is now fashionable, exhibits no successful results, while I have formerly seen recovery occur in a few cases under active treatment at the onset. In the beginning of the attack I applied, according to age, three to six leeches behind the ears and an ice-bag to the head, gave calomel 0.05 every two hours, and inf. sennæ comp. or syrup spinæ cervinæ if the evacuations were not profuse, and ordered inunctions of unguent. ciner. (0.5–1.0) on the throat and neck a couple of times a day. Although we can hope for success from this treatment in only very exceptional instances, it cannot prove injurious in a disease which always proves fatal when left to itself. But this plan should only be adopted in the first few days of the disease; later all methods are useless.

XVIII. Simple Meningitis.

Meningitis simplex, whether it affects the cerebral meninges alone or those of the spinal cord at the same time, is much less frequent than the tubercular variety.

The disease is characterized anatomically by the absence of all tubercular formations, either in the brain or the other organs; though, of course, a tuberculous individual may accidentally—for example, in consequence of fracture of the skull—be affected by simple meningitis. Apart from these and a few other cases (those due to pyæmia), almost

[1] Beitr. zur Kinderheilk., S. 13, Berlin, 1861; u. N. F., S. 55, 1868.

every meningitis in a tuberculous patient assumes the anatomical and clinical characteristics previously described. Meningitis simplex affects the convexity of the hemispheres more frequently and severely than the tuberculous form, but the inflammation may also extend to the base of the brain, the medulla oblongata, and more or less into the spinal canal (cerebro-spinal meningitis). In addition to marked congestion of the pia mater, smaller and larger ecchymoses, and partial adhesions of the arachnoid to the dura mater, the tissue of the pia mater is found infiltrated with yellow or yellowish gray pus, which in part follows the course of the larger blood-vessels, in part is spread out like a membrane, and may also be present in varying quantity between the arachnoid and dura mater, while in other cases the exudation presents a firmer, more fibrino-purulent character. The cortex of the brain is often adherent to the pia mater in various places and softened by serous imbibition. The ventricles are usually empty, though not always; in two cases I found them dilated with opaque, purulent fluid, the ependyma being normal, but the choroid plexus covered with pus. If the spinal cord is affected, it presents similar purulent or fibrino-purulent infiltration of the pia mater and the loose mesh-work of the arachnoid, most marked on the posterior surface. The inner surface of the dura mater of the brain and cord is injected and coated with pus in many cases. All these appearances are common to epidemic as well as sporadic cases of the disease.

This is also true of the symptoms. I have observed sporadic cases much more often than the epidemic variety. So far as my experience goes, the criterion of a violent course, which is regarded as characteristic of simple meningitis as opposed to the tuberculous form, will not always hold good, as cases are not lacking which last as long as the tuberculous variety, or even much longer. It is very difficult to give the numerous variations of the clinical history in one description, and I therefore consider it more practical to illustrate them by cases. 1. *The mild form.*—In discussing cerebral congestion (page 117), I drew your attention to the fact that in little children, during the period of first dentition, cerebral symptoms may develop which can alone be explained by congestion of the brain or meninges, and may readily pass into true meningitic symptoms. The not infrequent recovery of such cases does not contradict the diagnosis of meningitis, since even in an epidemic of cerebro-spinal meningitis, milder cases terminate quite often in complete recovery. The cases to which I refer pursued the following course: in the midst of perfect health the children suddenly became strikingly quiet and apathetic, had a moderate fever (38.5°–39.5°) with a frequent (132–160), but usually regular pulse, and strong tendency to somnolence, which gradually increased to slight coma. The head was hot, the fontanelles elevated and pulsating; frequent starts, both spontaneously and after slight irritation; half-closed lids, fixed stare, strabismus. Of great importance is a moderate rigidity of the neck, with retroversion of the head, which is noticeable in the prone position, but becomes more marked on raising the child, and is occasionally associated with slight rigidity of the limbs. Vomiting may occur, but is not constant; the bowels are almost always constipated. The symptoms may continue in this manner for two or three weeks, until they suddenly begin to improve: the child rouses from the somnolence, begins to smile, the rigidity of the neck and limbs disappears, and complete recovery soon occurs. Moderate antiphlogosis and the constant administration of iodide of potassium appear to be useful in these cases. 2. *The severe form.*—This does not always present the same symptoms. The

disease may continue in a mild form for two weeks or longer, and finally prove fatal from increase of the rigidity of the neck and collapse. In other cases an extremely high temperature constitutes the chief feature and leads to a diagnosis of typhoid fever, until indisputable cerebral symptoms develop.

A. W——, aged nine months, was suddenly taken sick on March 8, 1877, with vomiting. The child was pale and quiet, took the breast unwillingly, but even on the next day presented no serious symptoms. March 10th and 11th, the apathy again attracted attention, and the thermometer showed a temperature of 40.8° at night. In the next four days the high fever constituted the only notable symptom. The thermometer showed:

	Morning.	Evening.
March 12th	40.0°	41.0°
" 13th	40.4°	41.8°
" 14th	40.6°	40.2°
" 15th	40.1°	38.8°

The diagnosis lay between typhoid fever and meningitis, and on my first visit, on March 15th, I did not venture to decide; but on March 16th (a week after the first attack of vomiting) there was moderate rigidity of the muscles of the neck, with deviation of the head to the left, and slight contracture of the right arm at the elbow-joint. All the measures employed were unsuccessful in reducing the high temperature; it varied until death from 40.0° to 41.4°, and only during the last two days did it sink temporarily to 38.5.° Pulse, 130-160, regular. March 18th, the neck was more easily movable, and the spleen appeared markedly enlarged; the child grasped a watch held in front of him, so that the diagnosis of meningitis again became doubtful; but the next day vomiting again occurred, with rigidity of the neck and contracture of the right arm. March 21st, twitchings of the entire body, face dark red, profuse perspiration. Frequent cries and repeated vomiting during the night. May 22d, an epileptiform attack lasting half an hour, followed by movements of mastication and nursing, converging strabismus and injection of the eyes. March 23d, convulsions, which returned at 10 P.M., and continued until death, March 24th, at 3 P.M. Autopsy.—Very intense cerebro-spinal meningitis; about one-half ounce of free pus on the surface of the brain; purulent exudation, 1 ctm. in thickness, between the arachnoid and pia mater, the softening extending about 1 ctm. into the gray matter; ventricles empty. Spleen enlarged three-fold; other organs normal.

This case presented some similarity to tubercular meningitis on account of its long duration, the initial vomiting, the moderate spastic symptoms beginning on the eighth day, and the terminal convulsions; the high continued fever constituted an important difference. A noteworthy feature was the considerable enlargement of the spleen, which is not infrequently observed in the epidemic form of meningitis, but occurred here in an apparently sporadic case. It is evident that Rilliet and Barthez go too far in the statement that meningitis always begins, in very young children, with repeated convulsions. This form of onset is not constant, though frequent, and when it does occur the initial spasms may remain absent for days and only reappear at the last. The following case shows, in opposition to Rilliet and Barthez, that convulsions may open the scene in children more than two years old:

A girl, aged five, was suddenly seized, in the midst of perfect health, with severe headache and vomiting, followed in three hours by general epileptiform convulsions and coma. The spasms then ceased for twelve hours, after which they returned and continued until death, which occurred forty-eight hours from the beginning of the attack. Autopsy.—The entire convexity of the hemispheres was covered with a yellow, purulent exudation; similar exudation at the base, around the optic and motor oculi nerves. Brain otherwise normal, ventricles empty. Other organs healthy.

At this age the children usually complain severely of headache, and press the hands against the forehead and temples, while groaning or crying; but younger children only manifest headache by grasping at the head and by piercing cries. Between the varieties just described is found: 3. *A moderate form*, which is not alone characterized by a protracted course, but also by alternate improvement and exacerbation of the symptoms. This change of symptoms is attended with corresponding fluctuations of temperature. Recovery occurs not infrequently in these cases.

E. P——, aged seven, admitted in November, 1872, with catarrh of the large bronchi and typhoid symptoms. Coma; dry, brown tongue; liver and spleen normal. Temperature, 39°–39.5°; later, 38.8°. Six days after admission, rigidity of the neck and rigid flexion of the lower limbs, dilatation of the left pupil, frequent outcries, then flexion of the fingers and supination of the hands. Temperature varying from 36.6° to 38.2°. On the twelfth day, improvement, tongue moist, tremor of legs, consciousness returning, appetite better. Exacerbation during next two days. Temperature normal. On the sixteenth day, sensorium clear; temperature, 38.5°–39°. After the twenty-second day, disappearance of all spastic symptoms. Euphoria. Pulse varied during entire disease, from 140 to 132, except on the twenty-eighth day, when it sank to 46, the temperature being 36.8°.

O. K——, aged seven years, admitted December, 1872, with gastric symptoms, pain in the head and abdomen, tense abdominal walls. From the third to the seventh day, violent delirium, somnolence, apathy, normal temperature. Decided improvement from the seventh day. Sensorium clearer until the eleventh, when an exacerbation occurred. Violent pain and moderate rigidity of the neck, contracture of the adductors of the thighs; temperature, 35.5°, and pulse, 60–64, until the evening of the twelfth day. All the symptoms increased; there was hyperæsthesia of the lower limbs, with repeated vomiting, pains in the back; temperature rose to 39.7°–40.4°; pulse, 110–142. On the fourteenth day the symptoms began to diminish, and recovery ensued.

The treatment in both cases consisted of the repeated application of leeches to the head and wet cups along the spine, lukewarm baths (with cold douche to the head and spine in the first case), inunctions with gray ointment; internally, calomel and purgatives.

In the summer of 1879 I observed, in rapid succession, a number of cases of this kind, among which was one that lasted seven weeks. In this case the chief symptom in the beginning consisted of attacks of intense headache, with vomiting and loud cries, which were soon combined with a fluctuating rise of temperature (occasionally 41.0° in the morning), painful stiffness of the neck, and deviation of the head to the left. Consciousness remained undisturbed, and, after numerous variations in the severity of the symptoms, complete recovery ensued.

But recovery is not always complete. I have repeatedly seen deafness or amaurosis left over, and deaf-mutism in young children. These disturbances are attributed to neuritic changes, due to the propagation of inflammation to the optic and acoustic nerves. Children who become deaf in consequence of meningitis before the development of speech naturally remain mute.

Next to epidemic influences, injuries and diseases of the cranial bones play a prominent part among the causes of meningitis. After a severe concussion of the brain from a blow or fall, symptoms of congestion of the brain may develop and pass into those of meningitis, as was remarked above (page 117). Fissures or fractures of the cranial bones are much more dangerous; in addition to meningitis, they may cause more or less extensive hemorrhages within the cranial cavity.

M. E——, aged five years, admitted July 1, 1875, had fallen on the head three days before. Sensorium clouded, right pupil narrow, bladder extends to the umbilicus. The head is turned to the right. Temperature, 39.8°; pulse, 120, regular; respira-

tions, 30. On the next day, violent delirium, severe pains in swallowing, but pharynx normal. July 3d, complete somnolence, moderate rigidity of the neck, slight twitchings of the arms. July 4th, death.

	Morning.	Evening.
Temperature on July 2d	39.6°	39.8°
" " " 3d	40.1°	40.5°
" " " 4th	41.5°	40.3°

Autopsy.—Marked congestion and purulent infiltration of the pia mater on the convexity, especially on the left side; also in the Sylvian fissures. Three fissures in the bones of the left side of the base of the skull, passing through the frontal bone, the greater and lesser wings of the sphenoid, and the temporal bone. Extravasations of blood between the dura mater and the bones corresponding to these fractures.

The absence of serious motor disturbances is a notable feature in this case. The pains on moving the head and in swallowing must be explained by movement of the fractured bones; in swallowing, the pterygo-pharyngeus and stylo-pharyngeus muscles must have exercised traction on the fractured base of the skull.

Meningitis may also be due to chronic diseases of the cranial bones. It also develops secondarily, at times, during various acute diseases, for example, pneumonia, scarlatina, pyæmia, and septicæmia, especially in the new-born, but the symptoms are usually so complicated with those of the primary disease that a positive diagnosis is difficult or impossible. The complication of scarlatina or pneumonia with meningitis must, however, be rare, as no case has come under my observation.

The cerebral symptoms occurring in all high fevers in children, especially in the initial stage, must be regarded as the result of the fever or the virulence of the disease, and not of meningitis. I also call special attention to the fact that otitis media, or even externa, in children, may produce violent cerebral symptoms, leading to an erroneous diagnosis of meningitis, until suddenly a profuse discharge of pus from the ear occurs, and the symptoms rapidly disappear. The external auditory canal should, therefore, always be carefully examined. But I must conclude, from my own experience, that cases are rare in which the symptoms of otitis really simulate meningitis, and still rarer are those in which the meningitic symptoms are due to rhinitis.

Finally, I will refer to the marked increase in the frequency of respiration which occasionally accompanies meningitis, and may lead to a mistake for diseases of the respiratory organs. As a rule, the respirations are also irregular, very frequent and superficial respirations alternating with slow ones, which are interrupted by sighs; but this characteristic is not constant, and the increased frequency is occasionally permanent.

A child, aged eleven months, suffering for a week from meningitic symptoms, viz.: fever, somnolence, with half-closed lids, slight rigidity of the neck, frequent twitchings, pulsation of fontanelle. Respirations, 50–60 per minute, superficial, but regular; no cough. Examination of the chest negative. On the disappearance of the cerebral symptoms the rapidity of respiration ceased.

Antiphlogosis plays the principal part in the treatment of meningitis. Even here many authors dread bleeding, but this must be regarded as a grave sin of omission. The bleeding must be regulated by the general condition of the patient. In small, poorly nourished, anæmic children, two to four leeches should be applied, according to the age of the patient, and the bites should not be allowed to bleed afterward; in older children, especially if robust, six to ten leeches, or an equal number of wet cups, to the neck or

spine, are requisite. Under these conditions I have repeated the bleeding even when new exacerbations occurred. So long as symptoms of collapse are absent, an ice-bag should be constantly applied to the head, inunctions of ung. ciner. mercur. (0.5–1.0 every three hours) applied to the neck, back, arms, and legs, and calomel given internally (0.015–0.03 every two hours). The ordinary antipyretic remedies are useless in these cases. When there is great restlessness or violent convulsions, resort may be had to injections of morphine (0.002–0.005) or chloral hydrate (P. 9), and also to lukewarm baths (25°–26°), with cold affusions to the head. The mild form described on page 127, especially when it occurs in infancy, does not require such active antiphlogosis. The ice-bag, continued small doses of calomel (0.01–0.015 every two hours), and mercurial inunctions (1.0 ung. ciner. twice a day), may suffice in acute cases, but even here we do not need to dread the application of one to four leeches to the head, if the children are vigorous. Under all circumstances I would recommend iodide of potassium (P. 13) after the termination of the acute stage. During the continued use of this remedy I have repeatedly seen the children gradually awake from the comatose condition, the contractures disappear, and complete recovery finally occur.

XIX. Neuralgic Conditions.

You will observe disorders of sensation much more rarely during childhood than in adult life. Anæthesiæ, hyperæsthesiæ, and neuralgias are exceptional, and so similar to the like condition in adult life that it is unnecessary to refer to them in detail. Anæsthesiæ are especially difficult to determine, even in older children, on account of the dread inspired by examination with the needle, and I have never been able to define accurately the anæsthetic regions even in serious diseases of the central organs (tumors, tubercles, sclerosis). Among the neuralgias of childhood, colic, which is either flatulent or associated with diarrhœa (page 52), and hemicrania (migraine), alone merit special attention.

Migraine occurs in children not much more rarely, and with almost the same symptoms as in adults. From an experience of many years I can state that it has become especially frequent during the last twelve to fifteen years, and the cause of this increase is the excessive effort required by the present methods of teaching. The increasing size of our city, which makes the enjoyment of country air more difficult, the mental exertion in the overfilled school-rooms, the scanty hours of recreation, which are still further shortened by home-work and music-lessons—all these factors, in combination with nervousness, whether congenital or due to improper breeding, appear to me to be the chief causes of the headaches which are so frequent after the age of seven years.

An hereditary factor must also be considered. The heredity may be transmitted from the father or mother, and under these conditions several children may be affected in one family.

Anæmia, which is quite frequent at the age of five to six years, and still more common after second dentition, favors the development of migraine, which is then usually combined with vertigo; nervous headache is also often complained of in the hysterical conditions previously described. In a few of these cases, headache possessing the characteristics of migraine often remains after the disappearance of these conditions.

The female genital system, however, need scarcely be considered in this connection, and the following case, therefore, appears so much more noteworthy:

A girl, aged seven, presented January 2, 1873; has suffered from migraine since May, 1872. Severe pains in the forehead and temples, nausea, exhaustion, photophobia; attacks continue a couple of hours; irregular return. Fluor albus since May, 1872; introitus vaginæ very red, hymen normal. Treatment.—Fomentations with lead-wash, vaginal injections of sulphate of zinc (0.5 to 200.0). Quinine internally, later bromide of potassium. All the symptoms disappeared until December, when the leucorrhœa returned, and with it the attacks of migraine. Further course unknown.

In cases of this kind we must remember that the vaginal catarrh and the headache may result from genital irritation due to onanism or ascarides.

As a rule, the migraine is not so strictly unilateral as in adults, but is generally confined to the middle of the forehead. The duration of the attacks varies from a few hours to two days, the intervening nights being often disturbed by restlessness, a feeling of heat, and speaking during sleep. Vomiting, dread of bright light and noises, were frequent; occasionally also general tremor and rapid respiration. The intervals were irregular—in some cases a few days, in others several weeks. Among the exciting causes none was more frequent than the atmosphere and mental exertion of school. I have also seen attacks produced by various emotions—fear of punishment, scolding.

Even after the most careful examination the physician is not infrequently in doubt whether he has to deal with migraine or headache due to cerebral disease (tubercle, tumor), and the diagnosis can be rendered certain only by prolonged observation during the intervals and careful examination into the etiological conditions.

The treatment depends upon the nature of the latter. While we are powerless against hereditary predisposition, we can much more decidedly combat the influence of mental overwork. I have repeatedly seen good effects from sending the children to country boarding-schools instead of city schools. In the majority of cases this cannot be done, and nothing remains but to restrict household duties, to secure regular hours of recreation, and prolong the vacations as much as possible. The often recommended cold frictions after rising from bed have done little or no service. I have accomplished more by cold baths and swimming exercises. Iron is advisable in anæmic patients. Quinine and bromide of potassium gave very variable results (quinia in doses of 0.05, t. i. d., bromide of potassium, 0.5–1.0, t. i. d.). A stay at the seashore or on the mountains, and mental rest, act better than drugs, though only temporarily. We should always bear in mind that simulation may also come into play, and the pains be very much exaggerated, in order to shirk attendance at school. When onanism is suspected or known, an earnest and intentionally exaggerated statement of its dangers is more effective than punishment.

PART IV.

DISEASES OF THE RESPIRATORY ORGANS.

I. Inflammation of the Nasal Mucous Membrane—Rhinitis.[1]

The mucous membrane of the nares, larynx, and bronchial tubes is subject, with uncommon frequency, to catarrhal affections in children of the lower classes, on account of their frequent exposure. The symptoms are like those in adults: stoppage of the nose, increased secretion of purulent mucus, sneezing, catarrhal affection of the conjunctiva, hoarseness, hoarse or hollow, barking cough, with or without febrile movement. These catarrhs also form a constant prodroma of measles. Under all conditions a catarrh of the upper part of the mucous membrane of the respiratory passages in small children is much more serious than in later life, because a simple coryza may soon give rise to symptoms of stenosis at the entrance to the larynx, or rapidly spread to the deeper bronchial tubes. Infants suffering from coryza or mild laryngeal and tracheal catarrh should, therefore, not be carried into the open air, but be carefully protected from inclement weather.

Though more rarely than measles, scarlatina and diphtheria give rise quite frequently to inflammation of the nasal mucous membrane of a diphtheritic character, and usually secondary to the already existing diphtheritic affection of the pharynx. A gangrenous, purulent nasal secretion flows over the upper lip, reddening and excoriating it and the nostrils. In severe cases, the vicinity of the nose is œdematous, the conjunctiva injected, the eye running tears from obstruction of the naso-lachrymal duct. I have not often been able to observe the diphtheritic patches on the nasal mucous membrane, as they are situated too high up. The examination of the naso-pharyngeal cavity with the mirror is even more difficult, or entirely impossible. The swelling of the mucous membrane is so marked in these cases, that respiration is interfered with, and a snoring tone produced, especially during sleep. As a rule, this rhinitis is a bad omen in scarlatina and diphtheria, though mild forms may occur without any bad effects. Diphtheria may begin with an affection of the nares; but I have seen only one case in which pseudomembranous rhinitis occurred alone:

A girl, aged eight, was taken sick with coryza and moderate fever. Snoring during sleep and frequent complaints of an obstruction to respiration near the alæ nasi in-

[1] Compare the descriptions of coryza neonatorum and syphilitica, pages 48 and 51.

dicated more marked stenosis than usually occurs in ordinary coryza. Simple catarrhal redness in the pharynx and upon the epiglottis. After the lapse of a few days, the child expelled from the nose a tough, white mass as long as a finger, which became swollen on the addition of acetic acid. In a few days a smaller mass was discharged, and the symptoms immediately ceased. The treatment was purely expectant.

Was this a case of true diphtheria confined to the nares, or a simple fibrinous rhinitis?

A chronic form of rhinitis occurs very often in scrofulous children in connection with other scrofulous symptoms. Swelling of the nose externally, snuffling and snoring respiration, discharge of a sero-purulent secretion from the excoriated nostrils, redness and swelling of the upper lip, are among the most frequent symptoms. This form of rhinitis not infrequently gives rise to repeated attacks of facial erysipelas (page 24). Chronic rhinitis may also develop after measles, scarlatina, or even after very severe coryza, without any scrofulous predisposition. Apart from the use of antiscrofulous remedies, to which I shall refer later, I obtained good results from pencilling the nose daily with a solution of nitrate of silver (1 to 30).

II. Pseudo-Croup.

If you learn that a child has had croup four or five times, you may rest assured that the disease was merely pseudo-croup. This affection always begins suddenly, usually soon after a mild coryza, and almost always at night—often shortly after falling asleep. The child starts from sleep in a paroxysm of hollow or hoarse coughing. Not only the cough, but also the deep inspirations which interrupt the latter, are accompanied by a decidedly croupy, saw-like noise. The cry may be entirely normal or somewhat hoarse. During the attack, many children sit up in bed with an anxious expression and red cheeks, breathe noisily and laboriously, are extremely restless, and repeatedly grasp at the throat. The skin is hot, often covered with perspiration; the pulse accelerated. The attack usually lasts only a few minutes, but the breathing remains somewhat noisy and frequent after its subsidence. On the physician's arrival the child is usually comparatively quiet or even asleep, the accessory muscles of respiration are no longer in action, and, at the most, the alæ nasi gently rise and fall. But the attack is often soon repeated; the children, on awaking from sleep, again cough with a croupous sound, and long-drawn, hoarse inspirations are heard during crying. Pressure on the larynx or trachea will immediately produce an attack. Many children are entirely well on the next day, and only an occasional hoarse or barking cough reminds us of the nocturnal attack; in others, a similar scene is repeated on the following night. After this an ordinary loose cough remains, and may continue for one or two weeks.

Examination of the pharynx shows, at the most, slight catarrhal redness and swelling, and nothing further is discoverable in the larynx. The affection consists of a nasal catarrh, which spreads to the entrance of the larynx, and in which, especially during sleep, an increased swelling of the mucous membrane occurs and causes the sudden waking, with want of breath, feeling of anxiety, and hoarse cough. The dryness of the cough is usually diminished by warm drinks (sugar-water, milk), and disappears with the occurrence of a more profuse catarrhal secretion. I order warm water or milk to be taken, hydropathic applications, or warm

poultices, to the neck, and keep the children in bed for a couple of days. It is also advisable to apply salt pork constantly to the throat. This usually produces slight erythema, or small pustules. It is bad practice to give emetics in these attacks, as is so often done by mothers in whose families the disease is endemic, as it unnecessarily weakens the children. There is no remedy which will prevent the recurrence of the paroxysms. Many children suffer from these attacks from their ninth or tenth month, but they gradually grow more infrequent and mild, and generally disappear about the sixth or seventh year. These children, especially when they have coryza, must be carefully protected from the air and kept indoors.

Similar attacks sometimes open the scene at the development of measles and whooping-cough. Both diseases, especially measles, may begin with such an attack, which then passes into an ordinary catarrh, and indicates its true character in the first case after a few days, in the second after one or two weeks.

Although the large majority of cases present the mild character described above, you should not neglect watching the child for a few days after the first attack. Although very rarely, I have now and then seen true croup develop after an attack of pseudo-croup, and this possibility renders it incumbent upon you to keep the children indoors until the loose catarrh occurs, *i.e.*, as long as the cough still retains a slightly croupous sound or a hoarse noise is heard during forced inspirations.

III. Pulmonary Atelectasis.

The tendency of the lungs to collapse is of decisive importance in all respiratory diseases of childhood. Atelectasis is that condition in which the alveoli of the lungs have a tendency to become destitute of air, and fall together in such a manner that their walls come in contact. At the autopsy you will find, on the external surface of the lungs, especially at the anterior borders, at the lower and inner edge of the lower lobe, and at the tongue-like projection over the pericardium, sharply defined bluish red or steel-blue, slightly depressed patches of varying size, at times scattered and small, at times more extensive and collected into long strips or spots as large as a dollar, or even larger. On section they appear firm, do not crepitate, do not give exit to air-bubbles, but only some bloody fluid, and sink in water. The cut section is smooth and the connective-tissue septa of the lobuli are readily recognized as white bands. If air is blown into the bronchus leading to the atelectatic portion of the lung, this becomes distended, thus showing that it is not due to pneumonic infiltration.

There are two factors in the causation of atelectasis: first, diminution in the energy of inspiration, which does not force the air into the alveoli; and second, filling the bronchi with mucus, which renders the entrance of air more difficult. When this can no longer enter the alveoli, the air contained in the latter, according to recent experiments,[1] is absorbed by the circulating blood, and the alveoli then collapse. Atelectasis will, therefore, occur most frequently in those cases in which the above-mentioned causes are combined, *i.e.*, in all exhausting diseases complicated with bronchial catarrh.

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[1] Lichtheim: Arch. f. exper. Path., X., S. 54.

Rachitic children with a narrowed thorax are especially predisposed to atelectasis, because a third cause is here added, viz.: the narrowness of the thorax, which interferes mechanically with the complete distention of the lungs. In stenosis of the larynx and of the larger and smaller bronchi, whether due to inflammatory processes or the presence of foreign bodies, multiple atelectasis develops in the lungs, on account of the difficult entrance of air into the alveoli and the constantly increasing diminution in inspiratory activity.

Atelectasis can rarely be diagnosed during life. Although the view that hyperæmia of the tissues, and finally broncho-pneumonia, may develop in the atelectatic parts, in consequence of the deficient pressure of air upon the vessels, has been rendered doubtful by experiments,[1] the increased insufficiency of the lungs from multiple atelectasis must be regarded as a factor which markedly clouds the prognosis. The difficulty in diagnosis is due to the fact that the small spots of atelectasis scattered in the parenchyma do not give rise to any physical signs; and even extensive atelectasis, involving a large part of the lower lobe, will merely give rise to signs of consolidation, which cannot be distinguished from those of pneumonic infiltration. The absence of fever would be significant of atelectasis were it not that pneumonia often occurs in badly nourished children without any rise of temperature, and that, on the other hand, atelectasis often develops as a result of febrile diseases (bronchitis, croup, typhoid fever). For these reasons a positive diagnosis of atelectasis can hardly ever be made—at the most, a probable diagnosis founded on our anatomical experience that it frequently occurs in children, in certain diseases, and in exhausting conditions.

In congenital atelectasis a greater or lesser portion of the lungs remains in the fœtal condition. The parts in question have not yet been engaged in respiration, and are therefore dense, of a steel-blue color, and heavier than water, as they are in the fœtus. In general, congenital atelectasis has the same causes as the acquired form, especially difficult or feeble respiration, such as occurs in asphyxiated or prematurely born children. As a rule, this form is more extensive than the acquired, and not alone causes distinct signs of consolidation, but also gives rise to stasis in the pulmonary artery and entire venous system, with cyanotic discoloration, on account of the marked disturbance in the pulmonary circulation. For this reason, also, the closure of the fœtal channels of circulation, especially the foramen ovale, does not always occur in a normal manner. Many new-born soon die in consequence of atelectasis and the weakness of the vital forces which has given rise to it, while, in other cases in which it does not affect both lungs to such a great extent, recovery may occur:

An extremely feeble, prematurely born child, aged three weeks, was presented for treatment in May, 1880; it had been cyanotic during the first week, and had severe dyspnœa. Dulness on the right side posteriorly between the scapula and spine; the normal respiratory murmur was absent here, and rales were present; no fever. The child has done well under good nursing, the use of wine, and chamomile-baths; at the time of my examination the percussion-note was very little different from that of the other side; vesicular murmur feeble, but distinct; in October only a mild bronchitis could be detected.

I think this case must be regarded as congenital atelectasis of a large part of the right lower lobe, as the symptoms were present from birth;

[1] Traube: Beitr. z. exper. Path. u. Physiol., Experiment 63, Heft I. 1846.

febrile movement had never been present, and good nourishment and care sufficed to gradually dissipate the threatening symptoms. In the following case a fatal issue occurred under the influence of extremely unfavorable conditions:

A child, aged six weeks, had been placed on the floor of a house in very cold weather by its unknown mother; admitted to the hospital January 8, 1873. Very small, thin child, with cyanosis of the lips and eyelids, turgescence of the veins of the face and head. Repirations extremely weak and superficial. Percussion-note slightly dull over entire chest; respiratory murmur very feeble. Feeding from the bottle impossible on account of weakness; sprue in the mouth and pharynx; subnormal temperature. Death in collapse. February 16th. Autopsy.—Heart normal; sprue in the œsophagus. Uric acid concretions in kidneys. Both lower lobes of the lungs atelectatic in great part, but some parts containing air were visible between the compressed portions. Scattered atelectatic spots in other lobes. Bronchi normal.

IV. Inflammatory Affections of the Larynx and Trachea.

Acute catarrh of the upper respiratory passages develops either out of a pseudo-croupous attack, or gradually with increasing hoarseness and a hoarse or barking cough. There are certain children and adults in whom every cough has a hollow, metallic sound, while all other signs of a laryngeal affection may remain absent. This peculiarity must be taken into consideration, as it readily gives rise to unfounded alarm. The hollow, metallic sound is less to be dreaded than the harsh, hoarse cough, which, when associated with a more or less thick voice, always excites alarm. If a moderate pressure with the finger is made, under these circumstances, upon the trachea or larynx, the children not alone distort the face in pain, but they usually cough with the harsh, hoarse sound which we are accustomed to call croupy. Inspiration, especially during crying, is accompanied by a sound like that of sawing, although the respiration may be quiet, without any trace of dyspnœa. I have often been called, in the first few days after an attack of pseudo-croup, because severe laryngeal symptoms had again developed, and I have almost always found that this was due to bad humor or crying on the part of the child. As soon as the agitation subsided, the symptoms disappeared. These local symptoms are often combined with loss of appetite, a mucous coating on the tongue, and perhaps moderate fever with nocturnal exacerbations. These cases always require attention, as we cannot foretell whether the symptoms will not assume a more threatening character in the next few hours.

Under these circumstances, the administration of emetics (P. 6) is advisable. After this has acted, you may prescribe a mixtura solvens (P. 15) and hydropathic applications to the neck. The child must remain in bed until the croupous sound of the cough is entirely lost and inspirations are performed noiselessly. The catarrh usually begins to resolve in a few days, the cough becomes loose and rattling, the hoarseness disappears, and everything is over in one to two weeks. But an exacerbation may ensue despite the best of care, though it is usually observed as the result of negligence. Within a few hours the symptoms may attain a severity which endangers life. This exacerbation of the symptoms is due either to rapidly increasing catarrhal swelling or to a fibrinous exudation upon the inflamed membrane, or finally, to a seropurulent infiltration of the ary-epiglottic ligaments and their immediate

vicinity. All these anatomical changes give rise to acute laryngeal stenosis.

The previously described symptoms, viz., hoarseness, harsh cough, sensitiveness of the larynx and trachea to pressure, noisy inspiration and expiration, are now suddenly combined with dyspnœa, action of the alæ nasi, associated movements of the head in respiration, increasing depression of the lower part of the neck, epigastrium, and lower part of the thorax during inspiration. The frequency of respiration is very little increased, and, even in severe cases, does not exceed twenty-four to twenty-eight per minute. The individual inspirations and expirations, which are accompanied by a saw-like sound, are unusually long.[1] The general euphoria of the child may, however, be quite undisturbed. The harsh sound which accompanies inspiration and often also expiration, in all such cases, may be best compared to the double sound of a saw cutting wood. Its intensity is not always the same, may disappear for a short time after vomiting, and is most marked during sleep.

If the treatment remains unsuccessful, the symptoms of stenosis increase almost from hour to hour: the child grasps the throat and bends the head forcibly backward; the face becomes pale and cyanotic, the eyes express terror and implore aid from those around; drops of perspiration are seen on the forehead and cheeks, but the skin is not warm, and indeed seems cooler at the tip of the nose and on the cheeks; the hoarseness of the voice rapidly increases to complete aphonia, the previously harsh cough becomes toneless, and finally disappears entirely. Fever does not play an important part in these affections. Although never entirely absent, it usually varies from 38.5° to 40.0° with remissions in the morning; the frequency of the pulse may be increased to 144 and more by the restlessness of the child.

This symptomatology, as I have remarked, only permits the diagnosis of acute laryngeal stenosis, but its cause cannot be decided forthwith. You must carefully examine the pharynx to convince yourself of the presence or absence of diphtheritic deposits on the mucous membrane. When present, the diphtheritic nature of the stenosis is rendered certain; if not present, the possibility of diphtheritic croup cannot be denied, because the patches in the pharynx may not be accessible to observation during life, or have perhaps been exfoliated. Examination with the laryngoscope will clear up the diagnosis, but this can only be performed in a small number of cases (page 4). If a diphtheritic process can be excluded, the disease is either a simple or a pseudo-membranous (fibrinous) laryngitis (croup), as it has been shown that the most severe dyspnœa—in fact, almost all the symptoms of croup, can be produced by acute laryngitis attended with considerable swelling of the laryngeal mucous membrane. These cases are naturally much more amenable to antiphlogistic treatment than the pseudo-membranous form.

M. F——, aged six, had a severe attack of pseudo-croup during the night of December 7th. Euphoria on the following day until 1 P.M., when a threatening series of symptoms suddenly developed. Saw-like noise in breathing, cyanotic face, head bent back, violent action of all inspiratory accessory muscles, the eyeballs rolled up between the half-opened lids, short, harsh cough, accompanied by a whistling sound; hoarseness of the voice. Nothing abnormal in the pharynx; can drink without difficulty. Vesicular breathing marked by laryngeal stridor; a sonorous râle at the root of the lung. Pulse,

[1] Concerning the significance of this symptom, compare Cohnheim: Vorlesungen über Allgemeine Pathologie, II., S. 168. Berlin, 1880.

120; skin hot and perspiring. I ordered six leeches above the manubrium sterni; internally, tartar-emetic (0.12 to aq. dest. 100.0—a teaspoonful every two hours). At 5 P.M. gave a full dose of pulv. rad. ipecac. and tartar-emetic, which was followed by repeated vomiting. At 8 P.M. the child was more quiet, stridor diminished, voice clearer. The solution of tartar-emetic was continued, and a blister applied over the larynx. December 9th, stridor almost entirely gone, cough diminished. At 2 P.M. a short exacerbation of the laryngeal symptoms occurred after the introduction of an enema, which was violently resisted; then rapid recovery occurred.

This is an example of the development of very severe laryngitis with a pseudo-croupous beginning, and also shows the efficacy of energetic antiphlogosis, which I cannot recommend too highly in violent cases. You should immediately apply two to six leeches, according to the age of the child, to the throat, at best immediately above the manubrium sterni—in the first place, in order to keep the region of the larynx free for other external applications; and secondly, in order to have a bony base for compression of the leech-bites in case the bleeding is too profuse. Even during the bleeding I have repeatedly seen considerable amelioration of the dyspnœa. After bleeding I give an emetic or tartar emetic in dosi refr. (P. 18), which, as is shown above, does not always produce vomiting or diarrhœa. I have never seen bad results follow if the remedy is immediately discontinued as soon as diahrrœa or too severe vomiting occurs. It is therefore preferable to give a full emetic dose rather than continued small doses of tartar stibiatus; inunctions of mercurial ointment (1.0 two or three times a day) in the lateral parts of the neck; finally, a blister to the larynx, to whose raw surface, as a rule, I apply unguent. ciner., complete the treatment.

If we consider the annoying stenosis of the nares which may suddenly occur in severe coryza, especially during the night, from increased swelling of the mucous membrane, we will understand how in like manner, though with more threatening symptoms, very acute swelling of the mucous membrane may occur in laryngeal and tracheal catarrh, and may subside almost as rapidly under appropriate treatment. Despite all our efforts, however, it may prove fatal, as all inflammatory processes near the entrance to the larynx are readily complicated by œdematous or sero-purulent infiltration of the vocal cords, the epiglottis and its folds (so-called œdema glottidis, better laryngitis submucosa), and suddenly cause danger of suffocation. Therefore, not only cases of acute laryngeal catarrh, croup, or ulcers of the larynx, but also severe pharyngitis, tonsillar abscesses, and deep phlegmons of the cellular tissue of the neck, are threatened by this danger. In England, scalding of the pharynx and entrance to the larynx by boiling water, which the children had sucked in from the mouth of a teapot, has been often observed as a cause of submucous laryngitis. In all these cases the previously described dyspnœal and stenotic symptoms acquire such an intensity, when œdema of the glottis develops, that suffocation must be feared at any moment. Occasionally the swollen epiglottis can be felt with the finger, or even seen projecting behind the tongue. The rapid performance of tracheotomy is the only measure which will save life.

But the danger of acute laryngitis in children is more often found in the tendency to fibrinous exudation on the mucous membrane. While in the above-mentioned form the autopsy shows only dark redness and swelling of the mucous membrane, at the most superficial erosions and sero-purulent infiltration of the epiglottis and the neighboring parts, especially the ary-epiglottic ligaments and vocal cords, we here find, upon

the mucous membrane of the larynx and trachea, isolated patches or larger pseudo-membranes of a grayish or yellowish white color, either of filmy delicacy or 1‴ and more in thickness, consisting then of several superimposed layers, the one nearest the mucous membrane being the least consistent. This membrane, which consists microscopically of an extremely fine net-work of fibrin and numerous young cells (epithelium, pus-cells), often extends far into the trachea, even to the bifurcation or into the large and medium-sized bronchi ; it then represents cylindrical moulds of the tubes, which are not adherent and can be readily removed. After removal of the false membrane, the mucous membrane is either more or less thickened and reddened, or it is perfectly pale, without any trace of vascularization. This condition is almost always accompanied by bronchitis and broncho-pneumonia.

My opinion that croup is the highest development of acute laryngitis is opposed to that of many authors who regard it as always diptheritic. I will acknowledge that croup has become more frequent since the epidemic and endemic spread of diphtheria ; but I see herein no reason to deny every other mode of development. We know from experiments that croup can be produced in rabbits and dogs by the application of caustics to the tracheal mucous membrane, and the inspiration of hot vapor of water. It is merely necessary, as Weigert and Cohnheim believe, that the epithelium, which remains intact in catarrh, be necrosed and carried away by secretion in order to cause coagulation of the fibrinous exudation secreted by the inflamed mucous membrane, and thus to form the croupous membrane. The irritation of the diphtheritic infectious matter, probably its aspiration from the pharynx, is undoubtedly the most frequent, though by no means the only cause of croup. Every severe laryngeal catarrh may pass into croup, and we therefore occasionally find croup very early in measles, a disease which is combined, from the beginning, with catarrh of the larynx and trachea, and although diphtheria has not been present. I have already reported two cases which tended to prove the existence of primary inflammatory croup independent of diphtheria. They occurred in children aged respectively seven and fifteen months, and the autopsies showed laryngeal and tracheal croup without the slightest change in the pharynx. I have since had repeated opportunities of making similar observations apart from the still more numerous cases in which autopsies could not be obtained, and which could not, therefore, be regarded as perfect evidence, as the pharyngeal diphtheria may have been so situated as to escape observation. The following case must be admitted as convincing :

M. R ——, aged one and one-half year, admitted April 4, 1877, with rachitis and slight bronchial catarrh. Spread of the latter in the next few days. In the night of April 9th, there was sudden croupy respiration and hoarse cough. April 11th, complete croup. The transmitted croupy sound was heard over the lungs, with rude breathing and sibilant râles posteriorly. Temperature, 39.0 ; pulse, 144 ; respiration, 42. April 12th, death. Autopsy.—Pharynx unaffected ; croup of larynx and trachea ; œdema glottidis ; broncho-pneumonia duplex ; rachitis.

Such cases, which begin as bronchial catarrh and suddenly pass into fibrinous tracheo-laryngitis, are described as "ascending croup." The performance of tracheotomy, which was proposed, was prevented by the extremely rapid course, and would have proven useless on account of the extent of the double broncho-pneumonia.

I consider it entirely arbitrary to hold that in such cases the diph-

theria has overleaped the pharynx and developed in the larynx and trachea. The unbiassed observer who carefully follows the clinical history of the disease, as well as the anatomical appearances, will accept the view of an inflammatory local affection, which has nothing to do with infectious diphtheria. The beginning with the symptoms of simple tracheal or bronchial catarrh, the absence of pharyngitis and all prodromal symptoms of infection, and of the enlargement of the glands below the jaw, are sufficiently significant.

The clinical symptoms of croup represent the highest grade of the acute laryngeal stenosis described above. It increases in intensity from hour to hour, and has a duration, in fatal cases of one to three or four days. Although short remissions may occur, usually in consequence of emesis, they are almost always illusory. In many cases the progressive course is interrupted from time to time by attacks of extreme dyspnœa; the panting child throws itself forcibly backward, respiration ceases, the face is cyanotic, the hands are clenched convulsively, and death seems imminent; but, after a few seconds, the air laboriously enters the larynx with a whistling noise, and the former symptoms reappear until a new attack begins. These attacks are probably due to spasm of the glottis, produced as a reflex from the inflamed mucous membrane. In this stage the stridor in respiration is often heard in front of the door of the sick-room, while the croupy cough becomes more infrequent with the increasing aphonia. The restlessness of the children is enormously increased; their terrified eyes plead for help, and this condition is interrupted only by short periods of slumber, during which the saw-like, whistling laryngeal sound reaches its greatest intensity. Examination of the lungs usually furnishes no results; at the most, dry or moist râles in various places, or, perhaps, dulness on percussion, indicating implication of the bronchi and pulmonary tissue. When the latter event occurs, the number of respirations is markedly increased, rises to fifty to seventy or more in the minute, and this symptom alone is sufficient for the diagnosis of a complicating bronchitis or broncho-pneumonia, though physical examination furnishes negative results.

In a number of cases, pseudo-membranous shreds and tubes are expelled, and this is to be regarded as the only reliable diagnostic sign of true croup, since all the other symptoms may be produced by the highest grades of simple laryngitis, especially by œdema glottidis. The nature of the expectorated masses is best recognized by floating them in water, when larger or smaller whitish shreds, often with jagged edges, are discovered, or complete cylinders, which not infrequently branch dichotomously, and thus form moulds, not only of the trachea, but also of the larger and medium-sized bronchi. Microscopical examination shows a finely fibrillar or meshed basis-substance, with considerable granular, amorphous exudation, pus-corpuscles, and altered epithelium. Expectoration of these shreds and cylinders occurs in about half the cases. Not infrequently the mother removes them with the fingers from the mouth. Great improvement is always noticeable immediately after their expulsion, especially when the pieces are large and cylindrical. But no faith should be placed in such remissions, as these cases, with few exceptions, terminate fatally. The expectoration of cylindrical exudation shows that the process involves the trachea and large bronchi, and small, dichotomously branched cylinders leave no doubt of the existence of bronchial croup of the medium-sized and even smaller tubes, so that they have an unfavorable prognostic significance, as the fatal termination is more certain the deeper the

croup extends. In addition, we must remember that the exudation may be reproduced within a few hours and again cause orthopnœa.

There is nothing characteristic in the febrile movement of croup. As a rule, it is moderate, with nocturnal exacerbations to 39.5°, while the morning temperature is 38°–38.6°; but there is no lack of cases in which the thermometer rises to 40° or more. Inflammation of the pulmonary tissue appeared to me to possess special influence in this respect. The originally strong pulse becomes progressively weaker, is often irregular and intermittent in the last stages, while the cyanosis reaches the highest grade, and the face, hands, and feet are covered with cold perspiration. Finally, in consequence of deficient respiration and carbonic acid poisoning, the child becomes somnolent, the eyelids are half-closed, respirations superficial, the stenotic sound becomes weaker, and the child dies in collapse, occasionally with convulsive twitchings in the facial or other muscles. The anæsthesia, upon which stress has been laid by Bouchut, is not characteristic; it is simply due to the terminal stupor.

The opinion that croup is absolutely incurable without tracheotomy is not by any means correct. Cases occasionally occur in which the most threatening croup-symptoms disappear without operation after the expectoration of pseudo-membranous shreds has removed all doubt with regard to the diagnosis. But, even after all the threatening symptoms have disappeared, the prolonged disturbance of respiration and the defective oxidation of the blood may lead to serious disturbances of cerebral activity, either because the blood which nourishes the brain does not recover its proper constitution with sufficient rapidity, or because venous stasis in the cerebral veins, and œdema of the pia mater or serous effusion into the ventricles, is produced.

A boy, aged eight, who had passed through an attack of croup lasting five days, and who was completely aphonic, remained as pale as a cadaver and extremely weak, despite the return of appetite. On the fourteenth day from the beginning of the disease the boy became somnolent, and died in thirty-six hours. Autopsy.—Larynx normal, with the exception of slight injection and swelling of the mucous membrane; brain extremely anæmic, and considerable serum in the ventricles and the meshes of the pia mater.

This was not a case of diphtheritic collapse, but a sequel of primary croup. It cannot be denied that the vigorous antiphlogosis formerly resorted to, in combination with the anorexia and the consequent deficient nutrition, may aid in producing such weakness and anæmia.

The same rules apply to the treatment of croup that I have already mentioned with regard to acute laryngeal catarrh. If local bleeding, emetics, tartar, stibiat. in refr. dosi, the energetic use of mercurials, and the application of a blister to the region of the larynx, do not produce rapid improvement, nothing can be expected from medicinal remedies. The more reliance we place upon emetics in this disease, the more unpleasant is it that they not infrequently fail to act. In such cases the sulphate of copper, 0.03–0.1, every ten minutes, may still produce an effect. I must, however, warn you against the frequent repetition of emetics in an already exhausted child, because, without producing any good effects, it increases the inanition to an extreme degree, and may lead to severe cerebral symptoms. I would also recommend that the children be not constantly kept in bed, but frequently carried around in the arms, as this produces temporary relief. Broth, milk, or wine should be frequently, but cautiously given, in order to maintain the child's strength.

The occurrence of the first threatening attack of suffocation, or even the forcible action of all the accessory muscles of inspiration, is, for me, the signal for tracheotomy. Too long delay only increases the exhaustion, the danger of carbonic acid poisoning, and the development of broncho-pneumonia. In my experience the chances for the success of tracheotomy are more favorable in simple primary than in diphtheritic croup. Even the presence of bronchitis or pneumonia does not constitute a contra-indication, as I have seen several cases recover, despite these complications. But, as the operation only seeks to secure the entrance of air into the lungs, it is always well to continue mercurial treatment afterward to a moderate extent, and, by the inhalation of warm vapor of water through the canula, to facilitate the exfoliation of any pseudo-membrane which may still be present on the mucous membrane. I have not tried any other methods, such as cauterization with a concentrated solution of nitrate of silver or the introduction of a tube into the larynx.

V. Bronchitis and Catarrhal or Broncho-Pneumonia.

Among the most frequent diseases of childhood are the catarrhs which spread from the bifurcation of the trachea over the mucous membrane of the large and medium-sized bronchi. They are most frequent during the period of first dentition, and this is regarded by many physicians as a cause of the catarrh. I have already remarked (page 68) that this influence is very much exaggerated; but it cannot be denied that, in many children, the eruption of every group of teeth is accompanied by a catarrh. Perhaps the great frequency of rachitis at this age plays a part in this connection, as such children present an unusual tendency to bronchitis.

A peculiar form of tracheal and bronchial catarrh is often observed during the first months of life. The little patients suffer from a frequent, hacking cough, which can be immediately produced by pressure upon the site of the bifurcation of the trachea; or even more frequently from stertor, which accompanies inspiration and expiration almost constantly. It depends upon the quantity of mucus present whether the stertor is rattling or dry, like that of the croupy sound; after coughing, it becomes weaker, or disappears entirely, but soon returns. Physical examination shows large mucous râles or rattling, especially between the scapulæ; rude breathing is alone present for a time after coughing. The patients may feel entirely well, but are usually somewhat pale. Fever is absent, the appetite good. I have occasionally been able to attribute it to catching cold immediately, or shortly after birth, either from a cool bath, or cold room, or from being carried out in bad weather. All the cases observed by me were very obstinate; many weeks, even months, elapsed before recovery occurred. The chief element in treatment consists in protecting the children from the influence of cold and damp, and, at the same time, enabling them to breathe pure air. I have seen scarcely any effect from drugs, but some good results from repeated small blisters over the manubrium sterni; they should be allowed to heal as soon as vesicles have formed. If internal remedies cannot be dispensed with, you may try small doses of sulphur aurat. (0.01 four or five times a day).

Tracheal and bronchial catarrh of children up to the fifth year differs from that in adults only in the fact that the former has a much greater

tendency to rapid and dangerous extension into the smaller bronchi. The endeavor of many mothers to give their children as much fresh air as possible often leads them to take children with a cough into the air during bad weather. As a rule, the children then present, for days or even weeks, merely the symptoms of a simple catarrh, until a fresh cold either produces the laryngitic conditions described above, or, more frequently, true bronchitis. We then learn that the cough has suddenly become more severe, the respirations shorter, the expirations moaning, the skin hot, and we can usually make a diagnosis of bronchitis, or broncho-pneumonia, before examination.

However different the severity of these diseases may be, the cough remains one of the most prominent symptoms. In many children it appears to be painful, as shown by crying and painful distortion of the face. The cough is usually frequent, short, and dry, and is increased on crying. Children who can cry for a long time without coughing do not suffer from bronchitis. In the severe form there are occasionally violent coughing-spells, associated with a dark red color of the face. The sputum is swallowed by young children, and is rarely ever expectorated. The number of respirations exceeds the normal to a variable degree, according as the inflammation extends more or less deeply into the bronchial tubes. Forty to fifty respirations per minute is a moderate amount for young children, and indicates that the inflammation is situated in the large and medium-sized tubes, while implication of the small and the finest bronchi gives rise to 60 to 80, or even more respirations per minute. The more rapid the respirations, the more superficial do they become; the auxiliary muscles of inspiration (alæ nasi, scaleni) are brought visibly into play, the head is moved with each respiration, and there is distinct inspiratory depression of the lower part of the throat, the lower part of the thorax, and the epigastrium. Each expiration is accompanied by moaning (*vide* page 4), which I regard as one of the most valuable symptoms in the diagnosis of serious respiratory diseases. Not infrequently we can hear, even at some distance from the thorax, wheezing sounds during respiration, and almost always, on auscultation, whistling, sonorous, or large, medium and small moist râles, which are either confined to the posterior surface, especially its lower part, or also extend over the anterior and lateral surfaces. The distribution of the râles is of less importance than their character. For example, we may hear sibilant and sonorous râles over the entire chest, although no marked dyspnœa is present, while fine, or even medium moist râles, which are heard not only behind, but also forward over a large area, cause serious alarm. Occasionally only inspiration or expiration is accompanied by râles, while, in other cases, both are. The percussion-sound is normal in the beginning. The local symptoms are always associated with fever of varying intensity, the temperature usually varying from 38.5° to 39.5°, and perhaps reaching 40.0° at night. Not infrequently I have found the morning temperature approximately normal (37.8°–38.0°), while there were evening exacerbations to 40.0°. To the frequency of the pulse, which varies from 120 to 180, I attach no special importance, but much more to its quality, though this is usually normal when the disease runs a favorable course. The changed relation between the frequency of the pulse and respirations is always of the greatest importance, as there are no longer three to four pulse-beats to one respiration, as in the normal condition, but the number of the latter is disproportionately increased—for example, 60 to 70 respirations to 144 pulsations (page 4). In the milder grades the other functions of the

body may be intact, but I have frequently observed a complication with diarrhœa, especially at times when epidemic intestinal catarrhs prevail. When the severity of the affection increases, the appetite suffers and nursing is interfered with, as the child must stop often in order to take breath. This is so characteristic of the higher grades of the disease that I advise you to have the child put to the breast in your presence, in order to convince yourself of the manner of nursing.

Whether broncho-pneumonia is present in addition to the bronchitis cannot be determined with certainty, nor can it be positively denied. The explanation of this fact is found in the following anatomical conditions.

The mucous membrane of the bronchi, to a variable extent, often into the small ramifications, appears reddened, either uniformly or in stripes, and is loose, thickened, occasionally eroded here and there; the lumen, especially in the lower lobes, is filled with a tough, yellowish white mucous secretion, and, if the disease has lasted for a long time, perhaps moderately dilated as far as the peripheral ramifications. In a number of cases there is more or less extensive inflammation of the finest tubes (capillary bronchitis), and purulent mucus can be squeezed from the cut surface at numerous points, which indicate cut sections of the finest bronchi. Under these conditions the inflammation extends to the terminations of the finest bronchioles and the pulmonary alveoli, which are occasionally visible under the pleura as whitish yellow, tubercle-like granulations as large as a millet-seed, and give exit to a few drops of purulent fluid on section (*bronchite vésiculaire* of the French). More frequently we find broncho-pneumonic spots, which at first assume a lobular form corresponding to the territory of the inflamed small bronchi. These spots are most frequent in the lower lobes, and appear as firm infiltrations of a reddish brown or more grayish color, from the size of a pea to that of a bean or hazel-nut. At first separated by interspaces of hyperæmic parenchyma containing air, they approach one another when larger numbers are present, and finally coalesce into extensive hepatizations. These extend upward generally in a wedge-shape from the base of both lower lobes, but are observed quite often in the upper lobes, especially in the lingula of the left upper lobe overlapping the pericardium, and may finally involve a whole lobe, or even the larger part of a lung (*pneumonie lobulaire généralisée*). The cut section of these foci, which sink in water, permits the exit of an extremely small quantity of fluid on pressure, and the microscope shows that the alveoli are filled with fatty epithelium and numerous larger and smaller lymphoid cells, which also undergo fatty degeneration and then impart a grayish yellow color. According to recent investigations (Charcot, Cadet [1]), fibrinous exudation is almost always demonstrable. Congestion of the surrounding capillaries and infiltration of the interstitial connective tissue is never absent. Emphysema of the borders or other intact portions is usually present, and not infrequently more or less extensive pleurisy and hyperplasia of the tracheal and bronchial glands.

It follows that broncho-pneumonia can be determined by physical examination only when the foci are so numerous or confluent that the intervening air-containing parenchyma is no longer capable of masking the symptoms of solidification. As soon as the infiltration extends over a

[1] Cadet de Gassicourt: Traité Clinique des Maladies de l'Enfance, I., S. 152. Paris, 1880.

larger part of the lung, you will find corresponding dulness on percussion, small, moist, tinkling râles, bronchial breathing and bronchophony—signs which are at first generally noticeable on both sides of the spine, from the base of the lung to the spine of the scapula, but not infrequently also at the apices, and especially over the lingula of the left upper lobe. I have repeatedly heard fine, tinkling râles over the heart earlier than over other parts. It is a noteworthy fact that tinkling râles and diffuse bronchophony may also be present without distinct dulness; the percussion-note remains normal or is even tympanitic, and this can be explained only by the fact that air-holding parenchyma is still present at the periphery of the lung, while auscultation already distinguishes the signs of the deeper infiltration. Under these conditions I would advise you to percuss very gently (page 3), as, on vigorous percussion, slight dulness may be concealed by the resonance of the normal lung. As more or less broncho-pneumonic spots are present in every extensive bronchitis in early childhood, we may assume that even the absence of physical signs does not exclude their presence, while extensive confluent infiltration may be diagnosticated whenever these signs can be detected, though only on auscultation.

In many cases extremely few or no râles are present, despite the existence of severe dyspnœa; percussion is normal, and an extremely rude respiratory murmur is present over the entire chest, either lasting several days until moist râles indicate more profuse secretion, or continuing until death, which usually occurs in a few days:

A child, aged eleven months; respirations, 72; pulse, 160. Normal percussion, rude respiratory murmur over the entire chest; on the right side, posteriorly at the base, were a few fine, moist râles. This condition lasted three days, when the respirations diminished to 56, the pulse to 130, the cough became looser, and stertor and diffused mucous râles soon developed. A rapidly fatal case occurred in another child, aged eleven months, who was taken with cough, and two days later presented an unusually rude respiratory murmur over the entire thorax, with here and there a few râles. At the autopsy I found a number of atelectatic spots, the smaller bronchi leading to them being filled with muco-pus. All the other bronchi were free of secretion, but the mucous membrane, from the bifurcation into the smallest branches, was markedly reddened and thickened.

Bronchitis without muco-purulent secretion may therefore endanger life by rapid hyperæmic swelling of the mucous membrane and the consequent narrowing of the lumen of the tubes (so-called *catarrh sec*). Rilliet and Barthez (loc. cit., I., 454) draw attention to analogous conditions of other mucous membranes, the nose, larynx, and trachea, whose rapid catarrhal swelling produces the well-known symptoms of acute coryza and pseudo-croup (page 134), and mention the case of a child, aged one year, in whom during life merely a considerably intensified puerile respiratory murmur was heard over the entire chest, and severe redness and swelling of the bronchial mucous membrane, but no fluid secretion, were found at the autopsy.

The deeper the inflammation extends into the finer branches of the bronchi, and the greater the number and extent of the broncho-pneumonic foci, the more will the respiratory process and the oxygenation of the blood be interfered with. All the efforts of the inspiratory muscles are insufficient to force the air into the alveoli, through the small bronchi filled with muco-pus, thus explaining the presence of numerous atelectatic spots in such cases. The respiratory insufficiency of the lungs is thus still further increased, and even the great frequency of the respiratory movements

(occasionally one hundred per minute) is unable, on account of their superficial character, to replace their deficient depth. Respiration is often irregular in so far that, for example, ten to fifteen respirations follow one another with extreme rapidity, and then a short pause occurs, reminding us somewhat of the Cheyne-Stokes phenomenon. The venous stasis—a natural consequence of the pulmonary infiltration and consequent overfilling of the right heart—soon causes cyanotic discoloration of the pale face and the visible mucous membranes, enlargement of the peripheral veins, occasionally slight œdema of the eyelids, the dorsal surfaces of the hands and feet; and the constant decrease in the power of the heart is shown by the smallness of the extremely frequent pulse, and by the diminution of temperature in the extreme parts of the body. The power of coughing also grows weaker, and I always regard it as an extremely unfavorable sign when the previously distressing paroxysms of cough grow weaker or cease, while auscultation reveals widely diffused mucous râles. At this stage, carbonic acid poisoning soon occurs. Somnolence, with half-closed lids and eyeballs rolled upward, occasionally also partial or general convulsions, put an end to the distressing condition.

The febrile movement presents a remittent, in nowise characteristic type, with exacerbations at night which not infrequently reach 40°, and numerous fluctuations. These fluctuations are due to the fact that the inflammatory process spreads from the bronchioles to hitherto intact lobuli, while it may be resolving in other places. In small, especially in feeble children, the fever may have very slight importance; in a syphilitic child, aged ten days, I found the temperature usually subnormal—a new proof of the fact that, under such conditions, the tendency to collapse predominates, and even considerable inflammations may run their course without fever or with subnormal temperature (page 8).

This relation changes about the middle of the first year. In a child, aged five months, suffering from bilateral broncho-pneumonia, the temperature repeatedly rose to 40° to 40.4, the frequency of the pulse being two hundred and sixteen per minute.

The diminution in the frequency and an increase in the depth of the respiratory movements, is the first favorable sign, but the disease always lasts a long time, and never ends with a true crisis. Rapidly fatal cases are exceptional, and even then it can usually be shown that a bronchial catarrh preceded, for a considerable time, the suddenly fatal increase to capillary bronchitis and broncho-pneumonia. On the average, the disease lasts two to three weeks, and often much longer. There is an undeniable tendency to a subacute or chronic course, so that many weeks, or even a few months, may elapse before a decided turn for the better occurs. The fever then diminishes considerably, the dulness on percussion subsides more or less, and almost complete euphoria develops, but the cough, fine râles, and frequent respirations, still indicate the continuance of the disease. Much more frequently, cases running a chronic course finally terminate fatally. In certain of these cases I have observed intervals, perhaps lasting for weeks, in which there was no febrile movement whatever, and in which the child acquired a better color, coughed less, and appeared to be on the road to recovery. But the continuance of an abnormal frequency of the respirations (50–70 per minute) was always a bad symptom. We should, therefore, not be induced to give a good prognosis on account of such intervals; the continuance of the fine râles, especially posteriorly, and the increasing emaciation of the children, warn us to exercise caution. In a number of these cases I found at the autopsy, in

addition to chronic bronchitis and broncho-pneumonia, fatty degeneration of the heart, with dilatation of its right side, especially when the disease had been complicated with whooping-cough. The great resistance to the function of the right ventricle, on account of the infiltration of the pulmonary tissue and the frequent paroxysms of whooping-cough, must be regarded as the cause of this degeneration, which occasionally produces fatal syncope.

In broncho-pneumonia, which has lasted for weeks or months, the interstitial connective tissue surrounding the alveoli, and separating the individual lobules from one another, is found to be sclerotic, the smallest bronchi passing through the infiltrated parenchyma are dilated, and there are occasionally small pulmonary abscesses, produced by rupture of the alveoli, which are distended with young cells and epithelium, and coalesce into larger cavities filled with a puriform fluid. The latter cannot be diagnosed during life on account of their small size. Under unfavorable conditions, chronic broncho-pneumonia not infrequently terminates in cheesy degeneration of the infiltration.

All influences which can produce irritation of the respiratory mucous membrane, also play an important part in the etiology of bronchitis and broncho-pneumonia. In the first rank belongs the irritation of cold, of the sharp east and north winds, which sometimes produces an almost epidemic diffusion of the disease. Again, the disease very often develops in combination with certain infectious diseases, especially measles and whooping-cough, and next diphtheria, particularly when it extends to the larynx and trachea. Whether tracheotomy is performed in such cases or not, broncho-pneumonia constitutes one of the gravest complications, the existence of which we should at once suspect if the previously normal respiratory frequency suddenly rises to 50° to 60° per minute. In measles, the broncho-pneumonia may occur in the stage of eruption, but it develops more often and more seriously after the disappearance of the exanthem and the subsidence of the fever, and then constitutes one of the most grave complications. This is also true of whooping-cough, with which it may be associated at any period of its course. The disease occurs much more rarely with scarlatina and variola, but the complication with broncho-pneumonia is observed not infrequently in typhoid fever, which is almost always accompanied by bronchial catarrh. The cases in which the disease is associated with these infectious diseases often run an unusually protracted course, and give rise to the suspicion of a tubercular or cheesy affection of the lung on account of the weakness, emaciation and protracted remittent fever. The frequency of the respirations, the distressing cough, the catarrhal and tinkling râles, resist all treatment for weeks, while dulness on percussion may be entirely absent, or it may subside in places where it had been previously present, and appear in hitherto unaffected parts. The diagnosis and prognosis vary with the daily change of condition, until finally, after the lapse of many weeks or months, either the fever diappears and the symptoms subside, or death occurs with phthisical symptoms from cheesy degeneration of the infiltration.

A. N——, aged twelve, attacked with typhoid fever in beginning of December, 1873. Severe cough and frequent respirations from the start. On the twenty-fourth day, threatening symptoms of collapse were relieved in a few hours, but the cough continued. Dulness on percussion on the right side posteriorly, from the apex to the spine of the scapula; bronchial breathing, bronchophony, fine, moist râles; mucous râles on the left side posteriorly. Nocturnal fever; pulse, 120–132; hectic flush on the cheeks. The threatening symptoms gradually disappeared under the use of simple expectorants, cod-liver oil, and nourishing diet. Complete recovery by middle of February.

I have observed three other cases in which the broncho-pneumonia lasted for months, with symptoms of progressive phthisis, and which recovered entirely under tonic treatment (nourishing diet, wine, cod-liver oil).

Exhausting conditions must also be mentioned as favorable to the development of broncho-pneumonia. Obstinate intestinal catarrh, tuberculosis, basilar meningitis, gangrenous affections, especially noma, hold the first rank among these conditions. Scarcely a single child dies in my wards in whom more or less extensive broncho-pneumonia is not found at the autopsy, especially in atrophic and feeble rachitic children; I have often been unable to rid myself of the idea that this is due to some infectious substance inhaled from the hospital air. Every rachitic change in the shape of the thorax which diminishes its size, presents an especially bad prognostic significance, apart from any tubercular predisposition. Apparently slight catarrhs, still more bronchitis and broncho-pneumonia, may produce death as unexpectedly as organic disease of the heart.

Finally, the bronchi and alveoli may be directly affected by an irritant, viz., by the introduction of milk or other nutritive fluids into the respiratory organs. The so-called ingestion pneumonia (Schluckpneumonie) is thus produced by aspiration of fluids from the bottle, by "swallowing the wrong way" in cerebral diseases attended with stupor, and especially after tracheotomy; this method of development has also been proven by experiments upon animals (section of the vagus or recurrent laryngeal nerve). But only the positive discovery of nutritive substances or other foreign bodies in the air-passages, places this manner of development beyond a doubt. In many children there is a well-marked predisposition to acute catarrh of the bronchi, so that they often become affected without any recognizable cause. Such children suffer from one or more attacks every year, and, as Rilliet and Barthez[1] have remarked, "they approximate the asthmatic attacks of adults by their short duration, frequent recurrence, intensity of the dyspnœa, and the slight febrile disturbance." I have repeatedly seen cases of this kind in infants, and still more frequently in the second period of childhood, when I was usually informed that the children had suffered from these attacks for years (relapsing bronchitis). The cause of this predisposition is unknown. In a portion of the cases chronic bronchitis was present, out of which the acute attacks developed; much more frequently nothing abnormal was found in the intervals.

I have frequently observed the affection begin with pseudo-croup, which then rapidly passed into bronchitis. The croupous sound in breathing soon gives way to a whistling or rattling sound, and auscultation shows either rude, indefinite breathing or sibilant and mucous râles. The dyspnœa is severe, the respirations 60 to 80, the pulse very rapid, the color pale or cyanotic, and the entire appearance so threatening, that the inexperienced physician considers the child lost. This fear is only justified if physical examination shows the presence of extensive broncho-pneumonic infiltration. But this has always been absent in my cases, and the fact that the attack usually reaches its end within a few days, and passes into an ordinary loose catarrh, induces me to believe that this is due, as in pseudo-croup, to rapid catarrhal swelling of the mucous membrane, which, extending into the medium-sized bronchi, stenoses their calibre.

Treatment.—Simple catarrh recovers spontaneously if the child is kept in the room, but two to three weeks almost always elapse before the disease has completely disappeared. The use of inf. rad. ipecacuanhæ

[1] I., p. 451.

(P. 16), combined with aq. laurocerasi (1.0–2.0) in severe cough, finds many supporters. I scarcely think that this remedy will shorten the duration of the catarrh, but will not deny its sedative effects on the cough; it is most serviceable when diarrhœa is present at the same time. When constipation and fever are present, I give ipecacuanha in combination with calomel (P. 17).

But if the disease is attended with great dyspnœa and high fever, more energetic treatment is necessary. The whilom customary antiphlogosis, by application of leeches to the thorax or the epiphyses of the bones of the forearm, has been almost entirely abandoned at the present time, because the loss of blood was regarded as dangerous in a disease like this, which manifests a tendency to collapse. This is true of the large majority of cases in hospital and dispensary practice, but it is otherwise in previously healthy, plethoric children. Earlier experience [1] had shown me that moderate local bleeding did not have the bad results (anæmia, collapse) attributed to it, and I cannot say that I have been more successful in the treatment of broncho-pneumonia since I have discontinued bleeding. My cautious attempts at antiphlogistic treatment in the last few years (in vigorous, previously healthy children, and at the beginning of the disease) have repeatedly furnished surprising results, whether the disease developed from an ordinary catarrh or in the eruptive stage of measles. Instead of leeches I now apply wet and, especially, dry cups (four to eight, according to the age), as these have, at the same time, a revulsive effect, and as bleeding is only employed in vigorous children, the adipose layer of the skin is suited to the application of cups. I repeat, however, that this method must be employed cautiously; the majority of the children are miserable, rachitic, weakened by other diseases, and in them only dry cups may be used.

I strongly advise hydropathic applications to the chest, from the neck to the umbilicus. A napkin or diaper is dipped in water at the temperature of the room, well wrung out, and then placed around the chest, without exercising any compression, so that the arms are free; this is surrounded by a roll of batting, and then covered with a layer of oil-silk or gutta-percha paper. When the fever is high, these applications should be renewed at least every half hour; later they may be kept for one or even two hours, and this continued for several days and nights; I have occasionally continued it for a week, the cool water being changed to a temperature of 26° to 27° R. These applications appear to me to act favorably in three ways: first, by the immediate deep inspirations following the cool application, which drive the air forcibly into the alveoli and may prevent atelectasis; second, by the derivative irritation of the skin, manifested finally by redness, papules, and desquamation of the epidermis; third, by the evaporation of water, which keeps the atmosphere around the child moist, and will be still further aided if steam is allowed to escape from a teapot in the immediate neighborhood of the bed. The applications often produce favorable perspiration, which should not, however, be allowed to become too profuse, as symptoms of collapse may develop in consequence, and disappear with the cessation of the fomentations. During the entire course of the disease it is judicious not to keep the child constantly on his back, but to change him from one side to the other, or carry him on the arm, in order to prevent hypostatic processes as much as possible.

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[1] Beitr. zur Kinderheilk., N. F., S. 173.

Among medicinal remedies, emetics have always been most highly esteemed, and I share this view in so far as it regards otherwise healthy children. When careful nursing and watching is possible, I recommend tartarus stibiatus in refr. dosi (P. 18); a teaspoonful of the solution is given every hour until vomiting occurs, and then every two hours. If vomiting, or perhaps diarrhœa, occurs after each dose, the drug must be discontinued forthwith. Should vomiting not be produced after the first three doses, I nevertheless increase the intervals to two hours, in order to prevent a cumulative effect. This plan is unsuitable for weakly children when diarrhœa is present, or in an advanced stage of the disease. When you desire, under such circumstances, to clear the bronchi and make the respiration freer, it is better to use a full emetic dose of ipecacuanha (P. 6), and avoid tartar emetic entirely. In vigorous nurslings I have, in the beginning of the disease, often successfully used an emetic[1] consisting of vinum stibiatum and oxymel scillit. (P. 19). But emetics must not be employed when the symptoms of carbonic acid-poisoning and prostration are present, as they would only increase the weakness.

As soon as numerous râles indicate the presence of profuse secretion in the bronchi, and the sinking powers prohibit the use of emetics, we may give a strong inf. rad. ipecacuanhæ (0.3–0.5 : 120), decoct. rad. senegæ or polygalæ amaræ (P. 20), to which liq. ammon. anisat. (0.5–1.5) may be added, in order to increase the expectoration. Mustard poultices to the sternum or back, small flying blisters to the thorax, are advisable at the same time. Milk, broth, wine (sherry, Tokay, port) must be given alternately to maintain the strength. If these substances are of no avail, and the loss of power progresses, we may resort to a mixture of camphor and acid. benzoic. (P. 21), although at this stage the remedy often proves useless in preventing carbonic acid-poisoning. Under such circumstances lukewarm-water baths, with cold affusions, repeated a few times daily, occasionally have surprising results.

A few words, finally, with regard to the treatment of relapsing bronchitis (page 149). During the attacks, the treatment is the same as that just described; but, in order to prevent the recurrence of the disorder, the use of salt baths in a climatic watering-place, especially in Reichenhall, is advisable. This course must be repeated several times, and then followed by a visit to the sea-shore, especially the North Sea (Norderney, Ostende, Blankenberghe, Scheweningen, Helgoland). I do not think it proper to advise sea air from the beginning, as the irritability of the mucous membrane not infrequently reacts by a fresh attack.

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VI. "Fibrinous" Pneumonia.

The former view with regard to the frequency of this form has long been exploded. It is quite frequent between the third and twelfth years, and is by no means rare in the first two years of life. Among forty-four cases observed in my clinic, there were:

10	from the age of		½ to	3	years.
12	"	"	3 to	6	"
22	"	"	6 to	12	"

[1] My experience with apomorphine has not been sufficiently large to justify a decisive opinion. The few experiments which I made were unsatisfactory, but referred to very severe cases of broncho-pneumonia.

Among these were twenty-four boys and twenty girls. Twelve cases occurred from May to August inclusive; thirty-two from October to April inclusive.

The disease is so similar to the pneumonia of adults, that I will only refer to the peculiarities caused by age. In fibrinous pneumonia the alveoli are filled with an exudation consisting chiefly of coagulated fibrin, and, in catarrhal pneumonia, by a mixture of degenerated epithelium and young cells. The latter always begins in isolated lobular spots, which coalesce, while the fibrinous form attacks a large part of the lung, even an entire lobe, with one blow, so to speak. The symptoms correspond, in general, to these anatomical differences. In the fibrinous form we find a rapid, almost sudden development, with severe febrile disturbance. Among seventy-four cases observed by me, the affection was situated in:

The entire right lung	in	2 cases.
Both lower lobes	in	2 "
The left upper lobe	in	4 "
The right upper lobe	in	21 "
The left lower lobe	in	27 "
The right lower lobe	in	18 "
		74

The differences mentioned above will only hold good in a general way. There are mixed forms, even from an anatomical standpoint. In opposition to Bartels and Ziemssen, Steffen[1] acknowledges the possibility that the product of lobular pneumonia may be croupous in character. Steiner and Damaschino[2] describe such foci, which were found in the same lung with broncho-pneumonic spots, and Virchow admitted at an earlier period that, in addition to cell-proliferation in the alveoli, fibrinous exudation may also occur in consequence of a higher grade of irritation. I have also seen analogous cases. The clinical history is not always as clear as we would suppose from the descriptions of authors. If, for example, physical examination shows extensive pneumonic infiltration of the right lower lobe, and, at the same time, a catarrh of the left lung, you should remember that in broncho-pneumonia there may be widespread coalescence of the foci in one lung, while in the other they may remain separate. On the other hand, the accompanying catarrh is not always characteristic of broncho-pneumonia, as I have not infrequently had an opportunity of observing fibrinous pneumonias which were complicated with bronchial catarrh.

The febrile movement always remains a valuable symptom in these doubtful cases. I subscribe to Ziemssen's conclusions[3] with regard to the regular course of the fever in fibrinous pneumonia and its relations to the critical days, while "a protracted course with, later, considerable fluctuations in temperature; with the continually recurring increase of fever which always corresponds to an advance of the local process; with the slow diminution of the fever interrupted by slight exacerbations; with the delayed resolution of the infiltration," is peculiar to the catarrhal

[1] Klinik der Kinderkrankh., I., S. 146.

[2] Des Différentes Formes de la Pneumonie Aigue chez les Enfants, p. 29. Paris, 1867.

[3] Pleuritis und Pneumonie im Kindesalter, S. 316, 1862.

form. This is undoubtedly true in the majority of cases, but not by any means in all. Not every croupous pneumonia ends with a crisis, but it may assume a more protracted course, and, on the other hand, I have occasionally observed pneumonias which presented a complete history of the catarrhal form, but ran an unexpectedly rapid and favorable course within five to eight days. Between a well-characterized case of lobar pneumonia on the one hand and broncho-pneumonia on the other, lies a transitional form which cannot be determined clinically with entire certainty.[1] I must, therefore, give a negative answer to the question whether it is possible to distinguish the two forms of inflammation of the lungs in every individual case during life. But I do not consider it advisable to substitute other terms (circumscribed and diffuse, according to Steffen, primary and secondary, according to Rautenberg) for the usual classification into a fibrinous and catarrhal form, because, in the majority of cases, the old terms are correct. Especially do I consider the classification proposed by Rautenberg as incorrect, because primary, *i.e.*, idiopathic, as well as secondary pneumonias, which develop in the course of some other acute or chronic disease, may present a fibrinous character. Thus I have not infrequently found well-marked lobar hepatization in children suffering from tuberculosis or cheesy degeneration of internal glands or other organs. This also occurs occasionally in acute infectious diseases, especially in measles. But I was most surprised by the case of a girl, aged twelve, suffering from severe typhoid fever, whose high temperature yielded to no antipyretic measures, but presented a continuous type of 40° and more until the end. At the autopsy, the entire left lung was found hepatized, and within it were found two isolated sequestrated foci, surrounded by a line of demarcation (dissecting pneumonia).

I have previously remarked that fibrinous pneumonia may develop out of a catarrh, whether acute or chronic, and that in this event catarrhal sounds are heard in the affected as well as in the healthy lung. But, in the large majority of cases, pneumonia begins suddenly; I have occasionally noticed the initial chill in children over five years, and more frequently repeated vomiting. This onset, and the rapid rise of temperature (up to 40°, in one case 41.2° during the first night), may lead to error, as the respiratory symptoms may be entirely latent and cerebral symptoms appear, especially somnolence, delirium, dark red face, glistening eyes. Slight pains in the throat, with congestion of the pharynx and gums, are often present in the beginning, and a slight redness of the skin, which is usually only partial, confuses the physician so much the more. Under such circumstances, we should pay special attention to the character of the respiration, which is hurried in comparison to the pulse, while the expiration is moaning in character. Examination of the chest either gives negative results or, at the most, shows diminished vesicular breathing in the affected portion. This latency of the physical signs, which may continue from four to six days, in connection with the predominance of cerebral or gastric symptoms, readily leads to the erroneous diagnosis of meningitis or the beginning of typhoid fever. In such cases the pneumonia probably develops gradually from the centre of the lung to the periphery, and only after it has reached the latter do the signs of consolidation distinctly appear. As soon as this occurs, the gastric or cerebral symptoms usually subside, and the diagnosis becomes clear at once, occasionally only after the fever is decidedly diminishing. I do not agree

[1] Vide Steiner: Prager Vierteljahrsschr., III., S. 12, 1862.

with the opinion of some authors, that pneumonia of the upper lobes especially leads to such mistakes. The cerebral symptoms inaugurating these cases (pneumonie cérébrale of Rilliet and Barthez) appear most frequently, in my experience, in a typhoid form, as apathy, somnolence, vertigo, delirium, dry tongue, and more rarely as attacks of epileptiform convulsions.

P. S——, aged four, admitted July 7, 1864. Continued fever and cough since the day before yesterday. On the 6th, repeated attacks of eclampsia. Pulse, 152; respiration, 64. Percussion normal; a few râles on the right side anteriorly. July 8th (fourth day of the disease), severe headache on the right side, above and posteriorly, with indistinct, later bronchial, breathing. Crisis on the seventh day.

A. H——, aged four, admitted May 11, 1877. Sick since yesterday. Headache and anorexia. Yesterday afternoon, suddenly severe general convulsions. Delirium during the night. Great apathy, the eyelids half-closed, pupils somewhat dilated. Temperature, 40.1°; pulse, 152; respiration, 42, very superficial, and somewhat dyspnœal. Cough scarcely noticeable. May 13th, increasing clearness of the sensorium with marked dulness on the right side below, posteriorly, and fine mucous râles. May 15th, complete crisis. May 18th, dulness considerably diminished. May 21d, everything normal.

H. S——, aged six, admitted February 4, 1878, on account of leucorrhœa. February 23d, suddenly slight angina, with temperature 39°, in the evening, 41.2°; pulse, 150. Coma developed at the same time, with severe twitchings of the muscles of the eyes, face, and extremities, continuing twenty minutes. February 24th, temperature 40.9°; angina continues; respiration, 60, and superficial. Catarrh on the left side, dulness over right supraspinous fossa, indefinite breathing, tinkling râles; later, bronchial breathing, continued high fever, sensorium clear. Crisis between the sixth and seventh days.

O. S——, aged seven. January 16, 1874, suddenly taken sick at night with fever and vomiting. January 17th, continued somnolence; apathy, from which the child can be readily roused. Temperature constantly 40° or more. January 19th, continued fever (41.8°), apathy, redness of face, and injection of conjunctivæ, crusted lips, dry tongue; respiration 40, slight cough. Dulness on the left side posteriorly and laterally, bronchial breathing, and bronchophony. January 20th, fever diminished to 38.5°, with general improvement, but rose on January 21st to 40°, with more marked dyspnœa. Respiration, 60. Crisis on the seventh day.

Opinions differ with regard to the cause of these initial cerebral symptoms. I believe that the more typhoid symptoms (vomiting, vertigo, headache, apathy, somnolence) can be attributed to the rapid rise and continued height of the temperature, and that convulsions may develop in this manner in case of a special predisposition. I have never been able to detect any connection with otitis, to which Steiner calls attention, and the existence of meningitis can be accepted only when the symptoms are not only present at the onset, but continue throughout the entire course of the disease until death. It is well known that cerebro-spinal meningitis may be combined with pneumonia, but the symptoms of the former are always predominant, and the latter appears only as a complication.

With the gradual development of pneumonia from the centre to the periphery, is connected the form described under the term "pneumonia migrans," which I have repeatedly observed in children. In this form the infiltration spreads in sudden jumps from the primarily affected part of the lung, and, after a while, may affect an entire lung.

A. S——, aged seven, admitted February 8, 1874, with pneumonia of left lower lobe and high fever (40.5°–40.9°). On the next day dulness and râles on left lateral surface; respiration, 76–84; pulse, 144–150. February 13th (tenth day of disease) the infiltration was resolving behind, while dulness and bronchial breathing extended in front to the clavicle. Crisis on the eleventh day. February 19th, complete recovery.

In this case the progress of the pneumonia could be followed from the lower part of the lower lobe over the lateral surface forward to the apex of the lung. This process continued five to six days. On the evening of February 12th the temperature, which had been falling, again rose suddenly to 40.1°, evidently in consequence of the final infiltration in the apex, with which the disease reached its end. On this occasion I may remind you that, in pneumonia of the upper lobe, dulness may soon develop at the base; this is due to pleuritic effusion which has flowed from the apex (Traube). The complication with pleurisy is as frequent as in adults, and is manifested in older children by complaints of pain or coughing, and on percussion and palpation of the intercostal spaces. As a rule, the pleurisy does not attain any considerable intensity. In only two cases did I see empyema gradually develop out of a pneumonia. The radical operation was performed, and recovery occurred. In one patient the pneumonia was primary, in the other it occurred during scarlatinous nephritis. The differentiation of pneumonia from pleuritic effusion is rendered difficult, in little children unable to speak, by the fact that vocal fremitus and the rusty sputum are here absent. The former should be determined when the child is crying violently. Only after the third year have I been able to determine increase or diminution of vocal fremitus so distinctly as to enable diagnostic conclusions to be drawn therefrom. I have observed rusty sputum almost exclusively in children of eight to twelve years; in one case, however, at the age of four and one-half.

By far the larger majority of cases terminate favorably in a complete crisis (36 times among 44 cases), more rarely (8 times) gradually. The crisis was observed 29 times between the sixth and seventh days, 5 times on the eleventh, 5 times on the ninth, 3 times on the fifth, and twice on the third day. It was usually, though not always, accompanied by copious perspiration, occasionally associated with symptoms of collapse, restlessness, cold extremities, pallor of face, very frequent and small pulse, so that stimulants became necessary. In almost all my cases the initial period of the crisis could not be determined definitely, as it often occurred during the night. It therefore remained doubtful whether the sudden depression of temperature took place at the end of an even or the beginning of an uneven day. I have repeatedly observed, in the course of pneumonia, that the continued high fever sank temporarily between the third and fifth days—for example, from 40° to 38.8°—but reached its former height after twelve to twenty-four hours, and the crisis occurred a few days later. It remained undecided in these cases whether this was due to a fresh pneumonic infiltration.

The crisis was not always complete, and I have frequently observed it spun out for a certain length of time, perhaps about twenty-four hours, for example, in the following manner:

A. P——, aged seven, admitted March 8, 1875, with pneumonia of the left lower lobe.

	Morning.	Evening.
March 8th	....	40.5°
" 9th	41.5°	40.9°
" 10th	40.3°	40.6°
" 11th	39.4°	39.6°
" 12th	39.3°	40.1°
" 13th	38.8°	39.4°
" 14th	36.0°	36.1°—Absence of fever.

A sudden, inconsiderable, purely ephemeral rise of temperature (38°–39°) may occur in the first few days after the crisis. During convalescence I have often noticed an irregular pulse, especially in the beginning, on sitting up.

One fatal case was remarkable for its fulminant course:

A boy, aged four, who had been successfully treated in the latter part of 1873 for diphtheritic nephritis. He had been convalescent for two weeks; temperature 36.9° On December 9th, in the evening, sudden sickness; temperature, 39.1°; pulse, 158. Constant cough, increasing dyspnœa. After a few hours, dulness on percussion, indefinite breathing, and râles on right side below spine of scapula Death in dyspnœa in morning at three o'clock. Autopsy showed hepatization of right lower lobe. Heart somewhat enlarged and pale. Unfortunately, the latter was not examined microscopically, as it seems probable to me that the rapid death was due to fatty degeneration of the heart, in consequence of diphtheria.

Examples are not wanting in literature, in which the disease lasted only one day in adults. The physical signs may also disappear within a few days after the crisis. In the majority of cases, normal percussion-sound and vesicular breathing were restored in one and one-half to two weeks at the latest, unless pleuritic effusion still produced slight dulness at the lower part of the dorsal surface. In one exceptional case the physical signs subsided before the occurrence of the crisis.

Fibrinous pneumonia, if it does not occur under very unfavorable circumstances (nephritis, typhoid fever, tuberculosis), is one of the most favorable diseases of childhood from a prognostic standpoint. Among forty-four of my cases, only two proved fatal; in one there was hepatization of the entire right lung; in the other, double pleuropneumonia and purulent pericarditis. The coexistence of catarrh, or of an abundant pleuritic exudation, clouds the prognosis. The more rare diminution of the fever in a lysis, which may continue for several days, need not be dreaded, although in such cases the possibility of a longer continuance of the infiltration, and its passage into a chronic condition, cannot be excluded. In only one case did I observe a termination in abscess-formation with final recovery.

The expectant treatment usually recommended in the pneumonia of adults, will also hold good during the period of childhood. I only use wet cups when there is intense dyspnœa and great extension of the pneumonia, and in a complication with severe pleurisy when required on account of violent pains in breathing and coughing. But when the pneumonia is restricted in extent, and the pleuritic complication is absent, or at least does not occupy the foreground, bleeding should not be employed, and it is best to resort to cold applications or fomentations to the chest (page 150), which, so long as the high temperature continues, are renewed every half hour; later, every hour or two. The application of an ice-bag to the head or abdomen is also advisable. I cannot speak in favor of the cool or cold baths recommended by Jürgensen—in the first place, because I dread a depressing effect upon the heart, which must be avoided, especially in pneumonia; and secondly, because I consider them unnecessary. The very large majority of cases run their course without any active treatment, and there is therefore no reason to expose the children to the danger of collapse, which I have seen in the cold-bath treatment of typhoid fever. In addition, I have found as little permanent effect, when the temperarure was at its height, from the use of cool baths (20°–22° R.), as from the administration of large doses of quinine (0.5–1.0). Even if the temperature is depressed ½°–1° for a few hours, the effect is only

temporary. I could present to you many temperature curves like the following:

	Morning.	Evening.	
May 11th.....	....	{ 40.6° Bath of 20° R.	
" 12th.....9 A.M.,	39.8°	....	
" " .....12 M.,	40.3°	....	
" " .....5 P.M.,	40.5°	....	Quinine, 0.5
" 13th.....	39.6°	40.6°	Quinine, 0.5
" 14th.....	39.6°	40.5°	Quinine, 1.0
" 15th.....	40.0°	40.1°	
" 16th.....	....	{ Bath of 22° Crisis.	

I have now ceased the use of quinine and other antipyretics, and confine myself to the local application of cold. Internally I prescribe infus. digitalis and kali nitricum (P. 22), which is, however, contraindicated by any gastric complication (repeated bilious vomiting, thickly coated tongue, nausea). You may then order acid. muriatic. (P. 3), or inf. rad. ipecacuanhæ (P. 16). In very rare cases I have given tartarus stibiatus in the previously mentioned manner (page 150), when bilious gastric symptoms (constant frontal headache, vomiting, fœtor of the breath) were prominent, but it was always very successful in such cases. Moderately nourishing diet (milk, bouillon, a little wine) should be given. The collapse, which occasionally occurs at the crisis, is best combated by large doses of wine, but this accident is not often observed.

VII. Chronic Pneumonia.

Acute pneumonia does not always resolve rapidly. The physical signs of consolidation may continue for weeks, or even months, and then always lead to a fear of cheesy degeneration, necrotic destruction, and the formation of phthisical cavities. This termination occurs much more frequently in broncho-pneumonia than in the fibrinous form, when circumstances (hereditary predisposition, bad conditions of life) favor such changes in the infiltration. You will remember (page 149) that broncho-pneumonia, after running a very protracted course, may unexpectedly terminate favorably and I think that fibrinous pneumonia may pursue a similar course.

M. K——, aged six, admitted March 17, 1873, with eczema capitis and bronchial catarrh. March 19th, sudden development of fibrinous pneumonia in right lower lobe. March 25th, the temperature sank to 37.8° to 38.2°, which continued four days (lysis). March 31st, normal temperature, but cough continued and physical signs in right lower lobe slowly improved. After a few days a slight rise of temperature was noticeable at night, occasionally in the morning, so that the temperature fluctuated from 37.8° to 38.4° until April 21st. The dulness was entirely lost only during the last days of April, while indefinite breathing and râles still remained, and a remittent fever was noticeable for a few days; increasing pallor and emaciation, prolonged expiration and râles over the affected part, mucous sputa, streaked with blood, and gradually assuming a purulent character. It was not until May 27th (two months from the beginning) that everything had returned to the normal.

This slow course in fibrinous pneumonia appears to me to be more frequent in children than in adults. In the above case, there is no doubt

that the disease was originally of a fibrinous character; in others, in which the onset had not been observed, it remains doubtful whether the fibrinous or catarrhal form must be regarded as the starting-point. To these doubtful cases belong, in part, those which I have formerly[1] reported as examples of "chronic pneumonia." The children in question were from one and one-half to four years of age, though I have no doubt that older individuals may be similarly affected. They were pale, more or less emaciated, and had an expression of suffering. According to the clinical history, the scene was opened weeks or months before, with "inflammation of the lung" either primary or following whooping-cough, measles, or typhoid fever. Obstinate cough, shortness of breath, moaning expiration, and moderate fever had persisted. In addition, anorexia, coated tongue, and diarrhœa, were not infrequently present. In most cases I found signs of consolidation in the upper lobes. The fever is almost always remittent, but may also be intermittent and thus lead to a mistaken diagnosis for malarial fever. Bloody sputa, *i.e.*, punctate or streaked hemorrhages mixed with muco-pus, are repeatedly observed in such cases. Occasionally the examination shows catarrhal sounds in the other lung, and an acute catarrh is superadded from time to time to the chronic affection. The suspicion of pulmonary phthisis is always justified under such circumstances, and in fact a part of these cases terminate in this manner by cheesy degeneration and necrosis of the inflammatory products, but experience has taught me that apparently desperate cases may recover. A long time may elapse before this occurs; remains of the consolidation may be detected at the end of a year, oftener after six to nine months, while the other respiratory symptoms have entirely disappeared.

I have repeatedly convinced myself, in post-mortem examinations upon children, that broncho-pneumonic infiltrations may last many weeks and even months without undergoing cheesy degeneration, and we must therefore admit the possibility of complete absorption of the contents of the alveoli after this length of time. On the other hand, induration of the pulmonary tissue may occur from hyperplasia of the interstitial connective tissue, and the diseased process then terminates. The proliferating connective tissue then retracts, and the parenchyma becomes firm and assumes a grayish white or bluish color. Especially in young children, an entire lung or one lobe (particularly the upper lobe) may become converted into a firm mass, which creaks under the knife, and in which the whitish cords of the obliterated bronchi are distinctly recognizable. The signs of consolidation then persist during life, unless marked by emphysematous dilatation of adjacent parts. If the upper lobe is affected, the subclavicular region is somewhat flattened, and expands less in inspiration than the healthy side.

Bronchiectasis occasionally occurs in the retracted lung, and the few cases which I have observed present the same symptoms as in adults, viz., dulness on percussion, numerous large, moist, occasionally tinkling râles, flattening of the corresponding anterior surface of the chest, elevation of the diaphragm, paroxysmal cough with profuse purulent, usually fœtid sputa, mixed at times with blood.

I have little to say with regard to the treatment of chronic pneumonia. Our chief aim is to facilitate the absorption of the inflammatory products and to prevent fresh catarrhs or inflammations. Protection against catch-

[1] Beiträge zur Kinderheilk., N. F., S. 189.

ing cold, and tonic treatment with cod-liver oil and quinine, occupy the first rank. I have seen good results from the use of decoct. cort. chinæ (P. 23) or ext. chinæ frigide par. (P. 24) when continued for months. Not more than two teaspoonfuls of cod-liver oil should be given daily, so as not to produce dyspepsia. A stay in mild, pure and calm air is strongly to be recommended, and many cases, which presented at first a very bad prognosis, were completely restored by spending the winter repeatedly in Montreux or Meran, or, better still, in the Riviera. Nutritious diet is one of the chief elements, together with careful treatment of any dyspepsia or diarrhœa which may be present.

In cases of extensive retraction of the lungs, with bronchiectasis, I have repeatedly tried oil of turpentine, with either very slight success or with bad effects, as fresh catarrhal irritation, even accompanied by fever, was sometimes produced.

VIII. Pleurisy.

The old view concerning rarity of this disease in children, has long been recognized as erroneous. Not only have I very frequently found old pleural adhesions in autopsies upon young children, but I have often found pleurisy, with effusion, at the age of five to nine months, though more frequently after the end of the first year.

Acute pleurisy, with its stitch pains, short cough, rapid superficial respiration, and more or less high fever, is perfectly analogous to that occurring in adults. Older children localize the pain accurately, while those who are only three to four years old often complain of pain in the abdomen; but percussion and palpation of the intercostal spaces usually cause pain and thus draw the attention of the physician to its true site. Small children, who are unable to speak, cry during cough, but this symptom is uncertain. However, I have occasionally seen the pain entirely absent in older children.

Acute pleurisy in children begins more rarely than fibrinous pneumonia with cerebral symptoms (vomiting, epileptiform convulsions). These symptoms are only found in children from one to five years old:[1]

O. N——, three and one-fourth years of age, fell upon his forehead toward the end of October, 1846. October 30th, had sudden fever at night, and October 31st, at ten A.M., an epileptiform attack. Consciousness restored in half an hour, headache, inability to sit up, pulse 160, somnolence. Two P.M., second attack of eclampsia. Six P.M., euphoria. Constant fever at night, vomited once. Same symptoms until November 15th, when physical examination of chest (performed now for the first time) showed extensive pleuritic effusion on right side. November 15th to 27th, hectic fever, emaciation, night sweats. Gradual improvement under tonic treatment. January 10, 1847, complete euphoria.

This case, which occurred in the second year of my practice, induced me never to omit examination of the chest in any febrile disease, even though no symptoms demanded it. As in fibrinous pneumonia, the initial cerebral symptoms in these cases consist of headache, vomiting and obstruction, somnolence and delirium, or, in small children, epileptiform convulsions. Fever also appears to be the cause of these symptoms, as they usually disappear when the former subsides, and the exudation appears more distinctly. Still more frequently the disease begins with gastric

[1] Journ. f. Kinderkrankh., Bd. xiii., S. 2 , 1849.

symptoms, nausea, anorexia, thickly coated tongue, fever at night, pains in the abdomen; in two children, one of whom suffered from left pleurisy, icterus also occurred. One child, suffering from left pleurisy, complained of pain in the left inguinal region. Some cases begin subacutely or very gradually, and present at first no striking symptoms, while the later history does not lead to a suspicion of a serious affection of the respiratory organs. In my experience, these cases of latent pleurisy occur more frequently in children than in adults.

E. B——, aged seven. In the middle of January, 1874, the child began to have fever at night, and the breathing was short; condition very little disturbed during the day. Appetite gradually lost, and color became pale. I was not called until February 5th. Flatness on percussion, absence of breathing and vocal fremitus on the left side below the fifth rib; puerile breathing above. Respiratory movements normal, no cough or pain, but the child remembered that she had slight stitches in the left side at times, during the month of January. Ordered rest in bed, warm poultices to affected side, and inf. digitalis with kali acet., on account of scanty urine. February 10th, profuse diuresis, no fever, percussion less dull. March 1st, recovery.

In this, and similar cases, the fault was due to the carelessness of the parents. But I must also add that incomprehensible mistakes are also made by physicians in these insidious cases of pleurisy; the latency of pleurisy is due, not to the character of the disease, but to the carelessness of the physician.

With reference to the physical signs, which coincide with those in the adult, I will only call your attention to the frequency of bronchial breathing in the pleurisy of childhood; this is not due to any pneumonic complication, but is entirely caused by the exudation compressing the lung. Those interested in the explanation of this circumstance may refer to Rilliet and Barthez[1] and Ziemssen.[2] I merely mention the fact that bronchial breathing is heard almost constantly, especially in recent cases, over those portions of the chest which are dull on percussion, and only gradually grows weaker, and finally gives way to entire absence of the respiratory murmur when the exudation increases in amount. In young children, therefore, the absence of sputa, and the difficulty of utilizing vocal fremitus as a diagnostic factor, will always leave it doubtful whether we have to deal with pleurisy or pneumonia. If bronchial catarrh happens to be present in a recent case of pleurisy, the mucous râles occasionally assume a tinkling character, on account of the compression of the pulmonary tissue, and may give rise to a suspicion of the formation of phthisical cavities.

Most children with pleuritic exudation lie upon the affected side. This may be observed as early as the first year, and explains the preference of infants suffering from pleurisy for that breast of the mother which enables them to rest upon the affected side while nursing.

Among the complications of pleurisy, pericarditis, especially in very young children, appears to me to occur more frequently than in adults. In one case there was considerable purulent effusion into the left pleura and pericardium, the visceral layer of which, especially on the anterior surface of the heart, was covered with fibrin. The strong adhesion of the left lung to the external layer of the pericardium, proved that the inflammation had extended from the left pleura to the serous covering of the heart. In the following case there was an old encapsulated exudation in the right pleural cavity, complicated with chronic pericarditis and endocarditis:

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[1] L. c., I., p. 555. [2] L. c., p. 71.

E. P——, aged three; admitted September 18, 1872; poorly nourished, pale; history unknown. Right thorax dull on percussion throughout, except upper part of anterior surface. Sternum and left thorax normal. Respiratory murmur absent on the right side, posteriorly and laterally; indefinite above, anteriorly, with bronchial expiration. Right thorax 1 ctm. narrower than the left, scarcely moved during respiration. Cardiac dulness increased toward the right, apex-beat in fifth intercostal space and mammary line, loud systolic blowing murmur at apex. Diagnosis: mitral insufficiency, dilatation of right ventricle, old right-sided pleurisy, with retraction of thorax. The child grew worse from repeated intestinal catarrh, and the bronchial catarrh also became more severe. From January 25, 1873, mucous sputa, mixed with bright red blood, were often expectorated, and very distinct tinkling râles, loud bronchial breathing and bronchophony were heard on the right side above, next to the sternum, and also above the clavicle, where the percussion sound was somewhat more resonant. Typhoid fever developed in January, and death occurred February 7th.

Autopsy.—Strong adhesions between pericardium and left lung; the former thickened, and both layers firmly united to one another. Mitral valves thickened, rigid and insufficient; both ventricles hypertrophied, right also dilated. Old fibrous patch beneath the endocardium, 1 ctm. below the orifice of the aorta. Left lung almost normal; right lung retracted, pushed anteriorly and upward, and adherent to pericardium. On its lateral and posterior surface is a colossal sac with extremely thick, firm walls, adherent internally to the lung and externally to the thorax; it contains a creamy, grayish red exudation. Right lung carnified. Catarrh of large bronchi. Lesions of typhoid fever.

The cause of this complication of pleurisy with pericarditis and endocarditis is unknown. The retraction of the right lung anteriorly and upward (on account of old adhesions to the pericardium), was the cause of a mistake in diagnosis. On January 25th, I thought that the bronchial breathing and tinkling râles heard above anteriorly, in connection with the bloody sputa, indicated the formation of cavities in the upper lobe, while the autopsy showed that this phenomenon was caused by catarrh of the right main bronchus, and the carnified lung resting immediately upon it.

Caries of the ribs gives rise to pleurisy in children more often than in adults. The following case is an interesting illustration:

M. M——, aged five; admitted April 5th. Numerous abscesses of connective tissue from birth. On admission, an abscess as large as an apple over the sternum, enlargement of glands in neck and inguinal regions. June 7th, a round, fluctuating tumor in close proximity to right mamma; this became as large as an apple, and was opened June 20th. Fever present after this (38.5°–39.4° at night), but often remained absent for days. Another abscess formed next to the right scapula; opened July 11th, and the probe came in contact with a carious rib. Examination showed dulness over right thorax, increasing toward the base, numerous râles, and indefinite breathing. July 10th, it was first noticed that, during vigorous expiration, pus, mixed with air-bubbles, flowed from the abscess-wound of the breast; this continued until death, August 18th.

Autopsy.—Caries of the right fifth, sixth, and seventh ribs; an entrance could be made between them through some small openings in the costal pleura into a cavity. Pericardial sac obliterated, pericardium adherent to right lung, which was very firm, and adherent to thorax. Costal and pulmonary layers of pleura formed thick layers; in immediate vicinity of the abscess wound in thorax was the previously mentioned cavity, situated between the two layers of pleura, and containing four ounces of purulent fluid. Pulmonary pleura bordering the cavity was absent, so that the sound entered directly into small bronchi. Almost the whole of right lung carnified.

In this case the caries of the ribs evidently formed the starting point of the abscesses near the breast and scapula, and also of the chronic pleurisy. This gave rise to the cavity filled with pus which communicated externally with the abscess of the thoracic wall, and finally necrosed the pulmonary pleura internally. Air thus entered the cavity from the lung, and escaped externally with the pus.

Tuberculosis and pneumonia, especially the fibrinous form, are also etiological factors. The pleurisy is usually subordinate to the pneumonia, but cases also occur in which the latter yields the field, while the former continues to progress, and leads to more or less considerable exudation (pneumopleuritis).

I have nothing to add with regard to the terminations of the disease: absorption, suppuration, perforation of the empyema externally or internally, or subsequent deformity of the chest. It is erroneous to believe that chronic purulent exudations lead to deformity of the chest more rarely in children than in later life, as considerable retraction of the affected half of the thorax not infrequently occurs.

Finally, a few words with regard to treatment. At the onset, when severe pains are present, I consider the application of a number (corresponding to the age) of wet cups (dry cups in feeble children) as necessary. Hydropathic applications should also be made constantly, as in pneumonia, while inf. digitalis (P. 22) is given internally, combined with nitrate of potassa. Calomel and digitalis (P. 25) also do good service, especially when constipation is present. With the increase of exudation, diuretic treatment comes to the foreground, viz., inf. digitalis and acetate of potash, with Biliner or Wildunger water (three to four wineglassfuls daily) as a drink. In subacute cases I recommend infusion of bark (P. 23), with acetate of potash (2.0), cod-liver oil, whey, and country or mountain air.

In my opinion there are two indications for the operative removal of the exudation :

First.—Rapid increase of the exudation, the mediastinum being pushed to the other side, and the dyspnœa considerably increased, so that the children can no longer lie down, but must often assume a sitting posture. Under these circumstances, especially when the exudation is bilateral, or is complicated with bronchitis or pneumonia, I advise the early performance of puncture, in order to relieve the lung as rapidly as possible from the pressure of the exudation. As a rule, the fluid rapidly returns, but the operation may then be repeated, if necessary, or, if only moderate in amount, we may wait until absorption takes place.

Girl, aged seven, first examined on July 6, 1879. Acute pleurisy of left side for past ten days. Left thorax filled with fluid, dulness over sternum, heart pushed to the right, and left lung forced upward and backward. In the beginning of third week increase of dyspnœa, small pulse. July 11th, puncture with aspirating apparatus, which was filled four times with a clear, greenish serum. Temperature remained at 38°–39.2° until July 17th, and the exudation again increased. Then rapid absorption, subsidence of fever, recovery.

In this case a single puncture and aspiration were sufficient to produce recovery. It is noteworthy that air entered the pleural cavity during the operation, but produced no bad effect on the exudation, and was absorbed in a few days.

Second.—When the exudation is not serous, but purulent (empyema). If the fluid does not perforate externally, the greatest weight in diagnosis must be attached to the character of the fever, whose continuance for weeks, with high noon or evening temperature, and with emaciation and loss of power, correspond to the purulent constitution of the exudation. But even this sign is not constant, and as shown in the above-mentioned case, the fever may continue at least seventeen days with high noon and evening temperature, although the exudation is purely serous. The only

certain method of determining the character of the exudation is the explorative puncture with a hypodermic syringe, Dieulafoy's apparatus, or Fräntzel's trocar, and which, when performed with antiseptic precautions, presents no danger. If the aspirated fluid is purulent, expectant treatment must be abandoned, and artificial removal performed. The method of operation is still under dispute. Examples of recovery after one or more simple punctures, with or without aspiration, are increasing every year. I have seen this result in two cases of purulent exudation. This plan should, therefore, always be first adopted in children. But it will only prove successful in the smallest number of cases, and, after two to four trials, we will finally be compelled to resort to the radical operation, *i.e.*, to open the thorax by incision, with or without resection of a piece of rib. By making the wound at the base of the dorsal surface, the best outflow will be secured for the exudation, and this may be aided by the introduction of a drainage-tube or a broad silver canula. Antiseptic dressings are advisable, in order to prevent the introduction of infectious elements into the thorax, but washing the latter with carbolic acid has come into discredit, as cases of carbolic acid-poisoning have been observed in consequence. Some do not wash out the cavity; others use solutions of thymol, boracic or salicylic acids. Finally, I cannot impress it upon you too strongly to perform the operation without delay as soon as the purulent character of the exudation has been ascertained, or repeated puncture has proven insufficient.

IX. Pulmonary Tuberculosis.

The dispute concerning the theory of tuberculosis, especially with regard to its relations to cheesy processes, is not yet settled. While some consider these conditions entirely distinct, others take a more intermediate stand-point, which, as I believe, is justified by clinical facts. It cannot escape the unbiassed observer, that the frequent simultaneous occurrence of miliary tubercles and cheesy degenerations, and the experimentally proven development of the former from cheesy spots present in the body, is a clinical proof of the intimate relationship, if not identity, of the two processes, which is more weighty than microscopical appearances. Children in the first few years of life present this proof more frequently than at an advanced age. When I think of the numberless cases in which miliary tubercles of the lungs or pleura were found in the immediate vicinity of cheesy degenerations of the pulmonary parenchyma, or in which miliary tubercles of the pia mater were found adjacent to cheesy nodules in the brain, while many other organs presented both conditions in combination, I cannot believe in their essential difference, and consider myself justified in discussing them together.[1] The symptoms of pulmonary tuberculosis in children past the age of six or seven years are so similar to those of later life, that we shall mainly consider the disease during early childhood. The younger the children are, the more the local affection falls into the background in comparison to the general disturbance of nutrition which appears under the symptoms of atrophy (described on page 33.) I have frequently found tubercles and cheesy infiltrations, which had

[1] I do not wish to deny that the anatomical product called cheesy matter may also develop from the necrobiosis of other cellular structures (sarcoma, cancer, etc.). This does not affect the specific character of the much more frequent tubercular or cheesy degeneration.

been entirely latent during life, in the lungs of little children ; but large cavities, occupying the greater part of one of the lobes, were also found in some children, only a few months old, who, during life, presented merely progressive emaciation, weakness, and slight cough, so that the examination of the chest alone revealed the advanced destruction. This predominance of general nutritive disturbances over the local symptoms, is chiefly due to the fact that tuberculosis is usually much more widely distributed in early childhood than at a later period. Cheesy spots and miliary tubercles are generally found in a series of organs: the lymphatic glands, spleen, serous membranes, liver, kidneys, bones, etc., and there are cases in which scarcely an organ is found free from tubercular deposits. All these changes may run a more or less latent course. The chief symptom is the progressively increasing emaciation, often combined with otorrhœa, eczema of the scalp and other parts of the body, enlargement of the cervical, occipital, and inguinal glands, or multiple abscesses in the subcutaneous connective tissue. A careful examination of the thorax is therefore necessary to make a diagnosis.

This examination is much more difficult than in older persons. Occasionally it reveals nothing abnormal, with the exception of very rude breathing or catarrhal râles, and we would be inclined to make a diagnosis of chronic bronchial catarrh, did not the emaciation, hereditary predisposition, or glandular enlargements render us suspicious. But in many cases, extensive broncho-pneumonic foci are present which have become cheesy (page 148), and then give the usual signs of consolidation. While in later life the development of phthisical processes in the lungs usually occurs from above downward, and the restriction of the physical signs to the upper lobes and apices are valuable criteria in the diagnosis of the first stages, we not infrequently find in young children an irregular distribution of the tubercles and cheesy masses throughout the entire parenchyma, so that examination of the supraspinous and subclavicular fossæ may reveal very little, and the lower lobes present signs of consolidation; or, if these are absent, catarrhal signs may be present throughout. Irregular febrile movement and dyspeptic symptoms, anorexia, and especially diarrhœa, are often observed. As extensive pulmonary tuberculosis, and even cavities, may be present without cough or noticeable dyspnœa, the diarrhœa draws our attention away from the respiratory organs, and we are astonished, at the autopsy, to find the chief changes in the lungs.

O. F——, aged four months; bottle-fed. Multiple abscesses over entire body since the sixth week. Increasing emaciation for last nine weeks, little appetite, cough, short breath. Percussion less resonant on both sides above, anteriorly and posteriorly; indefinite breathing and bronchophony on right side above. Rales on both sides posteriorly. Pulse 150, temperature normal; fever said to have been present in the beginning. Father died of phthisis. Death in a week. Autopsy.—Great emaciation. Cervical and inguinal glands enlarged, partially cheesy. Partial synechia of pericardium with the heart and mediastinum, and miliary tubercles on its visceral layer. Left lung contains numerous gray nodules as large as a pea. Right lung firmly adherent throughout, upper lobe contains a cavity as large as a pigeon's egg, communicating with a larger one situated posteriorly. Large and small tubercular nodules scattered through the parenchyma. A large cheesy mass in the lower lobe. Tracheal and bronchial glands enlarged and cheesy, one of them containing a cavity. Miliary tuberculosis of liver and its capsule. Spleen adherent to neighboring parts, enlarged, and contains tubercles. A few small nodules beneath capsules of kidneys. Mesenteric glands partly cheesy. Few ulcers in ileum, with small gray nodules at their borders.

The latency of extensive tuberculosis is especially noticeable in small children who finally die of tubercular meningitis. Without any pro-

dromal symptoms of note, the meningitis suddenly develops, and, at the autopsy, the beginner in medicine is surprised to find miliary tubercles and cheesy masses in a number of organs which had presented no symptoms during life.

In older children, from the age of three years to the period of second dentition, tuberculosis not infrequently begins with dyspeptic symptoms. The children lose appetite, have a coated tongue, suffer frequently from diarrhœa, become emaciated, and complain of vague pains in the chest or abdomen, long before the cough attracts attention. They are moody, toward night show increased heat of body, thirst, and dry lips, and sleep uneasily, while a remission occurs in the morning, and only a slight elevation of temperature and an unusually rapid pulse indicate the latent disease. In such cases the symptoms are liable to be attributed to an obstinate dyspeptic condition, or to supposed helminthiasis. Careful examination of the chest cannot be too strongly urged under these circumstances. The suspicion of beginning tuberculosis gains ground, if there is an hereditary predisposition, and cough develops, or, if cheesy, scrofulous processes are noticeable; for example: suppuration of the bones and joints, spondylitis, enlargement of the glands, abscesses in the neck or other parts, chronic inflammations of the eye, eruptions on the scalp, otorrhœa. In a few months the local pulmonary symptoms—cough, frequency of respiration, etc.—become so prominent, that the examination forces itself upon us. Although, as a rule, the early examination furnishes no decisive results, catarrh at the apex can often be recognized, and this is sufficient to prepare the family for the probability of the threatening danger. At this age (three years and upward) a remittent fever or hectic develops sooner or later, but is not always present in young children.

This absence of febrile movement is never noticed in older children. Even without the thermometer, the exacerbation of fever is readily recognized by the increased warmth of the head and hands, the thirst, and increased feeling of sickness. The temperature rises to 39°, and slight perspiration, which is never as copious and regular as in the hectic fever of older patients, often ushers in the remission. I have repeatedly observed very irregular temperature-curves, the morning temperature being often higher than that at night. A child, aged two years, with miliary tubercles and extensive cheesy processes in both lower lobes, presented the following curve:

	Morning.	Evening.
August 22d	37.8°	39.5°
" 23d*	40.4°	37.6°
" 24th*	38.8°	37.9°
" 25th	37.8°	38.3°
" 26th	37.9°	38.4°
" 27th*	39.6°	38.4°
" 28th	37.0°	40.7°
" 29th*	39.8°	39.5°
" 30th	38.4°	40.0°
" 31st*	39.5°	38.5°, etc.

The days marked * show a higher morning temperature.

The absence of expectoration also renders the diagnosis difficult in children up to a certain age. Much the more noteworthy are those cases in which sputa are indeed expectorated, although this is done rather by hawking or by the aid of the mother, who removes them from the

mouth with her fingers. Among others, I observed profuse grayish yellow fœtid sputa, which occasionally contained elastic fibres, but no blood, in a boy, aged seven months, with extensive cheesy degeneration and formation of cavities in the left upper lobe. On the whole, hæmoptysis is extremely rare in children before the period of second dentition, although at least twelve to fifteen phthisical children, from the age of two and one-half to five years, have come under my notice who, during severe coughing spells, expectorated small quantities, occasionally even a teaspoonful, of blood, either pure, or mixed with mucus and pus. In one case, the compression or perforation of a branch of the pulmonary artery or vein by cheesy bronchial glands, and the simultaneous perforation of a bronchus, gave rise to an enormous hæmoptysis.

I will take this opportunity of entering a little more in detail into the predominant tendency of the tracheal and bronchial glands, especially the latter, to enlargement and cheesy degeneration. If tubercles or cheesy processes occur in any part of the body, we may be certain that these glands will also be affected. Among the countless autopsies upon tubercular children, I can remember very few exceptions to this rule, thus showing that the tendency of these glands, in children, to enlargement and cheesy degeneration, is greater than that of the lungs. I think the enormous frequency of the glandular enlargements is due to two circumstances, viz., to the peculiar predisposition on the part of many children to glandular enlargements on the whole, and to the great frequency of bronchial catarrh and whooping-cough. The irritation of the mucous membrane is conveyed through the lymphatics to the neighboring glands in the same manner as it is to the mesenteric glands in intestinal catarrh, typhoid fever, etc. The glandular affection is not infrequently the main disease in children, while the lungs only contain a few tubercles and infiltrations. The bifurcation of the trachea and the larger bronchi are found surrounded by isolated or conglomerated glands, occasionally in masses as large as a hen's egg, which are, in part simply hyperplastic, congested, and grayish red, but for the most part either partially or entirely tubercular, or converted into a homogeneous, whitish yellow mass. Upon section of the lung, small cheesy glands are often found at the bifurcation of the medium-sized bronchi. Some glands contain a central or more peripheral cavity filled with softened detritus, and, after its adhesion to the pulmonary pleura or a bronchus, may open into an adjacent pulmonary cavity or into a large bronchus. Large masses of glands at the root of the lung may compress the neighboring vessels, especially the pulmonary artery and vein and their branches, the superior vena cava and common jugular, or the pneumogastric nerve and its branches (recurrent laryngeal). The latter is sometimes so completely surrounded and flattened by the mass of glands, that it is hardly possible to trace its course through them. Adhesions of the glands to the œsophagus, the pulmonary artery, or one of its branches, have also been observed, and these parts have not only been displaced, but may be gradually thinned by the pressure, and finally perforated.

In the large majority of cases, this affection of the bronchial glands cannot be diagnosed during life. The symptomatology described by authors, reminds us somewhat of the study. It is said that pressure symptoms are readily produced, and indeed cases occur in which œdema of the face and dilatation of one or both jugular veins in the neck were produced by compression of the venous trunks, and hæmoptysis and hemorrhagic infarctions of the lungs were caused by pressure on the

pulmonary veins. In a girl, aged eighteen months, I observed compression of the right bronchus by tubercular glands, so that the entrance of air into the right lung was considerably diminished, and vesicular breathing on this side was extremely feeble. Compression of the pneumogastric and recurrent laryngeal nerves may also, as I have repeatedly found,[1] produce certain nervous symptoms, especially change in the voice (hoarseness), attacks of spasmodic cough, with inspirations like those of whooping-cough, or asthmatic attacks, with wheezing respiration and cyanosis of the face. But, according to my more recent experience, such cases are extremely rare. We often find large clusters of cheesy bronchial glands which presented no symptoms during life. Even the distention of the external jugulars and the œdema of the face, upon which stress has been laid, may occur as the result of stasis in the right heart, due to extensive infiltration of the lung, without any compression of the venous trunks within the thorax. I therefore regard the diagnosis of the glandular enlargements during life as extremely problematical, and attach very little value to the dulness on percussion said to be present in the interscapular space. I, at least, have never observed a glandular tumor of sufficient size to produce dulness in this region. Rilliet and Barthez state that large masses of glands in the posterior mediastinum act as good conductors of all sounds occurring in the lungs, and that loud bronchial breathing and râles may therefore be heard between the scapulæ, although the lung itself is not infiltrated or traversed by cavities. A mistake from such a cause has not hitherto come under my notice; moreover, it would soon be cleared up by percussion, since, if these sounds are really due to infiltration of the lungs or to cavities, distinct dulness on the dorsal surface will rarely be absent. I cannot, therefore, ascribe a definite, independent symptomatology to enlargement and cheesy degeneration of the bronchial glands. In the majority of cases it can only be suspected; exceptionally the diagnosis can be made with a certain amount of probability from the pressure symptoms.

Tuberculosis of children, until the period of second dentition, runs a more violent course than in later years. Very chronic cases are extremely rare; death usually occurs after the lapse of several months, at the most a year. Tubercular meningitis, broncho-pneumonia, or pleurisy, occur much more frequently than in adults, and terminate life more rapidly than would otherwise be the case. The pleura is affected almost as often as the pia mater, either in the form of miliary nodules scattered over the pulmonary and costal layers, or as large, cheesy spots on the free surface, or in the subserous connective tissue of the costal pleura. Small extra-pleural cavities may develop from softening of the latter, and either rupture into the pleural cavity, or, after adhesion of the pleura to the lung, may empty into pulmonary cavities or the bronchi. More or less extensive pleural adhesions are common, and in other cases subacute or chronic pleurisy develops with profuse purulent, often hemorrhagic exudation.

The fatal termination is often considerably hastened by the rapid development of acute miliary tuberculosis. This may occur during the course of chronic pulmonary tuberculosis, and constitute the fatal termination, or in apparently healthy children, who are not even suspected of a tubercular diathesis. In both cases the chief symptoms consist of violent fever, with irregular exacerbations, very frequent and superficial

[1] Romberg and Henoch, Klinische Ergebnisse, S. 165. Berlin, 1846.

respiration, sharp, saw-like respiratory murmur, replaced later by widely diffused fine râles, with which enlargement of the spleen and cerebral symptoms may afterward be associated, and thus lead to a mistaken diagnosis for typhoid fever or basilar meningitis:

W. K——, aged three, admitted March 15, 1860, with traces of scarlatina desquamation; pleuro-pneumonia of the right lower lobe. April 30th, almost all thoracic signs had subsided. August 6th, the child, who had been well since April, was again presented; for the past five days, headache, vomiting, and constipation. Pulse, 92; temperature slightly elevated; physical signs unchanged. August 8th, vomiting; pulse, 132. August 15th, the pupils sluggish, somnolence, abdomen somewhat retracted. An epileptiform attack yesterday, lasting three hours. In the next few days, increasing coma, frequent perspirations, left pupil dilated; respiration, 48; pulse, 128. August 21st, continuous spasms and contractures. Death during the following night.

The diagnosis of tubercular meningitis appeared to me undoubted. What did the autopsy show?

Pia mater congested, otherwise perfectly normal; considerable serum in the dilated ventricle. Bronchial glands enlarged and cheesy; right lung adherent, costal pleura thickened and studded with gray nodules. Cheesy degeneration of the anterior lower border of the right lung. Left lung contains numerous miliary tubercles. Fatty liver, tubercles in the spleen; a few tubercular ulcers in the intestines.

M. K——, one and one-half year old; stupor, dry crusted lips, contraction of both pupils, respiration irregular. Percussion normal, rude breathing, with large mucous rales. Abdomen distended and tender; constipation. Pulse very small, 144; temperature, 39.7°; toward night, 38.5°. Same condition on the next two days; on the day following, sudden rise of temperature to 40.8°, with respiration, 76; pulse, imperceptible, cyanosis, trismus, rigidity of the limbs, death. Autopsy.—Passive congestion and œdema of the pia mater. Congestion of the brain; ventricles contain a moderate amount of clear serum. Numerous miliary tubercles of pleura, lungs, spleen, and liver. Enlargement and cheesy degeneration of bronchial and mesenteric glands, and of intestinal follicles.

In both cases the pia mater presented no tubercles or exudation, although the characteristic symptoms of tubercular meningitis were present during life. Congestion of the pia mater in the first case, and œdema in the second, with accumulation of fluid in the ventricles, were alone observed. The same appearances were found in two cases of miliary tuberculosis which appeared under the guise of typhoid fever. In one case I observed a marked hemorrhagic diathesis in the course of acute miliary tuberculosis.

O. K——, aged four, admitted December 9, 1879. Taken sick on November 26th, with violent fever and hemorrhage from the mouth and nose, which have continued with short intermissions; no hemorrhagic diathesis previously. Pale, emaciated child, slight jaundice, superficial veins distended; œdema of scrotum. Temperature, 38.7°; respiration, 40, superficial; pulse, 156. Abdomen tympanitic; stools thin, black. Urine brownish red, acid, slight amount of albumen, no casts or intact blood-globules (hæmoglobinuria). Death on December 10th.

Autopsy.—A few submiliary nodules on the pericardium and in the heart-muscle below the aortic orifice. Numerous tubercles in the lungs, pleura, spleen, kidneys, liver. Enlargement and cheesy degeneration of the bronchial glands. No tubercles in the thoracic duct.

I must leave it undecided whether the hemorrhages in this case were really caused by the acute miliary tuberculosis; this must be determined by further observations.

At times acute miliary tuberculosis appears to occur in jumps, accompanied by more or less fever with apyrexial intervals. The following is an example of this rare form:

H. K——, aged six, admitted February 2, 1878. Slight cough, without abnormal physical signs. February 13th to 28th, diarrhœa; euphoria. March 6th, sudden

anorexia and fever (temperature, 40.9°; pulse, 134; respiration, 44). Rude breathing, percussion normal. Fever uninterrupted for four days; two chills. March 11th to May 8th, apyrexial interval; increased strength. May 8th, sudden fever lasting two days; temperature, 40.8; pulse, 145–160; respiration, 60. Catarrhal sounds in both lungs, and respiration 40–50 continuously. May 10th, temperature gradually subsided and remained normal until May 25th, the catarrh and rapid breathing continuing. May 25th, another attack of fever lasting five days. After some apyrexial days, a remittent fever (morning, 38.2°; evening, 39.2°–39.9°) began on June 1st and continued until death (July 5th); increasing weakness, bronchial catarrh, constant diarrhœa. Slight dulness on the right side below posteriorly; bronchial breath, extreme dyspnœa, death.

Autopsy.—Extensive pleuritic adhesions; enormous miliary tuberculosis of the pleura, lungs, peritoneum, spleen, liver, and kidneys. Cheesy infiltration at the base of the right lower lobe, cheesy degeneration of the bronchial and mesenteric glands.

With regard to treatment I have little to say. I have had no real success in any case which presented the signs of tuberculosis, or even of advanced pulmonary phthisis, while the previously described cases of "chronic pneumonia" are not very infrequently entirely cured. I refer you to the treatment recommended in the latter affection (page 158).

X. Gangrene of the Lungs.

Gangrene of the lungs in children differs clinically from that in adults in the fact that it is more difficult to diagnose on account of the frequent absence of sputa. The gangrenous odor of the breath is also of less value in children, because gangrenous processes may be present at the same time in the mouth and pharynx. In general it appears to be more frequent in children—not as a termination of fibrinous pneumonia, of which I have not seen a single example, but as the result of the entrance of septic material into the respiratory organs, whether through the circulation or by aspiration. It may be produced in the first-mentioned manner by gangrenous processes in the skin, which occur not infrequently in the miserable children of the poor, especially after infectious diseases. Pulmonary gangrene is also caused by direct aspiration of septic material in noma, and diphtheria of the pharynx. I have repeatedly observed putrid bronchitis in scarlatinal necrosis of the pharynx and in true diphtheria; and, twice in the latter affection, several gangrenous cavities as large as a pigeon's egg were found in the midst of a broncho-pneumonic infiltration. In phthisis pulmonum of children, gangrenous destruction of the walls of cavities are occasionally found, and extreme general weakness may also lead to gangrenous softening in broncho-pneumonic spots. In one boy, two and one-half years old, an attack of broncho-pneumonia developed, which, at the end of two weeks, was suddenly associated with extreme loss of power, pallor of the skin, and intensely fœtid odor of the breath. The autopsy showed a spot of gangrene, nearly as large as a hen's egg, in the right lower lobe, surrounded by infiltrated parenchyma. I also observed a similar condition in a case of typhoid fever, but I have reason to believe that both cases were due to the introduction of nutritive substances into the air-passages.

XI. Whooping-Cough.

Whooping-cough is undoubtedly an infectious process, but I consider it advisable to discuss it among diseases of the respiratory organs, because its symptoms chiefly belong to this sphere.

There are certain signs which may lead the physician to make a diagnosis of whooping-cough, even before he hears the child cough. To these belong the statement of the parents that the child suffers from a paroxysmal cough, occurring with especial frequency at night, which is associated with crowing or whistling inspirations and with a dark red color of the face, and terminates in strangling or the vomiting of mucus. The suspicion of the existence of whooping-cough is strengthened if the child's face, especially the lower lids, is somewhat swollen and the veins of the latter are dilated.

The disease is usually divided into three stages, which pass imperceptibly into one another. As a rule, the first stage (stadium catarrhale) is in nowise different from an ordinary tracheal or bronchial catarrh. More rarely the cough has a peculiar character, a more paroxysmal manner of occurrence, with a tendency to strangling at the close, from which we can at once suspect the development of whooping-cough. The catarrhal stage then lasts only a few days, and children in the first year appear to me to be especially predisposed to this peculiarity. As a rule, the duration of the first stage varies from ten to twelve days, and during this time the cough very gradually assumes a paroxysmal character. I do not deny that this stage may last five or six weeks, as some authors claim, but am inclined to believe that these were cases of ordinary catarrh, during the course of which the children became infected with whooping-cough. In children with a tendency to pseudo-croup I have seen the catarrhal stage begin with an attack of this kind. The first stage gradually passes into the second (stadium convulsivum, acme), which constitutes the height of the affection. The characteristic paroxysms, which give the name to the disease by their interrupted whooping inspirations, now occur with more or less frequency, with greatest severity and frequency during the night.

Frequently, though by no means constantly, the individual attack begins with a sort of aura, *i.e.*, with prodromata which foretell the approach of the paroxysm to the child and those around him. The child suddenly becomes restless, anxious, ceases to eat or play, clings to the mother or any firm object, as if it could better resist the approaching attack in this manner. In an infant, three weeks old, I noticed an anxious beating with the hands before each attack; in a boy, fourteen weeks old, a rapid discharge of urine and fæces; and, in some older children, prodromal vomiting. They suddenly ran with great haste to a corner of the room, discharged the contents of the stomach, and the attack then began. In a child, two years old, it began with restlessness and frequent, rapidly following sneezing, which also occurred at the close; while a nine-years-old girl presented an aura consisting of very rapid and dyspnœal breathing, with moaning expirations, which continued over an hour and then passed into the attack. Immediately after the latter and during the intervals the respiration was perfectly quiet, and only now and then a slight rattle was heard. The attack itself consists of rapidly following coughs, which are interrupted from time to time by a whooping inspiration. While the paroxysm lasts the child is bent over forward. The more rapidly the cough occurs, *i.e.*, the more rarely an inspiration is taken, the more the signs of suffocation appear, a dark, somewhat cyanotic redness of the face and neck, distention of the veins of the skin, cyanosis of the visible mucous membranes, especially of the tongue. Tears in the eyes, epistaxis, ecchymoses under the conjunctiva and the subcutaneous cellular tissue of the face, frequently accompany and follow the attack. The

action of the muscles concerned in respiration is considerably increased, especially of the abdominal muscles and the sterno-cleido-mastoids. During the whooping inspirations there is a temporary abatement of these symptoms, which immediately increase with the next series of coughs. The series of suffocating cough and forced inspirations is repeated from three to six times, and after the lapse of two or three minutes the paroxysm ends either without or more frequently with the discharge of bronchial mucus, either pure or tinged with blood and particles of food, many mothers aiding their expulsion by the introduction of the fingers into the mouth. After a very short pause a second milder attack is almost always observed, and this may be followed by a third, so that the entire paroxysm really consists of two or three rapidly following attacks. Complete rest now ensues. While many, especially small children, are perfectly exhausted, the older ones continue almost uninterruptedly in their occupation as if nothing had happened. The slight influence of the frequent nocturnal paroxysms is especially remarkable. The children start up, pass through the paroxysm, and then drop off to sleep immediately. If the chest is examined during an attack, no vesicular murmur can be heard even during the whooping inspirations, because these conceal everything else, and the air does not enter the alveoli in a normal manner.

The number of attacks occurring during twenty-four hours is extremely variable. While many children have, at the most, ten to twelve attacks daily during the entire course of the disease, in others the number rises to thirty to sixty, without any diminution in their intensity. You will understand that the danger of the disease increases with the number of the paroxysms, partly on account of the continually increasing exhaustion caused thereby, partly by the constantly recurring stasis of the venous system which accompanies every paroxysm and may acquire serious significance. Trousseau properly advises, therefore, to note accurately the number of attacks by lines on a slate, in order to obtain a measure of their increase and decrease, and thus of the danger of the disease. Although the attacks usually occur spontaneously, they are readily caused by emotions (crying), by the transition from the prone to the upright position, occasionally by distention of the stomach. I am usually able, by pressure upon the larynx or by percussion, to produce an attack for purposes of clinical demonstration. It is also noteworthy that when a number of such children are gathered in one room, a paroxysm in one very readily excites the same in the others.

The intervals between the paroxysms are entirely free from all morbid phenomena. No cough occurs, the respiration is quiet, and examination discloses either the normal respiratory murmur, or, at the most, a few catarrhal rhonchi. The disease is only noticeable on account of the previously mentioned slight œdematous swelling of the eyelids and the dilatation of the small veins around the eyes, which are accustomed to occur, after a length of time, in consequence of the constantly recurring stasis. To the same cause are due the hemorrhages from the nose, the bloody sputa, and the ecchymoses under the conjunctiva, which usually occur in patches, but sometimes attain considerable dimensions, so that I have found the cornea surrounded by an extravasation covering the entire sclera, the conjunctivæ palpebrærum suffused with blood, and both eyelids of a blackish blue color. But the pressure of the stasis occurring during the paroxysm may also manifest itself in other ways. In a child suffering from eczema of the ear, I saw a hemorrhage follow from the

diseased skin during every severe paroxysm. Hemorrhage from the external ear also occurs occasionally, and is explained by rupture of the membrana tympani, which, especially when otitis externa is present, is caused by the blow from the air, which is violently compressed during the cough and forced through the Eustachian tube into the cavity of the membrane. But these ruptures almost always heal without leaving any sequelæ, and cases of suppuration of the cavity of the membrane as the result of this process are exceptional. Barrier also observed a hemorrhage between the dura mater and arachnoid as the sequel of a paroxysm; and I have previously reported (page 101) a case of hemiplegia, which developed during a paroxysm of whooping-cough, and was undoubtedly due to a cerebral hemorrhage.[1]

In very many children who have suffered for some time from whooping-cough, a whitish gray erosion or deeper ulceration of the frænum linguæ is observed, and may lead to its partial or total destruction. The fact that this ulcer occurs exclusively in those children who already possess their incisor teeth shows that it is due to the constantly recurring friction suffered by the frænum during the paroxysms from the protrusion of the tongue over the lower middle incisors. I have also seen the ulcerations a few times on the lower surface of the tip of the tongue, or next to the frænulum, and even upon the dorsum of the tongue, and the injury is then due to the lower lateral or the upper incisors. But the ulcer is not always present in children who have teeth, and its occurrence depends chiefly on the fact whether the frænum is long and lax, or short and tense, in which latter event the protrusion of the tongue during the attack, and therefore the friction against the teeth, do not occur to a sufficient extent to remove the epithelium from the frænulum. Since my attention has been directed to this point, I occasionally meet with analogous cases of ulceration in children, who have either not coughed at all, or suffer from an ordinary bronchial catarrh, but have exceptionally sharp teeth.

The convulsive stage lasts, on the average, four weeks, and in the latter part of this time the nocturnal attacks diminish considerably in intensity and frequency. The suffocating character of the paroxysm gradually disappears, the whooping inspirations become shorter and weaker, the terminal strangling ceases, and a transition thus gradually occurs into the (third) stadium decrementi (stage of decrease), which may be again termed catarrhal. In this there is a loose cough, which reminds us of pertussis by certain characteristics, especially by a tendency to paroxysmal occurrence and unusual redness of the face. In about two or three weeks this cough also disappears and the child is then convalescent. The entire disease therefore has an average duration of eight or nine weeks, though every physician meets with cases which last three or four months. But the disease is then never continuous; in the midst of the stadium decrementi a relapse unexpectedly occurs, and the affection therefore lasts much longer. In many cases a chronic catarrh of the large bronchi remains after the disease has run its course, and every exacerbation caused by accidental cold or other cause (for example, measles) again produces paroxysms of cough, whose character reminds us of whooping-cough. I have seen such attacks suddenly develop a half-year or even a year after the beginning of the disease. We are not then justified in assuming a new disease; we rather have to deal with paroxysms of cough

[1] Vide a similar case of hemiplegia and aphasia, in Jahrb. f. Kinderheilk., Bd. X., S. 400. 1876.

which are produced by fresh irritation of the mucous membrane and assume the spastic character "from habit" (which plays such a prominent part in neuroses), but soon give place to an ordinary catarrh. I have never observed a true second infection with whooping-cough. Many simple, but protracted tracheal or bronchial catarrhs, are regarded by the parents as whooping-cough, especially when the cough has a very hoarse or slightly whistling character, as is peculiar to so many children.

The ordinary course not infrequently presents notable variations with regard to the paroxysm itself, as well as to the intervals.

With reference to the former I must call your attention to the unfavorable significance of those paroxysms which are characterized by protracted apnœa, in which cough, with very few or no inspirations, is alone observed, and therefore no whooping tone is heard. Such attacks are chiefly observed in little children during the first year of life. The cyanosis rapidly reaches its highest grade, the suffocation is threatening and may indeed prove fatal, especially when the disease is complicated with diffuse catarrh or broncho-pneumonia. Under these circumstances, partial spasms (deviation of the eyes, contracture of the fingers, toes, arms, etc.), or even general and fatal convulsions, may occur during the attack or immediately afterward, either in consequence of the protracted venous stasis in the brain, or the accumulation of carbonic acid in the blood, which must ensue in the absence of sufficient inspirations. But I must not fail to mention that whooping may be absent during the paroxysm in older as well as in younger children, without indicating a bad prognosis, so long as the attacks are short, and the cyanosis, like the symptoms of suffocation, does not exceed the usual severity or is even less marked. On account of the frequent recurrence of the above-mentioned severe attacks, which seriously interfere with the excretion of carbonic acid, cerebral symptoms may be developed which continue in the intervals and cause death under the guise of meningitis:

W. H——, aged one, admitted February 14, 1873, with pertussis. Severe attacks with prolonged apnœa and epileptiform spasms, at first only in the paroxysms, but later during the intervals. March 3d, double convergent strabismus. March 7th, repeated movements of mastication. After March 18th, somnolence, rigid retroversion of the head, contracture of the arms and legs; increasing coma, and death. March 23d. After March 9th there had been a remittent fever, due to bilateral broncho-pneumonia of the lower lobes; the paroxysms of pertussis were extremely frequent. Autopsy.—Congestion of the brain and pia mater; œdema of the latter in places; broncho-pneumonia; other organs normal.

The intervals may present still greater dangers than the paroxysms. The most frequent complication is diffuse bronchial catarrh and the broncho-pneumonia developing from it. If a child suffering from pertussis is not entirely well during the intervals, but breathes rapidly and superficially, has a moaning expiration and febrile movement, you may at once conclude that this complication is present, and an examination of the thorax will confirm the suspicion. Although broncho-pneumonia carries off a large number of children affected with whooping-cough, we should never abandon hope. I have seen complete recovery even in very small children and very bad cases, with extensive bilateral infiltration. I have only exceptionally observed fibrinous pneumonia and pleurisy, while emphysema of the apices and borders of the lungs, in combination with extensive broncho-pneumonic infiltration, is scarcely ever absent. I have never observed the occasionally described rupture of dilated pulmonary

alveoli and subsequent pneumothorax. In one child, suffering from phthisis, a tubercular ulcer of the right main bronchus ruptured during a paroxysm of pertussis, and was immediately followed by emphysema of the subcutaneous tissue of the neck and breast.

The broncho-pneumonia complicating whooping-cough has a tendency to become chronic and continue for months, the coughing-spells persisting with undiminished severity. In such cases I have often found, after death, dilatation and partial fatty degeneration of the right heart—changes which can be explained by the persistent venous stasis and the obstacles in the pulmonary parenchyma, which must be overcome by the heart. Œdema of the dorsal surfaces of the hands and feet, and sudden death from collapse or syncope, have repeatedly come under my notice in these conditions. The weakness of the heart also probably explains the enormous frequency of the pulse, which has struck me in the course of many cases of broncho-pneumonia complicating pertussis, even with comparatively low temperatures. This is not always a fatal symptom, though it may suddenly lead to exhaustion of the heart.

Chronic bronchial catarrh and pulmonary phthisis, due to cheesy degeneration of broncho-pneumonic products, are not infrequent sequelæ of pertussis. Acute miliary tuberculosis or tubercular meningitis may also develop years afterward as a result of the hyperplasia and cheesy degeneration of the bronchial glands, which are induced by the catarrh of the mucous membrane in protracted cases of whooping-cough. Finally, I have seen in a number of cases the development of a deformity of the chest (chicken-breast) very similar to that occurring in rickets, even in previously well-developed, non-rachitic children. This is explained, as I believe, by the deficient inspirations, and the consequent incomplete dilatation of the lungs during the paroxysms, but especially by the complication with chronic broncho-pneumonia.

Concerning the etiological relations of whooping-cough, we know really nothing. The disease may occur in earliest infancy, and I have seen it in children aged respectively three and six weeks. The greatest frequency occurs between the second and fifth years; but older children are also often affected, adults very rarely.

There is no doubt that pertussis is contagious, and readily conveyed from one child to another. It is much more inexplicable that in my wards, in which children suffering from whooping-cough are not isolated, an example of its propagation is observed only exceptionally. I possess no certain data with regard to the period of incubation, but have repeatedly observed that, if a child conveys the disease from school to her family, at least ten to twelve days elapse before the cough begins in the brothers or sisters. It is supposed that the contagium is carried in the inspired air to the respiratory mucous membrane, and, as a matter of course, bacteria have been described as the cause of whooping-cough. For the present, however, I regard the spores found by Letzerich [1] in the sputa of patients, and used in vaccinating the trachea of rabbits, as problematical from an etiological standpoint as the assumption of Tschamer [2] that whooping-cough is produced by the introduction into the air-passages of a fungus occurring on the rind of oranges and apples.

The action of the unknown contagium on the bronchial mucous membrane is not confined to the production of an ordinary catarrh of the

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[1] Jahrb. f. Kinderkrankh., III., S. 354, 1870; S. 436, 1873.
[2] Ibid., X., S. 174, 1876.

trachea and its bifurcation. Every one who has heard a paroxysm of whooping-cough must acknowledge that, in addition to the catarrh—whose existence I will not deny[1]—another factor, a nervous one, must also be taken into consideration, manifesting itself, on the one hand, by the spasmodic expirations, and, on the other, by the apnœa and the whooping tone of the spasm of the glottis. I would also remind you of the symptoms described as the aura of the attack, and the almost constant vomiting. I will acknowledge that the strangling and vomiting of mucus at the close of a paroxysm may be regarded as a mechanical act—as the result of the violent contractions of the abdominal muscles during the coughing-spells—as they are not infrequently observed, when the stomach is full, in other violent paroxysms of coughing which are unconnected with pertussis. But we should remember that many children vomit after very mild paroxysms of whooping-cough, and that cases occur, furthermore, in which the emesis is the most prominent symptom of the attack, and may even excite grave fears on account of its persistence. Some children, indeed, vomit constantly during the intervals between the paroxysms, and gradually sink into a serious condition of weakness, although no cause can be found in the digestive organs. Vomiting of this kind must be regarded as nervous in its origin. The relapses referred to above (page 172), which are certainly not due to fresh action of the contagium, also speak in favor of this view. In what manner this specific poison exercises its influence upon the central nervous system is as yet an unsolved problem. All changes which are found on post-mortem examination must be regarded merely as due to complications of the disease.

Whooping-cough often occurs in more or less wide-spread epidemics, which do not depend, in general, on the seasons of the year. A certain relationship to measles cannot be denied. Not only is the combination or succession of both epidemics often observed, but one individual, suffering from one of these diseases, appears to me to have a special predisposition to the other. The combination of both these infectious diseases in one individual I regard as dangerous, because extensive broncho-pneumonia almost always develops in such cases. The situation is still more serious if a child suffering from pertussis and broncho-pneumonia becomes affected with measles. In such cases I have seen cyanosis develop before the appearance of the eruption, which became at once bluish, and death occurred in a few days, with the symptoms of carbonic acid poisoning. The not infrequent combination of whooping-cough and diphtheria is also a grave matter, though it should not lead us to despair.

You will see from this description that whooping-cough, which is a prognostically favorable disease *per se*, may seriously endanger life either on account of the tender age at which it occasionally occurs, or from certain complications (bronchitis, broncho-pneumonia, convulsions), and that, even after its complete recovery, residua may be left in the lungs or bronchial glands, which may form the starting-point, at a later period, of miliary tuberculosis.

The enormous number of remedies employed in this disease proves its incurability. I have come to rely upon one drug alone, viz., morphine, which can at least moderate the violent paroxysms, especially the nocturnal ones, and diminish their frequency, although it does not influence the course of the disease on the whole (P. 10). But you should not neglect to

[1] Rehn (Wien. med. Wschr., 52 u. 53, 1866) demonstrated it with the laryngoscope in two adults.

warn the mothers that, as soon as unusual drowsiness develops, the remedy should be at once discontinued. When carefully administered, I have never seen any bad results follow, even when one to two teaspoonfuls of the mixture were given daily for weeks, and I therefore prefer it to all other narcotics, especially to the dangerous atropine. But I would recommend morphine in severe cases only, in which at least twenty paroxysms occur in the twenty-four hours. In milder cases you may try inhalations of carbolic acid, which may also be combined with the use of morphine. These inhalations, which are justified theoretically by the undoubted existence of an infectious substance in the respiratory passages, have had a good repute in recent times. My own experience does not permit any decided opinion, as I have sometimes had strikingly favorable, then doubtful results, and at times none whatever; I have never observed any bad effects. We can employ either a one or two per centum solution of carbolic acid by means of a spray apparatus; if this is difficult of performance, the air of the room may be impregnated with its vapor and a sponge dipped in the solution hung at the head of the bed, and a similar one held several times a day in front of the child's nose, so that its vapor can be inhaled for several minutes. I do not possess sufficient experience with regard to other inhalations, viz.: chloroform, benzine, salicylate of soda, oil of turpentine, tannin, quinine, etc.; from the unsatisfactory results of such trials as I have made, I have no desire to continue their use. This is also true with regard to bromide of potassium and chloral hydrate.

In fine weather the fresh air should be enjoyed as much as possible; but in rough, windy weather, or when severe bronchial catarrh is present, this should be strictly prohibited. Many physicians believe that a change of residence is advisable, but the results of my experience do not confirm this opinion. I have hardly ever seen any good results from sending the patients to a watering-place, whether at the sea-shore or in the mountains. The sole effect of such a course was the infection of healthy children who came in contact with the patient. In exceptional instances I have seen beginning pertussis, with characteristic paroxysms, disappear in Reichenhall within two weeks, but I do not think such isolated cases prove the beneficial effects of a change of locality. Many so-called abortive cases of pertussis occur and recover with surprising rapidity without any treatment; Trousseau mentions a case which lasted only three days.

PART V.

DISEASES OF THE CIRCULATORY ORGANS.

I. Congenital Disease of the Heart.

Pathological changes in the heart in children are not much rarer than in adults, but disease of the large vessels is extremely rare. Hodgson's observation of ossification of the temporal artery in a child aged fifteen months, and Andral's case of calcareous plates in the aorta in a child aged five years, are exceptional instances. Congenital stenoses of the aorta, which are usually situated in the region of the ductus Botalli or the beginning of the descending aorta, are diagnosed much more often in youth than in childhood, although a part of them seems to be connected with the involution of the ductus arteriosus which extends to the aorta. I may take this opportunity of mentioning that the closure of this channel, which is about as thick as a branch of the pulmonary artery in the new-born, is the result of obliterating endarteritis from new formation of connective tissue, thickening of the walls, and narrowing of the lumen. The process is noticeable as early as the ninth day after birth, leads to a stricture in the middle of the duct on the fourteenth day, then progresses in both directions, and is usually completed by the end of the third week, while obliteration of the foramen ovale is accomplished within a few days after birth. All causes producing deficient filling of the left ventricle in the first period of life, such as extensive atelectasis, fœtal pneumonia, or stenosis of the pulmonary artery, must delay the closure of the ductus arteriosus, because the blood of the pulmonary artery endeavors to enter the imperfectly filled aorta through this channel, and the delayed obliteration of the duct in such cases may compensate for months the evil effects of an otherwise unavoidable stasis in the right heart and general venous system. This is also true of the patency of the foramen ovale, which, apart from the above-mentioned causes, may also be produced by local anomalies in its own structure or that of its valve.

You are probably aware that the patency of these fœtal channels, the ductus Botalli and foramen ovale, was formerly regarded as the chief cause of congenital cardiac cyanosis. As this was supposed to be due to a mixture of arterial and venous blood, the cause of the anomalous color was sought in the patency of the fœtal channels and in the abnormal communication of both arteries or ventricles through a defect in the septum. But we now know that cyanosis occurs when the mixture of both kinds of blood does not take place, and that, on the other hand, the ab-

normal communication referred to above may be present in children, and even in adults, who manifested no trace of cyanosis during life.

At birth, or shortly afterward, a bluish violet color of the cheeks, tip of the nose, hands and feet, and visible mucous membranes (tongue, mouth, nares, conjunctiva) becomes noticeable, and increases considerably during crying, nursing, vigorous movements, and after the action of cold air, but may be so slight in the intervals that it is scarcely noticeable by the laity. After a considerable time—occasionally within a few months —a club-shaped (like a drumstick) swelling develops in the last phalanges, and occasionally a claw-shaped deformity of the nails. I was also struck, in a few cases, by the spongy scorbutic condition of the dark violet gums, which readily bled spontaneously and on contact. The hands and feet are not infrequently somewhat œdematous, the eyes are prominent, the temperature of the extreme parts of the body lowered. These symptoms are often associated with general weakness, slowness of movements, tendency to somnolence, diminished growth, and, finally, the entire complex of symptoms peculiar to various heart diseases, viz.: attacks of suffocation—especially after exertion—syncope, enlargement of the liver and spleen, etc. Physical examination usually reveals distinct enlargement of the heart, especially the right side, systolic or diastolic valvular murmurs, or, in rare cases, no abnormality.

These symptoms permit a diagnosis of congenital heart disease, but the special diagnosis of the deformity is usually impossible. I would refer those interested in this subject to Rauchfuss'[1] elaborate article, though the writer admits that we can make only a probable diagnosis. We have to deal, in these cases, with openings through which both auricles or ventricles communicate with one another, or with larger defects, which constitute in their highest development complete absence of the septum, or with stenosis and atresia of the conus of the pulmonary artery, the vessel itself, the aorta, the openings into the auricles; finally, also, with transposition of the large vessels, the pulmonary artery taking its origin from the left ventricle, the aorta from the right. The difficulties of diagnosis are still further increased by the fact that two or more defects of structure are present in the majority of cases; but the symptom to which special attention is paid by the physician, viz., congenital cyanosis, may be entirely absent. Children of this kind have often come under my notice during the first few months, or, at least, the first year of life, who either suffered merely from attacks of dyspnœa or presented no cardiac symptoms, and were supposed to be suffering from a pulmonary or intestinal affection. The following case will serve as an illustration:

A child, aged thirty days. Hereditary syphilis. March 19th to 21st, pneumonia of upper lobe; no cyanosis; nothing abnormal discovered in the heart. Autopsy showed, in addition to pneumonia, a marked deformity of the heart. Both ventricles communicated with one another through a large opening. The septum was almost entirely absent. The tricuspid valve was absent and one of the mitral valves inserted into the right side of the heart.

If the children remain alive for a few years, more or less marked symptoms usually develop; generally, also, cyanosis, either as the result of accidental diseases of the respiratory organs or of endocarditis, which develops as often from the abnormal openings, or the congenitally diseased valves and orifices, as it does in adults with old valvular disease (endo-

[1] Gerhardt: Handb. für Kinderkrankh., Bd. IV. 1878.

carditis recurrens). Under these circumstances, the previously latent abnormalities become manifest, and examination now shows that we have to deal with an anomaly of long standing. The most marked effects are produced by stenosis and atresia of the pulmonary artery or its conus, which also constitute the most frequent cause of congenital cyanosis. In many cases it cannot be determined whether the retraction and partial atresia of this artery are the results of a fœtal endocarditis and myocarditis, or of a primary failure of development, with which an inflammatory process was associated at a later period. In consequence of these stenoses, dilatation of the cavities of the right heart always occurs with stasis in the entire venous system, of which cyanosis constitutes the expression. The cardiac dulness extends beyond the right border of the sternum, the impulse of the heart can be seen and felt over a large area, and is often accompanied by a perceptible thrill; and a systolic murmur is heard over the heart, loudest over the pulmonary valves, occasionally heard over the entire chest and back, but, as a rule, not extending into the carotids. The coexistence of other cardiac malformations may produce variations in the symptoms, which render diagnosis more difficult, but cases are not lacking in which the heart-sounds were perfectly normal.

We can predict nothing with certainty regarding the course of congenital heart disease. The greater the obstacles to the venous circulation, and the less they are compensated by other defects (opening in the septum, patency of the ductus Botalli), the shorter will be the child's life. When there is sufficient compensation, life may be prolonged to the period of youth, or even longer. I have repeatedly seen febrile diseases—for example, the acute exanthemata—pursue a favorable course in such children. The fatal termination finally occurs, as in all diseases of the heart, either suddenly from syncope, or under the influence of a disease of the respiratory organs, which in itself is not dangerous, a diffuse catarrh or pneumonia, more rarely with the symptoms of gradually increasing venous stasis and dropsy. Cheesy pneumonia may also act as the cause of death, and Rokitansky's statement concerning the immunity of cyanotic patients from pulmonary tuberculosis is by no means in accordance with fact.[1]

As previously mentioned, valvular lesions and their sequences may be accidentally found in children who have presented no subjective symptoms, or, at the most, a scarcely noticed palpitation or shortness of breath in running or going upstairs, and the most careful clinical history fails to give any clue with regard to the origin of the affection. Despite the absence of cyanosis in such cases, we must assume the existence of a congenital defect. I will take this opportunity of calling to your attention that small, spherically projecting hemorrhages may occur on the valves of the heart, especially on the free border of the mitral, in very young children—even in the new-born. Parrot[2] has often observed these hæmatomata of the valves in the new-born, upon the venous orifices of both sides of the heart, in the form of very small (sometimes as large as a cherry-pit), black or violet, round or conical prominences. These hæmatomata, which must be attributed to a rupture of intravalvular blood-vessels, are always situated under the most superficial layer of the endocardium, develop very soon after birth—perhaps even before delivery—and undergo retrograde changes in the first few months, their covering retracting more and more, while the epithelium and connective tissue proliferate around them.

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[1] Rauchfuss, l. c., S. 92.

[2] Arch. de physiol., No. 4 u. 5. 1874.

They also appear to give rise to small, broad, or pedunculated, hard nodules, covered with epithelium, which are not infrequently situated in the same places. It is possible that an anomalous retrogression of these hæmatomata may give rise to retraction of the edges of the valves, and thus to stenosis of the orifice or insufficiency of the leaflets, the origin of which can no longer be demonstrated in older children. The valvular lesion is then not strictly congenital, but has developed during the first months of life.

The treatment of such cases must be restricted to the recommendation of the most quiet mode of life possible. In other respects the treatment does not differ from that of other organic diseases of the heart.

II. Inflammatory Affections of the Heart.

In a series of cases acute rheumatism is the starting-point of organic disease of the heart. The former affection occurs less frequently and in a milder form in children than in adults, but the complication with endocarditis or pericarditis is rarely absent. Even in very mild attacks of rheumatism, which present scarcely any fever, you should not fail to examine the heart, and you will not infrequently find pericardial or endocardial murmurs, for which you were unprepared on account of the apparently trifling character of the affection. The clinical history, therefore, often shows, in cases of well-marked valvular disease, that one or more attacks of acute rheumatism, especially in the joints, occurred months or years previously. The complete similarity of these valvular diseases and their results to those in adult life render it unnecessary to enter in detail into the physical signs. With regard to the subjective symptoms, I will merely call attention to the fact that, although cases of complete compensation and consequent latency of valvular affections are not rare in adults, they appear to me to occur much more frequently in children. Not even vigorous movements during play or going upstairs produce any appreciable disturbance, and in many cases the disease is first detected by the mother, who notices the violent action of the heart while undressing and washing the child. Only after the compensation begins to be disturbed do the well-known cardiac symptoms develop which sooner or later terminate fatally. There are also no anatomical differences from similar conditions in adult life. In both we find dilatation and hypertrophy of the ventricles, brownish red induration of the lungs, hemorrhagic infarctions, passive congestion of the liver and kidneys, enlargement and induration of the spleen, œdema and dropsical effusions into the cavities and pulmonary alveoli.

In many cases the valvular affection, developing as the result of rheumatism, becomes manifest only after a number of months or years; but some instances of a much more rapid course also occur.

A. M——, aged seven, formerly healthy. Middle of December, 1878, had acute articular rheumatism, lasting a few days. During Christmas week she was suddenly taken sick with palpitation of the heart, diminished secretion of urine, cough, frequent pain in the region of the heart. Admitted to the hospital, February 12, 1879. Examination showed general anæmia, catarrh of both lower lobes, cough, and dyspnœa. Cardiac dulness extends to the right border of the sternum upward to the third rib, on the left to the line of the nipple. Indistinct apex-beat outside the line of the nipple in the fifth intercostal space; loud systolic murmur with first sound of the heart. Urine scanty, considerable albumen. February 21st, sudden rise of temperature to

40.0°, and rapid depression. February 22d, distinct pulsus bigeminus, pericardial friction-sound at the left border of the sternum. February 25th, death in collapse.

Autopsy.—Heart hypertrophied three-fold, both ventricles markedly dilated and thickened. Aortic and mitral valves thickened at the free edges, somewhat retracted, and covered with reddish gray vegetations. Recent partial synechia of pericardium. Diffuse bronchitis, œdema, and brownish red induration of the lungs.

In a girl, aged seven, who in October, 1874, had passed through a mild attack of acute rheumatism, associated with endocarditis, I found, in March, 1875, not only the signs of mitral insufficiency, but also marked hypertrophy and dilatation of both ventricles.

Thus, eccentric hypertrophy of the ventricles, as the result of valvular disease, developed to a marked degree within a few months after the first appearance of a mild acute articular rheumatism. The course was so rapid that compensation did not occur at all, and was hastened in the first case by the diffuse catarrh and the final occurrence of fresh pericarditis and endocarditis. This "endocarditis recurrens" was found in all old cases of valvular disease, whether they were congenital or acquired. Occasionally this process, which, as a rule, is first recognized at the autopsy, can be determined clinically.

In September, 1872, I treated a girl five years old, who was suffering from acute articular rheumatism, with endocarditis, after whose recovery a systolic mitral murmur persisted, although the patient felt well. In January, 1875, a fresh endocarditis developed in the markedly dilated and hypertrophied heart, manifested itself by fever, increase in loudness of murmur, great dyspnœa, and terminated fatally.

On the other hand, experience teaches that rheumatic endocarditis is more readily recovered from, and its sequelæ retrograde more rapidly, in children than in adults.

C. F——, aged three. October, 1871, was attacked with rheumatism, high fever, rapid breathing; at the end of the first week there was loud systolic blowing murmur at the apex of the heart. All symptoms disappeared in two weeks, with the exception of the murmur, which grew weaker in the spring of 1872, and disappeared entirely in November.

P. H——, aged six. February, 1868, pains in the abdomen, dyspepsia, moderate fever. February 16th, severe chill. After a day and a half, acute articular rheumatism, moderate fever, bronchial catarrh, heart normal. After temporary improvement, exacerbation on February 29th, loud diastolic murmur, especially in the mammary region, diminishing above. March 22d, everything normal, except anæmia and diastolic murmur. In the spring of 1869 this had entirely disappeared, and the boy remained healthy afterward.

Cases occur occasionally in which the endocarditis is the first indication of rheumatism, and the articular affection is noticeable only at a later period.

P. F——, aged five, has felt sick for past twelve days; irregular fever, rapid breathing. Five days ago a systolic murmur was discovered at the mitral. May 13, 1875, sudden development of acute articular rheumatism; temperature, 39°-40°. May 26th and 27th, fresh joints affected, increased dyspnœa, dulness over the sternum and vicinity, heart-sounds feebler, indicating pericarditis. June 3d, death; autopsy not allowed.

In this case the endocarditis developed at least five days before the articular rheumatism. So long as the endocarditis does not affect the valves or orifices, all abnormal sounds may be absent; indeed, some cases of ulcerative endocarditis in adults prove that even ulceration of the valves may be present without valvular murmurs. In the wife of a colleague, who for two weeks presented no other symptom but general

malaise and remittent fever, with very rapid pulse, no organic change could be detected despite careful examination. After the lapse of two weeks, I discovered a continually increasing systolic murmur, and made a diagnosis of endocarditis, which was confirmed on autopsy. Cases of this kind, which may readily be mistaken for typhoid fever, also occur in children. I recently saw in a three-year-old boy, who had suffered a few months previously from mild rheumatism, an endocarditis manifest itself for three or four days, merely by high fever (39.5°–40.5°). Then endocardial, and soon afterward friction sounds, developed. The case of P. F—— on the preceding page is exactly similar. The complication, observed in both cases, with rheumatic pericarditis, may render the diagnosis more difficult by the intervention of its auscultatory signs:

E. P——, aged eleven. I was first consulted December 19, 1879. Febrile angina for the past week. A few days afterward, acute articular rheumatism, successfully treated with salicylic acid. Since day before yesterday, severe pains in left breast and increased fever. A loud systolic blowing murmur at the apex, growing weaker above; over the lower part of the sternum a friction-murmur accompanying both sounds of the heart, extending over the epigastrium and as far as the mamma: percussion unchanged. At the end of a week the fever and pericardial friction had disappeared, but the endocardial murmur was unchanged. The boy complained of stitch-pains and often interrupted speech to draw breath. January 3, 1878, euphoria, with the exception of rheumatic pains in the left shoulder. The mitral murmur still continues.

In this case the rheumatic endocarditis was complicated, after the lapse of a few days, by pericarditis.

I have previously referred to the relations of chorea to rheumatic affections of the heart (page 79), and then maintained that the chorea and endocarditis were due to one and the same cause, viz., rheumatism, and that the former cannot be regarded as dependent on the cardiac affection alone. I also appeal to the fact that the primary rheumatic affection may be very slight and—especially in children who suffer only from vague muscular or articular pains—may be entirely overlooked, and that only its results, viz., chorea and endocarditis, are recognized by the physician, who is then inclined to attribute the neurosis entirely to the latter affection.

Infectious diseases, especially scarlatina, give rise to the development of pericarditis or endocarditis, which may be followed by permanent valvular disease, much more rarely than rheumatism. Although every temporary systolic blowing murmur in scarlatina cannot be regarded as a sign of endocarditis, there can be no doubt of the existence of the latter if the murmur persists for some time in the same manner, and associated with febrile movement. This complication is observed in scarlatina itself, and also in the subsequent nephritis.

W. K——, aged six, admitted February 1, 1876, with scarlatina. The fever fell to 38.5° at night toward the end of the second week. February 12th, a short systolic murmur was heard, which grew more distinct every day, especially loud at the apex; pulse 136, somewhat irregular. During the next few days a short creaking was also heard to the left of the sternum at the level of the third rib, at the height of inspiration, but often isochronous with systole; this disappeared in a few days. April 22d, the murmur at the apex of the heart was scarcely audible, and by April 25th had entirely disappeared.

That this was really a case of scarlatinal endocarditis is proven by the continued febrile movement, the rapid, somewhat irregular pulse, and the systolic murmur, which disappeared completely only after the lapse of

two months. To the long continuance and gradual diminution of the murmur I attach especial weight, which cannot be claimed, as I have already remarked, by temporary murmurs. I have repeatedly observed, especially in the course of scarlatinous nephritis, systolic murmurs in the mitral region, which could be heard for only twenty-four to thirty-six hours, were occasionally associated with irregularity of the pulse, and then disappeared without a trace; while in other cases a separation of the first sound, or a galloping rhythm of the sounds, was heard for a few days, or even weeks, and then disappeared without leaving any residua. In a case of scarlatinal synovitis terminating in suppuration of the acromio-clavicular joint, as shown upon autopsy, a systolic blowing murmur had accompanied the febrile onset of the disease, but became inaudible in a few days, and the valves were found normal. But it cannot be denied that scarlatinal joint affections show a tendency to combination with inflammatory processes in the endocardium—more rarely the pericardium. That chorea may develop under such conditions has been previously remarked (page 80) in reporting a case, though it does not prove that the former depends upon the endocarditis, since chorea is occasionally observed after scarlatina, uncomplicated with synovitis or any cardiac affection.

Diseased conditions of the neighboring parts—especially pleurisy of the left, less that of the right side, pneumonia, caries of the ribs—may become important in the development of pericarditis, on account of their spread to the pericardium. Either a sero-fibrinous or purulent exudation may occur into the pericardial cavity, or, if the affection is chronic, readily leads to more or less extensive adhesions of the heart to the pericardium. Purulent pericarditis is especially observed in connection with empyema in very young children, and the diagnosis may be rendered very difficult, or even impossible, by the small quantity of pus on the one hand, and the extensive dulness dependent on the pleuritic exudation on the other (page 160).

R. L——, aged eight months, admitted March 10, 1877. Rachitis, frequent respirations, painful cough. Flatness and bronchial breathing over the left thorax; heart-sounds normal. Bronchial breathing disappeared in a few days; no respiratory murmur could now be heard; the dulness extended 1 ctm. beyond the left border of the sternum, but no displacement of the heart could be distinctly proven. Temperature almost always subnormal; increasing collapse. March 21st, death. Autopsy.—Left pleural cavity entirely filled with purulent exudation; compression of the left lung; fibrino-purulent pericarditis (pericardium contains a couple of tablespoonfuls of pure pus; both layers covered with recent fibrin).

Endocarditis may also develop under these circumstances. I have previously mentioned a case in which, in addition to an old encapsulated pleurisy of the right side, there was marked synechia of the pericardium, with marked thickening and insufficiency of the mitral valve, and stenosis of the ostium venosum. In two other children extensive broncho-pneumonia of the left lung became complicated with an endocardial systolic murmur, which continued in one case until death, and in the other persisted after the recovery of the pulmonary affection.

Finally, tuberculosis must be mentioned as a not very rare cause of acute chronic pericarditis in childhood. The development of miliary or submiliary nodules in the pericardium, especially on the visceral layer, is not of frequent occurrence, even in general tuberculosis; but pericarditis, with sero-fibrinous or hemorrhagic exudation, or, finally, more or less ex-

tensive synechia of the pericardial cavity, may develop without these local products.

P. M——, aged eight, admitted May 20, 1878. Felt sick for past week; respiration, 36; temperature, 38.6°; pulse, 136. A loud friction-murmur accompanying both sounds was heard in the region of the heart, and 2 ctm. beyond the right border of the sternum; percussion normal; apex-beat not felt distinctly. May 24th, friction-sound had disappeared, and cardiac dulness extended upward to the third rib, and beyond the right border of the sternum; pulse very small. The fever gradually subsided, the pulse grew stronger, and on June 6th both sounds of the heart were found to be feeble, but perfectly clear. June 13th, distinct friction with both sounds; extent of pericardial dulness diminished. July 6th, a faint friction-sound still heard over the sternum, otherwise everything normal, and on August 7th the boy was discharged as well. Readmitted in October, with considerable ascites, presenting the symptoms of chronic tubercular peritonitis. Nothing abnormal could be discovered in the heart during his entire stay in the hospital; died May 5, 1879. Autopsy.—Left costal pleura thickly strewn with tubercles, the pulmonary pleura to a less extent. Pericardial cavity entirely obliterated by old adhesions; the muscular tissue of the anterior wall of the right ventricle was found in a condition of fibrous degeneration in various places; the pericardium and heart entirely free from tubercles; valves normal; œdema and thickening of the anterior mediastinum; tubercular peritonitis and meningitis.

Three factors in this case appear to me especially noteworthy: 1st, the complete latency of the total synechia of the pericardium, which must be regarded as the result of the pericarditis recovered from a few months previously; 2d, the onset of the disease with pericarditis; there is no doubt that this pericarditis was due to tuberculosis of the left pleura, which first gave rise to inflammatory irritation of the anterior mediastinum; it is especially noteworthy that no tubercles developed in the mediastinum or pericardium, but only inflammatory products were found; 3d, the implication of the myocardium, not in the form of peripheral fatty degeneration, as so often occurs in pericarditis, but of interstitial myocarditis, which is so seldom observed in children. I have detected an affection of the myocardium in only one other case:

Pericardial cavity dilated; contains half a tablespoonful of hemorrhagic fluid. Both ventricles firm; epicardium shows slight fibrous thickening throughout. At the apex of the left ventricle is found, in addition to marked atrophy, an aneurismal dilatation as large as a hazel-nut, the walls of which are scarcely ½ ctm. in thickness. Endocardium of the left auricle is very much thickened; retraction and warty condition of mitral and aortic valves.

The symptoms of valvular disease and hypertrophy of the heart could alone be determined during life. The cardiac aneurism was evidently due to partial chronic myocarditis, which developed in connection with the endocarditis and the inflammation of the epicardium, and gradually converted the corresponding part of the muscular tissue into a fibrous cicatrix, which gradually grew thinner under the pressure of the blood. I have never observed in childhood the extensive chronic fatty degeneration of the heart-muscle so frequent in adults, with or without sclerosis of the coronary arteries. I have, however, quite often observed a partial fatty degeneration, especially in the right ventricle: 1st, in children with obstinate pertussis and chronic pneumonia (page 148), in which it develops on account of the obstacles which must be overcome by the heart in the pulmonary circulation, and which may give rise to fatal syncope; this category also includes the partial fatty degeneration which finally occurs in hypertrophy of the heart from valvular lesions; 2d, in children who have long suffered from exhausting diseases, suppuration,

etc.; 3d, most frequently after acute infectious diseases, especially after scarlatina and diphtheria.

I have observed simple hypertrophy and dilatation of the heart only in exceptional instances during childhood, a couple of times in chronic nephritis, and in two children who probably had congenital enlargement of the heart. In all other cases the hypertrophy and dilatation were due to an affection of the valves.

The treatment of diseases of the heart in childhood is similar to that in adults. Although the chronic forms (valvular disease followed by hypertrophy) are generally better tolerated by children, and may last until the period of puberty or later, before serious disturbance of compensation develops, cases are not lacking in which distressing symptoms render medical interference necessary. I was struck by the frequency of anæmia in these children, by the proper treatment of which with ferruginous preparations (P. 12) it was often possible to moderate at least part of the symptoms (palpitation, weakness), and improve the general condition, although the cardiac disease remained unaffected. The use of iron was never attended with injurious effects in these cases. The treatment of acute (inflammatory) affections of the heart must naturally be antiphlogistic; local bleeding, ice-bags, calomel, digitalis, and blisters are here in order.

PART VI.

DISEASES OF THE DIGESTIVE ORGANS.

I. Inflammatory Affections of the Buccal Mucous Membrane.

The simplest form of these diseases, which occur very often in childhood (stomatitis simplex s. erythematosa), is characterized by a general or partial bright or dark red color of the mucous membrane, especially the gums, which appears thickened and sensitive on pressure. An unusual quantity of saliva flows from the half-opened, often somewhat swollen and reddened lips. In attempting to take the breast, nurslings let go the nipple on account of pain, and cry, and the mother or nurse often notices considerable increase in the warmth of the buccal cavity while nursing, even before other symptoms are observed. The increased secretion of saliva gives the entire buccal cavity an unusually slippery, shining appearance, and a whitish gray coating, composed chiefly of desquamated epithelium, covers the dorsum of the tongue entirely or partially. The region of the lower jaw is not infrequently œdematous, and, partly on this account, partly from enlargement of the lymphatic glands in this locality, appears fuller than usual. Slight febrile movement, restlessness, and insomnia may also be present.

This form of stomatitis is observed most frequently during the eruption of the teeth (page 64). In older children it occurs not infrequently in acute infectious diseases, especially measles and scarlet fever, and may then readily pass into a more severe form, attended with fibrinous exudation. Simple cases require no special treatment, as it usually disappears spontaneously after the eruption of the teeth, or the subsidence of the exanthem.

The second form (stomatitis aphthosa) is far more characteristic and perhaps even more frequent than the first. Most of these children vary from nine months to the middle of the third year (period of dentition); the affection occurs much more rarely before, and especially after, this period. In addition to the symptoms of stomatitis simplex, we observe at the edges, tip and dorsum of the tongue, more rarely upon its lower surface and near the frenulum, grayish yellow or grayish white, round, occasionally notched patches, surrounded by a narrow red border, and varying from the size of a pin's head to that of a pea, or even larger; they may either be sparse or present in much larger numbers, and occasionally coalesce into grayish white patches or various-shaped figures. In a few cases I saw grayish white, firm plaques, as large as a five-cent

piece, projecting beyond the level of the mucous membrane, the edges being loosely applied. Small patches are often seen on the mucous membrane of the cheeks, palate, and even the tonsils, and also upon the reddened, thickened lips, which readily bleed when touched, and are therefore often covered with dark brown or blackish crusts of dried blood. They are not infrequently associated with groups of herpes vesicles at the border of the lips, and whitish gray excoriations at the angles of the mouth, and in these severe cases the lower part of the face may be appreciably swollen, on account of the usually perceptible enlargement of the submaxillary lymphatic glands, and perhaps by slight œdema of the surrounding connective tissue. The tongue, except at the edges, is almost always covered with a thick, grayish yellow coating, the secretion of saliva increased to such an extent that it is continually flowing from the half-opened mouth; the gums are dark red and swollen, and covered here and there with delicate whitish layers of epithelium. Increased temperature, especially at night, irritability and restlessness, pain which interferes with eating and drinking, or render them almost impossible, are always present.

In some cases there is a bad smell from the mouth, and the congested rim of the gums surrounding the teeth is then found converted into a grayish yellow, crumbling detritus, which can be readily removed by the spatula.

Despite its great frequency, the pathogeny of this affection is still obscure. I have never been able to detect the formation of the plaques from vesicles, as was formerly held. We should not be deceived by the fact that some of the usually perfectly flat patches occasionally project more or less; this is not due to a vesicular detachment of the epidermis, but to the greater thickness of the exudation. The process is due to the deposit of a fibrinous exudation in the most superficial layers of the mucous membrane. We are never able to detach a patch with the forceps, as it is always firmly connected with the mucous membrane, and, as Robin has shown, by fibres and an amorphous mass. A certain superficial similarity to "diphtheritic" products arises in this manner. The disease always remains a local inflammation, attended with fibrinous exudation, which, so far as my experience goes, has always terminated favorably. Under proper treatment it recovers within eight to ten days; the secretion of saliva diminishes, the fœtor oris, which may have been present, disappears, and the patches become smaller from the periphery toward the centre. Thick, whitish gray patches become more yellowish, translucent, lose their red border, and disappear without leaving a trace, except perhaps a dark redness of the affected spot. A superficial necrosis of the infiltrated part appears to occur, and the shallow erosion thus produced is at once covered with new epithelium.

The process becomes more serious when fibrinous stomatitis is associated with molecular necrosis of the rim of the gums mentioned above. I have, indeed, seen such cases heal as rapidly as the ordinary ones; but it should be remembered that this is a combination with the beginnings of a more serious form (stomatitis ulcerosa, or stomacace).

It was formerly maintained that the disease is contagious, and I have occasionally seen cases of this kind. Thus, a boy became affected with stomatitis, after he had repeatedly taken a bite of an apple which was partaken of by another child suffering from the disease. But such cases are exceptional, and do not justify the assumption of their parasitic origin.

In the treatment, I would especially recommend chlorate of potash, which acts as a specific—and most rapidly, in my experience, in those cases

associated with fœtor oris and affection of the edge of the gums (P. 26). The slight pain due to the contact of the remedy with the diseased mucous membrane should not be taken into consideration in view of the surprisingly rapid disappearance of the fœtor and salivation. Many cases recover in five or six days under this treatment; much more rarely we meet with obstinate cases, which resist the employment of chlorate of potash, or even grow worse under its use. Under such circumstances, good results were obtained, as a rule, by the local application two or three times daily of sulphate of zinc (1.0 to 20.0 aq. destil.) or sulphate of copper (0.5 to 20).

In acute exanthemata, especially scarlatina, I have repeatedly observed stomatitis fibrinosa very similar to that just described, but more severe and wide-spread. Stomatitis may also occur in combination with simple catarrhal angina of childhood. In a boy, aged five years, suffering from angina, I saw the velum palati above the tonsils covered with numerous yellow, round patches, the central depression in which indicated their relation to the follicles.

Stomatitis ulcerosa, or stomacace, is rarer than the form just described. While the fibrinous patches are either entirely absent or play a subordinate part, the affection of the gums absorbs the attention of the physician. It is dark or bluish red, swollen, bleeds readily, and becomes converted more and more into a grayish yellow mass, starting from the border around the teeth, by which the crowns of the teeth are laid bare and finally loosened. Upon pressure, a purulent fluid flows from between the detached gum and the teeth, a fœtid smell is exhaled from the mouth, and the surrounding soft parts, the cheeks and submaxillary connective tissue, are often œdematous. This swelling and the increasing pallor of the child often disfigure the face in an alarming manner and lead to the suspicion of developing noma. But the slow progress of the process and the persistent softness of the swelling prove that this fear is not justified. The impossibility of taking food in sufficient quantity, on account of the pain, becomes much more serious, because the disease, as a rule, runs a protracted course, which may extend over many weeks. Under these circumstances the process not infrequently extends to the periosteum of the jaw, descends into the alveoli, and may lead to loss of the teeth, and, finally, partial necrosis of the jaw.

A child, aged eighteen months, presented October, 1878. Fœtor oris, considerable swelling of the right cheek and submaxillary region, weakness, pallor, fever, inability to eat. The gums, especially on the right side below, red, swollen, detached from the teeth, which were surrounded by pus. The left lower alveolar border swollen and painful, and contained two loose teeth, which were at once extracted. Frequent injections of the buccal cavity with one-half per cent. solution of permanganate of potash, internally, decoct. chinæ (5 : 100), with chlorate of potash, 3.0. January 29, 1879.—Considerable improvement, but necrosis of edge of right lower jaw, from which a few sequestra were removed. Swelling considerably diminished; suppuration subsiding. Further course unknown.

When the disease lasts very long, the protracted suppuration and deficient nutrition may finally lead to death, though such a termination has not come under my notice. With two exceptions, all my cases were of a milder character and yielded to the persistent use of decoct. chinæ with chlorate of potash (P. 27) and disinfecting injections of permanganate of potash, carbolic or salicylic acid. Pure country air should be urgently recommended. The extraction of loose teeth or sequestra, as soon as this can be done without too much violence, hastens recovery.

In children of five to eight years of age, *i.e.*, during second den-

tition, ulcerative stomatitis is also not infrequent. The gum is red and swollen, its edge in a condition of molecular necrosis, while the chief changes occur in the mucous membrane of the tongue, cheeks, and lips. Large, uneven ulcers form, covered with a grayish yellow pulp, with wall-like swollen edges of mucous membrane, which bleed readily on contact, and have an extremely fœtid smell. If the ulcer is situated at the edge of the tongue, a similar one is almost always found at the corresponding part of the inner side of the cheek, so that we cannot doubt its propagation by contact from one surface to the other. I have observed this also in the adjacent surfaces of the lower lip and gum. The swelling of the surrounding mucous membrane makes the ulcer appear deeper than it really is. I am unable to furnish any definite information concerning the etiology of these affections, which are associated with necrosis of the mucous membrane, or even of the bones. The children observed by me presented no trace of any distinct dyscrasia, but had been perfectly healthy prior to the disease. A few showed the symptoms of "cachexia pauperum." There is no doubt that dentition plays some part in the process, as congestions, hemorrhages, and small abscesses of the gums occur very often during this period, and may readily be intensified into higher grades of inflammation. The treatment coincides with that recommended in aphthous stomatitis.

II. Gangrene of the Mouth.—Noma.

This terrible disease, also known as cancrum oris, is peculiar to childhood; but it is fortunately so rare, that it only comes at long intervals to the notice of even the busiest physician.

At the first examination, as a rule, the physician observes considerable swelling of half the face, especially the cheek and half the upper lip, occasionally also the lower lip and chin, perhaps extending to the lower lid, so that the eye is half closed and the features disfigured. The swelling is pale, usually shows a fatty gloss, from increased secretion of sebum, and is very little, or not at all, sensitive to contact, but very tense, so that the pressure of the finger leaves a depression. On careful palpation, a more or less extensive hardness, which is gradually lost in the surrounding parts, is felt, deep in at the most prominent part of the swelling.

Although ulcerative stomatitis may also be associated with a similar swelling of the soft parts, we should always, under such conditions, think of the possibility of noma, especially if the patient is cachectic or has recently passed through a severe illness. Examination of the buccal cavity then shows not only a fœtid, but a decidedly gangrenous odor, which increases constantly, and may infect the atmosphere around the child. But this is not always so. In two cases the odor was found so faint that I had to apply my nose near the lips of the child in order to detect it. Opening the mouth and depression of the tongue with the spatula is rendered very difficult, on account of the swelling and tension of the cheek. If we are able to gain a view of the buccal cavity, we will find upon the mucous membrane of the swollen cheek, more rarely of the upper or lower lip, an extensive loss of substance, at least as large as a "mark" piece, of a brownish, greenish, or dirty gray color, in the neighborhood of which the mucous membrane is œdematous and projects above the edges of the ulcer. This gangrenous destruction of the mucous membrane makes such rapid progress, that a large part of it is converted,

within a few days, into a grayish brown shred-like, stinking pulp, and the gums and mucous membrane of the palate soon suffer the same fate. Crumbling shreds of the gangrenous detritus can be removed with the spatula or forceps, but the masses usually adhere more firmly to their base than we would be inclined to believe at first sight. Fœtid saliva usually flows from the mouth, the submaxillary lymphatic glands are swollen, and the swelling may extend more or less down the neck, on account of the œdematous infiltration of the surrounding connective tissue.

We should imagine that such an extensive gangrenous affection would seriously affect the entire organism, but this does not by any means occur to the extent supposed. Only in those cases in which the noma develops immediately after an exhausting local or general disease, does the loss of vitality make itself evident from the beginning. Otherwise the general condition may remain surprisingly good for days. The children are found sitting up in bed, perhaps playing, eating with a good appetite, although parts of the gangrenous detritus are swallowed with the food. But we often notice obstinate diarrhœas, which are not explained by the autopsy, and are probably due to gangrenous decomposition of the intestinal contents, produced by contact with the ingested septic matter. Despite the apparent euphoria, fever is almost always present, the temperature fluctuating in some of my cases from 39.4°–40°, with a corresponding frequency of the pulse and respiration.

Fatal collapse may suddenly occur in this stage, before the destructive process has perforated the entire thickness of the soft parts. More frequently life is prolonged, not only until the gums and periosteum of the jaw have become gangrenous, but also after the corresponding side of the tongue and palate have necrosed, and the perforation of the cheek or lip is complete. The most projecting part of the swelling first assumes a rosy hue, becomes as hard as a stone, and then blackish, until finally a black, dry gangrenous scurf is formed, occupying the entire thickness of the soft parts, and this, spreading rapidly, may finally extend upward to the eye and downward to the neck. A pale redness, surrounding the gangrene, denotes the demarcation, and a part of the gangrenous scab is then cast off, either spontaneously or with the aid of the physician, and a correspondingly large, sharply defined opening permits a free view of the buccal cavity. A large part of the cheek, lips, and eyelids may be lost in this manner, though the child manifests scarcely any signs of pain, and is occasionally found sitting up and asking for nourishment. Increasing loss of power, constant diarrhœa, or extensive broncho-pneumonia, which are partly due to the aspiration of gangrenous fluids into the air-passages, finally put an end to the process after it has lasted two to three weeks.

A few cases have a favorable termination, even in the last stage, after perforation of the soft parts has occurred. With the cessation of the gangrenous destruction, the child's vitality increases, and cicatrization begins with a string-like retraction of the remaining soft parts. In these rare cases, cicatricial deformities always remain, such as ectropia of the lower lid, adhesion of the jaw to the cheek, narrowing of the buccal cavity, which may be partly removed by plastic operations. But we must not forget that the term "noma" is occasionally misused, the name being applied to cases of stomacace described above, and not to true "cancrum oris," which can only be accepted when the soft parts of the cheek or lips have undergone more or less gangrenous destruction, and deformities consequently remain.

Autopsies show little more than what is seen during life, but the extension of the gangrenous destruction posteriorly toward the pharynx is shown better in the dead body. Analogous gangrenous changes are occasionally found in the internal organs, especially putrid bronchitis, broncho-pneumonia, gangrene of the lungs, and catarrh of the intestinal mucous membrane; the latter is, however, absent in many cases which die from severe diarrhœa.

Among the etiological factors of noma, the "cachexia pauperum," due to poor nourishment, uncleanliness, unhealthy damp dwellings, plays a prominent part. I have seen noma develop under these conditions, either spontaneously, or more frequently after bronchitis, pneumonia, or dysentery, especially when the weakness due to these diseases has been increased by debilitating treatment. Whether the protracted administration of mercury, especially calomel, is capable of producing noma, is still a mooted question. If we consider that this agent produces stomatitis and salivation far more rarely in children than in adults, we would at once be inclined to agree with those who deny this action of calomel, and, indeed, my own experience favors this view. It goes as a matter of course, however, that calomel should not be administered for weeks at a time in weak, miserable children. On the other hand, infectious diseases —scarlatina, measles, typhoid fever—may at times be followed as readily by noma as by gangrenous affections of other parts, especially the skin and vulva. The following case, which was only observed in the later stages, appears to show that ulcerative stomatitis, under unfavorable circumstances, may pass into noma:

A. P——, aged four years; cerebral symptoms while convalescing from severe bronchitis Treatment consisted in applications of ice to the head and inunction with tartar emetic ointment, which led to a gangrenous sore over the occiput. Unclean ulcers formed on the gums and tongue, with salivation and fœtor oris; and six days later a grayish green ulcer on the mucous membrane of left cheek, which soon assumed the characteristic appearance of noma. Death from collapse after complete perforation.

There is no doubt that noma, in almost all cases, first develops as a gangrenous ulcer of the mucous membrane, and then spreads through the entire thickness of the soft parts. But it cannot be denied that the gangrene, in certain cases, may appear from the start in the external parts of the cheek without implication of the mucous membrane. I have only observed this condition in one case, in which the noma developed from a phlegmon of the cheek.

The treatment of noma must be the most strengthening possible (decoct. chinæ, wine, bouillon, yolk of egg). If swallowing is hindered by the extension of the process, we should try nutritive enemata (yolk of egg, milk, bouillon, peptone). In order to limit the gangrene, caustics are recommended, especially fuming nitric acid, liq. ferri sesquichlorati, concentrated carbolic acid, or a thick paste formed of camphor. I have seen no good results from any of these agents. If the soft parts are already converted into a gangrenous scurf, or this has been thrown off, the hot iron alone can do any good, and indeed I would advise you to use it from the beginning as soon as you are convinced that noma has developed in the mucous membrane. Pacquelin's thermo-cautery is the instrument best adapted to this purpose. After the gangrene has developed fully, the entire necrotic portion can be removed by a blade-shaped cautery-iron, and care must be taken to remove all the gangrenous tissue by cutting within the normal tissue. But a favorable termination is not as-

sured, even if the gangrene subsides after the operation, as the children may die from sudden collapse or from some complication (diarrhœa, broncho-pneumonia). Injections with solutions of carbolic acid, salicylic acid, or thymol, and dressing the wound with charpie dipped in vin. camphorat., should not be omitted.

III. Inflammatory Affections of the Pharynx.

Children over four years of age suffer from the milder forms of catarrhal angina perhaps even more frequently than adults, but much more rarely under this age. The symptoms in general do not vary from those of later life.

In the majority of cases the local symptom, pain in swallowing, is not marked, or may be entirely absent, while the fever begins with such severity as to lead us to suspect a serious acute disease. The angina begins, as a rule, with bad humor and a feeling of fatigue; the children refuse nourishment, and may vomit once or twice. A chill, followed by heat, or the latter alone, now opens the scene; the temperature rapidly rises to 39° or 40°, or more. On a few occasions I have seen epileptiform convulsions occur after the initial rise of temperature (page 68). As a rule, it falls considerably on the next day, perhaps to the normal, and fever may then remain absent; or a slight rise occurs at night, the local symptoms becoming more marked. Many irritable children have a very frequent pulse (136–144), which becomes considerably slower, however, within twenty-four to thirty-six hours. I do not believe, with some, that the high initial fever suffices to stamp angina catarrhalis as an infectious disease.

Certain anginas present some similarity to diphtheria. On the second day of the disease, round yellowish white patches the size of a pin's head, or larger, very often appear upon the reddened and more or less swollen tonsils; occasionally they are isolated, but are often dense, and then partially confluent, so that the tonsil appears covered with an irregular whitish yellow mass, which looks suspicious (angina follicularis). But usually the character of these patches leaves no doubt of their benign nature; they are composed of a purulent secretion, which, passing out from between the folds in the mucous membrane of the tonsils, is only loosely adherent, and may be readily detached. The yellowish color of the patch is also different from the gray or white color of a diphtheritic exudation. However, cases occur not infrequently in which judgment must be suspended for twenty-four to thirty-six hours, especially when the patches are large and confluent, as they are sometimes quite firmly adherent; the high initial fever and the enlargement of the submaxillary glands then appear to favor the diagnosis of diphtheria. Within twenty-four to thirty-six hours, however, simple catarrhal angina is either at a standstill or is subsiding, while diphtheria progressively increases in severity.

I attach no importance to the fever and enlargement of the lymphatic glands, as they are common to both affections. Microscopical examination will not decide the question, as bacteria are found in both affections. White, tegument-like shreds are also occasionally found on the tonsils in catarrhal angina, consisting of amorphous fibrin and epithelium, thus making the diagnosis still more doubtful. In one child, suffering from follicular angina, I also saw the tip of the uvula covered with a whitish coating of this kind. Under such circumstances it is

preferable to isolate the child and observe the course of the disease for twenty-four to thirty-six hours before forming an opinion. Many cases of severe catarrhal angina are undoubtedly regarded as diphtheria by superficial observers, and this explains the fact that many physicians cure almost every case of diphtheria.

It cannot be denied, however, that some of these doubtful cases, which recover within six to eight days, may be mild cases of true diphtheria. It has appeared to me especially suspicious when several brothers and sisters have been affected in the same manner simultaneously or successively, or if the patches, which were originally confined to the tonsils, have also developed on the edge of the velum palati or uvula. If we find that these children are attacked repeatedly, perhaps even yearly, by this doubtful angina, it is better to make a confession of error than to plume ourselves on the repeated cure of diphtheria in one and the same child. Finally, I may mention that children occasionally present extensive, somewhat deep, grayish yellow, uneven ulcers on one tonsil (rarely on both), which, to judge from the yellow points of pus visible here and there, have been produced by the coalescence of small abscesses. They are unconnected with diphtheria or syphilis (as is sometimes supposed), and almost always recover spontaneously in eight to ten days.

In every catarrhal angina I would advise that the children be kept in bed for a couple of days, and a mild purgative (inf. sennæ comp, elect. e senna, P. 7, 28) given if the passages are scanty. I do not expect any benefit from chlorate of potash, and gargles can only be used in older children. When there is a tendency to these anginæ, we can endeavor to prevent their recurrence by brushing the tonsils daily with a solution of nitrate of silver (1 : 20).

The tonsils may become hypertrophic from the frequent recurrence of these inflammations, but I have observed this hypertrophy much more often in children who have very rarely or never suffered from angina; the presence of scrofula could only be demonstrated in a part of the cases. The development of this tonsillar hypertrophy is so slow, that morbid phenomena appear only after the end of the first few years of life, and the affection is rarely observed at the age of one or two years. Three symptoms are observed, viz.: unusual snoring during sleep, or noisy breathing during the day, a nasal twang to the voice, and difficulty in hearing, due to pressure of the enlarged tonsils upon the opening of the Eustachian tube. Examination shows marked projection of both tonsils, more rarely of one, so that the entrance to the pharynx is more or less narrowed, and may be almost entirely closed in the higher grades. The tonsils may then be in apposition, and the uvula and velum be pushed upward and backward, though deglutition is not interfered with. The patient may suddenly start from sleep on account of the interference with respiration, and true attacks of pavor nocturnus are not wanting under these circumstances. I will take this opportunity of reminding you that neither in angina nor in very considerable tonsillar hypertrophy can the enlarged tonsils be felt from the outside; this is rendered impossible (Luschka) by the interposition of numerous structures (musc. constrictor pharyngis superior, bucco-pharyngeal fascia, platysma, parotid gland, integument).

If marked tonsillar hypertrophy develops at an early age, the obstruction to the free entrance of air into the lungs may lead to a deformity of the chest like that of rickets, viz., chicken-breast (Dupuytren, Shaw, and others). On account of the insufficient supply of air in the lungs, the

external atmospheric pressure is greater than the internal, and forces the yielding costal cartilages inward, thus flattening the thorax laterally, and causing projection of the sternum. Narrowing of the nostrils, and diminished growth of the upper jaw, with greater concavity of the palate, the teeth being pressed closely against one another, have been observed in these children. Exceptionally the obstruction to respiration is so great that tracheotomy must be performed.

The only method of treatment consists of excision, or at least partial resection of the enlarged tonsils. All other measures, including the parenchymatous injection of iodide of potassium, or a solution of iodine in glycerine, are much inferior, as their results are uncertain and their performance more difficult than the rapid surgical operation.

IV. Contagious Parotitis.

(Angina Parotidea.—Mumps).

Various morbid processes occur in children associated with swelling in front of the ear and below the angle of the jaw, which are readily mistaken for one another by the inexperienced physician. Especially during first dentition, but often much later, you will find in this region diffuse but usually unilateral swellings, which are at first doughy and normal in color, but gradually grow harder and red, finally fluctuate, and discharge pus either spontaneously or after incision. I would not mention these connective-tissue abscesses, which are probably due to adenitis of the lymphatic glands, were it not for the fact that they are often mistaken in the beginning for angina parotidea. This is also true of the œdematous swellings developing after stomatitis or alveolar periostitis.

In the majority of cases of mumps, the children are presented to you after the disease is fully developed, and you then find on one or both sides a diffuse, quite soft, but often harder swelling in front of the ear, which extends downward over the angle of the jaw, and terminates behind it with an often distinctly perceptible rounded tip, the lower end of the parotid gland. When both sides are severely affected, the swelling may coalesce under the jaw, while otherwise there is a prominence merely in front of the ear and behind the lower jaw. Enlarged lymphatic glands can often be felt under the inferior maxilla. The skin over the swelling usually presents a normal color, more rarely a pale red, and is very slightly or not at all sensitive on pressure. In a few cases I have found the veins of the forehead and around the eye markedly dilated, probably from compression of the facial vein by the swollen parotid gland. I have never noticed any changes in the saliva, but mild angina tonsillaris is often present. In many cases, euphoria is not disturbed, though the thermometer usually shows a rise of temperature to 38°–38.5°, perhaps only at the beginning or on the first day of the disease. I have only observed exceptional instances of high fever (39°–40°), headache and vomiting in children, but repeatedly in adults.

The disease usually lasts five to seven days. The swelling increases for a few days, remains stationary for about forty-eight hours, and then gradually disappears. When both sides were successively attacked, I have seen its course prolonged to ten or fourteen days. I have never observed the metastasis to the testicle occasionally seen in adults, nor the termination in suppuration or permanent hardness of the gland.

The fact that the disease, almost without exception, attacks an individual only once; its undeniable contagiousness, its stage of incubation, lasting about two weeks, and its not infrequent epidemic spread, render the infectious nature of parotitis certain. We must suppose that the unknown infectious matter passes through Steno's duct into the parotid gland, and there produces an irritative condition, with parenchymatous swelling of the gland, perhaps with partial retention of saliva. I have never detected any enlargement of the spleen and many lymphatic glands, adduced by some authors as proof of its infectious character. Whether the sublingual and submaxillary glands may also receive the contagium is still undecided. Some cases observed by Penzoldt[1] and Soltmann[2] favor this view, and I myself treated an adult in whom, after gastric and febrile prodromata, both submaxillary glands became enlarged, and finally metastasis occurred to the right testicle, although the parotid gland was unaffected.

It is best to keep the child in bed on the first day, and in the room during the next few days. The swelling should be covered with cotton. Isolation is unnecessary.

I will here report a case whose nature is not entirely clear:

A girl, aged nine, admitted May, 1878, with bilateral otorrhœa, otherwise healthy. October 1st, complained of pain in the throat. On the following day the floor of the mouth was swollen and tender, the mucous membrane pale. Opening of the mouth and movements of the tongue painful; abundant flow of saliva. Temperature: morning, 38.4°; evening, 39.8°. October 3d, swelling increased and visible externally below the jaw; the swelling was doughy and œdematous. Tongue pressed upward and immovable. Upper and lower jaws separated 1 ctm., the latter immovable. As the symptoms did not diminish, five leeches were applied below the jaw, and chlorate of potash (3.0 : 120.0) given internally on October 4th. Improvement and diminution of swelling on the same night. Fall of temperature on the following day. Pain and swelling diminished, lower jaw movable, no swelling of the tongue—whitish coating on its surface, showing ordinary fungi and fatty epithelium under the microscope. Rapid recovery, so that the entire process lasted about a week. At the end of three weeks a relapse occurred, which lasted six to seven days.

I regard this case as due to an irritative condition of the sublingual glands and surrounding connective tissue, but I am unable to determine its cause. The flow of saliva and the occurrence of a relapse negative an affection analogous to contagious parotitis. The effects of mercury could also be excluded. We might think of small concretions in the sublingual glands, although these are very rare in children,[3] but they could not be detected. The most suitable name for this extremely rare disease is subglossitis, applied by Holthouse to a similar case.[4]

V. Diseases of the Œsophagus.

Among diseases of the œsophagus in childhood, sprue and diphtheria, on account of their clinical latency, possess only an anatomical interest. Stenosis of the œsophagus alone possesses any practical significance. In extremely rare cases this is congenital, and ingested milk at once escapes

[1] Deutsche med. Wochenschr., IV., October 19, 1878.

[2] Jahrb. f. Kinderheilk., VII., S. 409.

[3] Closmadeux (Revue Méd., Août, 1855), among 112 cases of salivary calculi, found 101 of the sublingual gland, but none under twenty years of age.

[4] Hirsch u. Virchow: Jahresber. f. 1871, II., S. 505.

from the mouth and nose from the first day. It is almost as rarely due to compression of the œsophagus by adjacent organs and tumors, as from carcinoma of its walls. But stenoses from cauterization of the œsophagus are much more frequent in children than in adults, the chief rôle being played by the drinking of lye used in washing, and often mistaken by the children for weiss beer. I have frequently seen this accident in children from two to eight years old, and once in a boy of fifteen months. If a few days have elapsed since the accident, distinct traces of caustic action are found in the mouth and pharynx, with marked difficulty in swallowing, and vomiting of mucus, occasionally mixed with blood. In the youngest child mentioned above, complete aphonia was present for a week (from swelling of the entrance to the larynx) and afterward gave place to severe hoarseness. As older children immediately eject the fluid, its effects may be confined to the pharynx, or only a small portion reaches the œsophagus, thus explaining the comparatively rare occurrence of inflammatory gastric symptoms. In a few recent cases, however, I observed symptoms of gastritis, continuous severe pains in the region of the stomach, vomiting of food and drink and of large quantities of mucus, constipation, high fever, tenderness of the epigastrium, and, in one case, a discharge of black blood from the anus without hæmatemesis. In most cases we do not see the children until four to six weeks after the accident, when the symptoms of stenosis of the œsophagus, caused by cicatricial retraction of ulcerative losses of substance, are distinctly developed. The chief symptom is emesis, or rather regurgitation of the ingested nourishment and tough masses of mucus immediately after swallowing. Fluids can be swallowed in the beginning, but not at a later period. The children suffer from starvation within a few months; increasing emaciation, sallow color of the peaked face, and extreme weakness, are unavoidable results. In a boy, ten years old, who was brought to my wards in this miserable condition, the temperature was subnormal, the skin and mucous membranes cyanotic, and there were only forty-four small, scarcely perceptible pulsations per minute, probably in consequence of a nutritive disturbance of the heart, which took part in the general atrophy. Death is inevitable if better nutrition cannot be secured.

In order to determine the situation and degree of the stricture, we introduce an œsophageal sound; or, if this, as is usually the case, does not pass through, an elastic catheter or a whalebone sound with a steel, olive-shaped tip. Two strictures are occasionally present, one of which can be passed more easily than the other. Older, intelligent children may be able to define accurately the site of the obstruction. The most certain method of radical cure is gradual dilatation by means of the daily introduction of bougies or sounds provided with a metallic or ivory olive-shaped tip. This manipulation requires great patience and caution in order to prevent perforation, and we must always use sounds or bougies small enough to pass through. If the instrument is introduced daily for five or six minutes—at a later period somewhat longer—we are often able to pass a larger one in a few days, whereupon the dysphagia diminishes and small quantities of food may enter the stomach. Strength and nutrition gradually improve, and I have often been astonished, under these circumstances, to see how rapidly the cheeks fill out and the complexion improves. But as the cicatricial connective tissue, whose retraction has caused the stenosis, retains this tendency to retract, even after artificial dilatation, success will only be temporary if dilatation is not continued daily for many weeks and even months. This can rarely be done in dis-

pensary practice, but I have observed several hospital cases which were entirely cured. But this treatment will not always prove successful, since, as a few autopsies have taught me, the stricture may not only be dense and rigid, but also of considerable length, and, after it has lasted for some time, dilatation of the œsophagus may occur above it, and into which the instruments enter. Gastrotomy, which has been occasionally performed successfully in recent times, remains the last resort for such incurable cases. During treatment, the nutrition should be aided by enemata of the yolk of egg, bouillon, wine, or peptone.

VI. Diseases of the Stomach.

Like the œsophagus, the stomach is much more rarely affected by serious diseases in childhood than in adult life. Cases of acute inflammation rarely ever occur, apart from those just mentioned, which were due to the ingestion of a caustic fluid, while chronic catarrh and the round ulcer are very infrequent, and carcinoma is one of the rarest exceptions. The symptoms of the round ulcer do not differ from those observed in adults. As tubercular ulcers, the small ulcerations of the new-born (page 31), diphtheritic processes in the gastric mucous membrane, hemorrhagic suffusions, etc., only present an anatomical interest, I will confine myself to the consideration of a few diseases which, though not occurring exclusively among children, present some peculiarities at this period. Among these,

Gastric Dyspepsia

occupies the first rank, on account of its frequency. I will only discuss the dyspepsia of older children, which is so common on account of their tendency to overload the stomach. Whenever this is done, spontaneous recovery may occur after the production of vomiting, or of copious, foul-smelling evacuations from the bowels. If this does not happen, the status gastricus s. saburralis (spoiled stomach) develops. Whether this is due to an acute catarrh of the stomach, a chemical change in the gastric juice, or gaseous distention of the walls of the stomach, or perhaps to a combination of these conditions, is unknown. The children have no appetite, a whitish or yellowish coating on the tongue, a bad smell from the mouth, and changed disposition. Many suffer from nausea, and vomit everything eaten. They are irritable, dull, and complain of headache. Fever is often present, occasionally to a considerable height (39°–40°), with rapid pulse (120 to 144, and more), thirst, redness of cheeks, nocturnal exacerbations, and delirium at night, more rarely in the day also. The bowels are usually constipated, less often the evacuations are thin and profuse, the epigastric region often tympanitic, tense, and tender on pressure. These symptoms may lead us to fear the development of typhoid fever or tubercular meningitis. I have previously referred to the diagnosis of the latter affection (page 120) and will here merely add that the thick, whitish yellow coating of the tongue and the fœtor oris are significant of dyspepsia. In acute dyspepsia, if it has only lasted a few days, there is no more certain remedy than the use of an emetic (P. 6). After it has acted thoroughly, the entire complex of apparently threatening symptoms often disappear as if by magic, and if the remedy has not acted as a purge at the same time, mild purgatives (P. 7, 28) are alone

necessary, or if the anorexia and coating of the tongue continue, small doses of muriatic acid (P. 3). The diet is an important element, and must be of a mild character (barley soup, light broth, zwieback, etc.), for several days, even in the slightest cases. The frequent dread of the administration of emetics is usually repaid by longer duration of the disease, and, after the lapse of six or seven days, no rapid effects can be expected from their use. In such cases keep the children in bed under absolute diet, and administer muriatic acid. If the bowels are constipated, inf. sennæ comp., or inf. rad. rhei, with kali tartaricum (P. 7) should be ordered. If the appetite returns slowly, you may prescribe tinct. rhei aquosa, two to three teaspoonfuls a day, for several days.

Reflex action from the nerves of the stomach in dyspepsia may give rise to epileptiform convulsions (page 67), or slowness of the pulse (page 120). A group of respiratory symptoms, which I have called "asthma dyspepticum," may also be produced in this manner.[1]

A girl, aged nine, presented May 10, 1875, with anxious countenance and slight cyanosis. Respiration, 70; very superficial movement of alæ nasi and other accessory muscles; moaning expiration; pulse very small, about 108. Great weakness; thoracic organs normal. Patient complains of dyspnœa, headache, and tenderness over stomach. She had been well until the night before, when she complained of stitches in the epigastrium; had been restless during the night, and the cyanosis and dyspnœa appeared in the morning. The child, after I had examined her, soon began to complain of nausea, and vomited repeatedly until night (a piece of a hard-boiled egg among other things). Immediately afterward, quiet sleep and euphoria.

You here find that the irritation of undigested food produces an apparently grave complex of asthmatic symptoms, which immediately disappear on the evacuation of the irritating substance. Compression of the thoracic organs by the dilated stomach could not be shown on examination. I have had two other similar cases. The experiments of S. Mayer and Pribram,[2] who observed increased arterial pressure and slowness of the pulse from irritation of the stomach, will not explain this process, though they account for cases like that mentioned on page 121, in which the pulse was rendered slow by the reflex irritation of inhibitory pneumogastric fibres as the result of dyspepsia. In my cases of dyspeptic asthma, however, the pulse was increased in rapidity, and there must, therefore, be other branches of connection between the gastric irritation and the respiratory disturbances. I would remind you of the peculiar oppression not infrequently occurring in dyspepsia, with or without an accumulation of gas, and which consists of the necessity experienced by the patient of taking as deep a breath as possible, although he does not succeed entirely. As soon as this is the case, the disagreeable necessity of taking a deep breath disappears for a short time, but soon reappears, and repeated spasmodic yawning then puts an end to the attack. This is also probably due to a reflex from the pneumogastric, which, under similar circumstances, occasionally produces intermittent action of the heart. I have repeatedly observed this form of asthma in children from six to ten years of age. In a couple of cases, this condition, which had existed with varying intensity for weeks, was so striking, that several of these deep and insufficient inspirations occurred in a minute, associated with action of the shoulder-muscles. In these cases, dyspepsia or distention of the large intestine with fæces could alone be detected, and treatment directed against these conditions usually produced rapid recovery. The nervous

[1] Berl. Klin. Wochenschr., No. 18, 1876. [2] Sitzungsber. d. Wien. Akad., Juli, 1872.

nature of the asthma was also shown by the fact, that when the attention of the patients was distracted, the asthma immediately ceased for a time.

Chronic dyspepsia in childhood is much more rarely the result of a gastric disorder than of some other serious chronic disease, whether general or local. Anorexia, distention of the stomach, nausea, and constipation are very often observed in anæmic children, and disappear with the improvement in the condition of the blood.

Cardialgia.

The interpretation of gastric pains, which are not infrequent in children after six years of age, is much more difficult than in adults, on account of the scanty information which we can obtain with regard to the situation and character of the pains. I have often found that, not the stomach, but the transverse colon was the seat of the pain, which was either situated in the epigastrium or one of the hypochrondriac regions, and radiated toward the umbilicus or descending colon ; these are cases of colic, not of cardialgia. I have rarely observed pains in the stomach as a result of indigestion, and they were then always associated with other dyspeptic symptoms. An emetic rapidly produced relief, and I therefore advise you not to shrink from their administration in acute dyspepsia from the fear of an inflammatory gastric affection, unless a definite cause is present, to which we may attribute such an action. Thus, in a child, who a few days previously had swallowed some scalding-hot turnips, I observed constant pains in the epigastrium, especially after eating. Here a lesion of the mucous membrane was thought of, and, indeed, the pains were relieved in a few days by an exclusive diet of milk and an oily emulsion.

Apart from the few cases in which a round gastric ulcer was diagnosed, I have only observed true cardialgic paroxysms in chlorotic girls from ten to sixteen years of age, appearing in the same manner as in adults. The distention of the epigastrium during these attacks, which causes the children to remove all tight articles of clothing, indicates a spasm of the orifices of the stomach, by which the gases within the organ are retained, and an exceedingly painful tension of its walls produced, which soon disappears after the passage of flatus and wind. In several girls, who were near the age of puberty, or had already menstruated a few times, and in one boy, I have also found dilatation of the stomach. The chief symptom is the unusual fulness or hemispherical protrusion of the epigastrium, which is more or less soft, or extremely tense and then tender, according to the degree of distention. The percussion sound, which is usually dull in the standing position, becomes tympanitic in dorsal decubitus, the distention and tenderness diminishing at the same time. The administration of an effervescent powder immediately restores the distention, often with distinctly recognizable contours of the dilated stomach. The ectasia does not always increase after eating or drinking, and as cardialgic and dyspeptic symptoms are almost always absent, the complaints are confined to a feeling of tension in the region of the stomach, eructations, or temporary nausea, but especially shortness of breath during movements or after eating. In one case of very marked dilatation, the heart was pushed upward for a distance of one intercostal space.

The etiology of these cases leaves much to be desired. In one case the affection was attributed by the mother to an attack of varioloid,

in another to an attack of typhoid fever ; but in the majority of cases the ectasia of the stomach was either preceded or accompanied by hysterical symptoms, viz.: crying-spells, cardialgia, somnambulism, ecstatic phenomena, which I have previously described (page 82). Anæmia was occasionally noticed. In one case epileptic attacks also occurred. In my opinion the affection is due, in the majority of cases, to spasm of the orifices of the stomach, to which too much importance should not be attached. After it has lasted for weeks or months, the disease usually recovers spontaneously, or gives place to other hysterical symptoms. I cannot decide from my own experience whether the appearance of menstruation acts favorably, but I consider it probable. Among the remedies which I have employed, the faradic current has proved successful, though only temporarily. If one electrode were placed on the spine, and the other over the distended epigastrium, the latter always retracted without any escape of ructus, and it remained doubtful whether this was due to contraction of the abdominal muscles or the muscular coat of the stomach. But this result was only temporary, sometimes lasting only a few hours, at the most a few days. No permanent results were obtained even in those cases in which electrical treatment was persistently used for three or four weeks, and I can therefore only recommend it as a palliative in severe cases.

The most frequent cause of dilatation of the stomach in later life, viz., stenosis of the pylorus or duodenum, has not come under my notice in childhood, and in only one case (a girl, aged eight) have I observed ectasia due to enormous filling of the stomach with food. In such cases, after the stomach and intestines have been emptied, a strict diet which excludes all vegetables should be recommended, and, at the same time, the atony of the stomach, due to its extensive distention, relieved by ice-bags, extr. nuc. vomic., and electricity.[1] This category also includes the dilatation of the stomach due to fermentation dyspepsia, which is recognized by occasional emesis of profuse acid, frothy masses of fluid containing fermentation fungi. I have repeatedly observed these symptoms in children from eleven to fourteen years of age,[2] but they did not differ in any respect from those observed in later life.

VII. Cholera Infantum.

This disease attacks children of every age, but is by far most common in the first and second years, at which period it exercises its most destructive influences. This very fact shows that the manner of nourishment, especially the artificial, or the transition from the breast to weaning, plays an important part, especially as infants who receive good mother's or nurse's milk are much more rarely affected than others. Thus far we stand upon a basis of fact, but beyond this everything is hypothetical. However, another factor, which we may boldly call "infectious," must very probably be taken into consideration. This view is favored by the epidemic occurrence of the disease in the hot summer months—June, July, and August—especially in large cities. This is so constant that the disease is properly called cholera æstiva (summer complaint). Every physician knows that, with the first warm days of early summer, cases of this disease make their appearance, increase in frequency every week

[1] Beitr. z. Kinderheilk., N. F., S. 282. [2] Ibid., S. 314.

until they become epidemic, and finally disappear gradually in September, although some "feelers" of the epidemic are observed in October. The nature of the disease is, however, entirely unknown. Definite forms of bacteria to which the infectious character can be attributed have not been found. But we may hope for more satisfactory results from further investigations if we take into consideration the cases of intestinal mykosis [1] reported in literature. These rapidly fatal choleraic diseases appear to be produced by fungi, like those of splenic fever, and are found not only in the intestinal contents, but also in the epithelium and submucous tissue of the intestines, whence they have migrated into the blood-vessels and lymphatics. At present, however, we can regard the mykotic theory of cholera infantum only as a very probable hypothesis. There is no doubt that high atmospheric temperature increases the tendency to fermentation dyspepsias which is present in imperfectly nourished children at all seasons (page 52), and causes them to appear not only epidemically, but also in an extremely acute form, which is not frequent under ordinary circumstances. This would lead to the conclusion that, in addition to the heat, infectious germs are present, which, being developed in great masses by the former, enter the stomach with the food and further the process.

The symptomatology of cholera infantum of small children is very similar, anatomically and clinically, to acute dyspepsia of nurslings (page 53). In both cases we find various grades, from more or less profuse diarrhœa to the most severe, rapidly fatal cholera infantum. In the beginning there are rapidly following, brownish yellow or greenish, thin evacuations. Pain is absent, or very mild. Apart from anorexia and increased thirst, the general condition may be unaffected, and with proper care the diarrhœa either disappears spontaneously, or, under appropriate treatment, within twenty-four to forty-eight hours, as soon as the abnormal contents of the intestines have been removed by increased peristalsis. For this reason, the same diet and treatment are advisable as in acute dyspepsia (page 56), especially hydrochloric acid or small doses of calomel. In older children, abstinence (gruel) and rest in bed should be urgently recommended. In another series of cases the disease begins with violent symptoms. Profuse watery evacuations and vomiting rapidly follow one another. The severity of the latter varies: at times it is rare, then very frequent, occurring whenever fluids are ingested; and cases are not wanting in which vomiting plays the principal part, and only a few thin evacuations occur in the course of the day. The rapid effect on the bodily powers is common to all, and occurs so much more rapidly and severely the younger the child, though it is not absent in older children, and, as you know, even in adults. Great weakness, pallor of the skin, sinking in of the eyes in the orbits, coolness of the cheeks, hands, and feet, increasing frequency and smallness of the pulse, feeble voice, slight cyanosis of the face and mucous membrane, indicate the depression of the power of the heart. The primary restlessness and jactitation soon pass into an apathetic, somnolent condition. The tongue and mouth are dry, thirst enormously increased, the abdomen very slightly or not at all distended, and not tender on pressure, the excretion of urine considerably diminished, on account of the large loss of water from the stomach and intestines.

In these severe cases the passages, which primarily had a fæcal color

[1] Burkart: Klin. Wochenschr., No. 13. 1873.

and bad odor, very soon become watery and slightly yellow, and finally almost colorless. There is usually no admixture with mucus or blood, and if traces of the latter do occur, they come from the lower part of the rectum or the neighborhood of the anus, which have been eroded by the copious evacuations. I have repeatedly examined these passages microscopically, and the only formed elements found have been desquamated intestinal epithelium and vibriones, as they also occur in other diarrhœal evacuations.

While many cases of this kind gradually recover under appropriate treatment, a large part of those which attack early childhood, especially under unfavorable surroundings, terminate fatally, and the mortality of the summer months furnishes a terrible proof of the fury with which this disease decimates the population of large cities. Death always occurs as the result of rapidly increasing exhaustion under symptoms of collapse and hydrencephaloid (page 118), cadaverous pallor, cyanosis, continuous somnolence with half-closed lids; finally, complete coma, depression of the open fontanelles, coldness of the extreme parts, and imperceptibility of the pulse. In the last stage a dark shadow is found almost constantly around the sunken eyes, especially on the lower lid, which is caused by the projection of the edges of the orbit beyond the sunken globe. From this symptom we can often recognize the severe form of cholera infantum at the first glance. Nor have I hardly ever failed, in the last stage, to observe the streaked injection of the conjunctival vessels and the flakes of mucus on the conjunctiva, which have been repeatedly referred to (page 121). Partial opacities of the cornea occur not infrequently, especially of that portion which is no longer covered by the half-closed lids. But, as in tubercular meningitis, these changes in the eyes always appear as a decided lethal symptom, which deceived me in only one case. The child recovered, although the characteristic flakes of mucus had already obscured the cornea; but it should be taken into consideration that a mild catarrh of the conjunctiva had existed in this case before the occurrence of the cholera infantum. The symptom therefore loses none of its decidedly lethal significance. Occasionally the evacuations from the stomach and intestines suddenly cease during the last stage, to the joy of the parents, who now indulge in hope. But I warn you against such precipitation, unless it is associated with increased strength and general improvement. I have often found that, despite the cessation of the evacuations, hydrencephaloid continued to develop, and terminated fatally.

No characteristic appearances are found in autopsies upon these children. Extreme pallor of the entire alimentary mucous membrane is often observed, perhaps also slight swelling of the solitary glands and Peyer's patches; in other cases, catarrhal redness and swelling in patches of the gastric and intestinal mucous membrane. Partial pulmonary atelectasis, venous congestion of the brain and pia mater; recent thromboses of the sinuses or other veins—for example, the renal veins—were frequently found, and must be attributed to weakness and deficient heart's action. The disease cannot, therefore, be regarded as a catarrhal affection. The usually negative results of autopsical examinations favor the view of an abnormal chemical process in the contents of the stomach and intestines, caused by unknown influences, and which, after lasting for a number of days, may give rise secondarily to catarrhal processes from the constant irritation. In this manner I explain the fact that many children, after recovery from cholera infantum, suffer for a considerable time from ordinary intestinal catarrh.

The great danger of the disease explains the large number of remedies which have been employed. There is no specific against cholera infantum, and we are unable to destroy the infectious germs which have entered the stomach and intestinal canal. Neither quinine, carbolic acid nor salicylic acid have proved efficient, and hydrate of chloral (1:120) only possesses a palliative effect on the vomiting. As it is impossible to destroy the true producers of the disease, it only remains to combat their effects, *i.e.*, the fermentative processes produced in the stomach and intestines, and in all cases in which the quantity of the infectious elements introduced has not been too large, we may succeed in securing recovery after the complete evacuation of the toxic and fermenting substances. In the opposite event, treatment will be useless, and the children will die, despite all our efforts.

It follows that, in the treatment of cholera infantum, we must resort to the same remedies which I have recommended in the treatment of fermentation dyspepsia (page 56), and that the immediate administration of opium, which retains the deleterious masses in the intestinal canal, is as much out of place here as there. In recent cases, *i.e.*, in the first two or three days, we may give small doses of calomel (P. 2) and hydrochloric acid (P. 3), or creosote (P. 4), if the former prove unsuccessful. When signs of weakness appear, order, twice daily, a warm chamomile-bath (28° R.), in which the children remain five to ten minutes, and port, Hungarian, or sherry wine (20 gtt. to a teaspoonful, according to age) every two or three hours. Wine is often retained when other articles of food (milk, bouillon) and the medicines are immediately ejected. I would advise the administration of milk only when cooled in ice and given by the teaspoonful (page 55). If the affection, nevertheless, continues, or the case comes under treatment only after the lapse of a few days, I no longer hesitate to employ opium, as it may be assumed that the infectious elements have already been discharged. I then add three to ten drops of laudanum, according to age, to the hydrochloric acid mixture, and perhaps give a starch injection, containing one to two drops of laudanum, several times a day. The sick-room should be as large as possible and the bed-linen be kept carefully clean. When the hydrencephaloid condition becomes more marked, injections of camphor (P. 14), cold champagne (a teaspoonful to a tablespoonful at a time); finally, hydropathic applications and effusions of the entire body should be tried. The almost unquenchable thirst of the little patients, which is manifested by greedily opening the mouth at sight of the cup or spoon, is best satisfied by the administration of cold milk or ice-water. If catarrhal diarrhœa remains after the subsidence of the severe symptoms, those remedies should be employed which I will mention in describing the latter affection.

VIII. Catarrhal Diarrhœa.

As I have already remarked, catarrh of the intestinal mucous membrane may be caused by the prolonged action of chemically abnormal contents, especially by fæcal masses in a condition of acid fermentation, and we must interpret the diarrhœa which often remains after cholera infantum in the same light. I have repeatedly observed that irritants of other kinds, especially foreign bodies, may exercise the same action. In a girl, aged two and one-half years, vomiting and obstinate diarrhœa resulted from the ingestion of pieces of chalk and egg-shells; and, in a hairdresser's child,

the ingestion of hair produced an extremely obstinate mucous diarrhœa, which was only relieved by a few doses of castor-oil, after its cause, the hairs, had been discovered in the evacuations. In addition to these direct irritants of the mucous membrane, atmospheric influences (cold, wetting) may also act as causes of intestinal catarrh. In a third series of cases it occurs secondarily, after certain general diseases, most frequently measles. As a rule, the colon is much more often and severely affected than the small intestines.

Catarrhal diarrhœa is especially recognized by the more or less profuse admixture of tough shreds or balls of mucus in the passages, to which points and streaks of blood not infrequently adhere. Tenesmus is very often observed in the form of repeated straining after defecation, during which a small part of the dark red mucous membrane of the rectum may be extruded. The straining is especially observed in those cases in which the lower part of the colon and the rectum are the site of the disease, while, when this is situated higher, the thin masses merely squirt from the anus in a noisy manner. Occasionally I have seen numerous live ascarides in the discharges, which have been dislodged from their nests by the vigorous movements of the intestines and the current of the fluids.

Fever may be entirely absent, or present a mild remittent type (morning temperature entirely or almost normal; evening temperature, 38.0° to 38.5°). The appetite may be normal or perhaps diminished, and the tongue has a moderate gray coat. The discharges are often preceded and accompanied by colic pains.

Almost all cases terminate favorably under good care and treatment. An exacerbation to severe grades, which may be termed acute enteritis, must be very rare, as such cases have only exceptionally come under my notice:

A feeble boy, aged two and one-half years, who had often suffered from diarrhœa, had been suddenly seized, twelve days before my first visit, with severe epileptiform attacks, high fever, and profuse diarrhœa. The two latter symptoms resisted all methods of treatment. From twelve to fifteen passages occurred every day; they were thin, of a spinach-green color, mixed with considerable mucus, and were preceded and accompanied by severe crying. In the next few days, increasing collapse, apathy, small and very frequent pulse, slight tympanites. Finally, cessation of the diarrhœa, considerable increase of tympanites, coma and death on the seventeenth day of sickness. Autopsy: general anæmia, fatty liver, severe follicular enteritis, extending from middle of small intestines to the sigmoid flexure; congestion and thickening of mucous membrane, numerous enlarged and ruptured follicles; in many places the mucous membrane is perforated like a sieve by very small round ulcers with congested borders; redness and meshed appearance of Peyer's patches.

The onset of this case, with high fever and reflex eclampsia, indicated the severity of the affection. The disease was an extensive acute follicular enteritis, which must probably be regarded as a sudden exacerbation of a primary chronic intestinal catarrh from some unknown cause (error of diet, exposure). The opposite condition is much more frequently observed, viz.: that a simple intestinal catarrh is allowed to last many weeks and months before the physician is consulted. This transition into a chronic condition, to which I have previously referred (page 53), is almost always the result of negligence, and occurs very frequently in dispensary practice. The abnormal evacuations continue for a number of weeks with varying frequency, at times occurring rarely, at others ten to twelve times daily; in some cases the shape of the abdomen remains

normal, and colic may be entirely absent, while in others complaint is made of pain before the evacuations and tenesmus, and the abdomen is somewhat tympanitic. The fæces are more or less fluid, their quantity and appearance vary greatly, either greenish brown, blackish, or much brighter, mixed with mucus, and very foul-smelling; not infrequently they contain small quantities of blood. If the diarrhœa is not very profuse, it may continue for months in vigorous children without any perceptible diminution of the appetite and nutrition. In the majority, however, emaciation, flaccidity of the skin and muscles, at first in the adductors of the thighs, and pallor of the integument, soon become noticeable. If the diarrhœa is not checked, these symptoms increase from week to week to a high degree of weakness and atrophy. Prolapse of the intestinal mucous membrane often occurs during an evacuation, and finally the fæces run almost continuously from the paralyzed anus. A remittent fever (38°–39° at night) almost always accompanies this sad condition, which terminates fatally in increasing collapse, not infrequently with the development of a terminal broncho-pneumonia. Toward the last, sprue often forms in the buccal and pharyngeal cavities, and œdematous swellings occur in the feet, hands and face, which must usually be regarded as the results of the failing heart's action and the venous stasis produced in this manner; they are much more rarely due to thrombosis of the larger veins, or to nephritis.

The intensity and extent of the anatomical changes cannot be determined before death, even in these apparently most severe cases. I have often found that the autopsy furnished results which did not harmonize with the symptoms during life. The congestion and swelling of the mucous membrane, which usually has a brownish or grayish red color in these chronic cases, may affect larger or small surfaces, may or may not be associated with swelling of the intestinal follicles, with extremely few small ulcerations in the region of the ileo-cæcal valve, or with very numerous follicular ulcers of the small and large intestines, although the symptoms during life did not correspond to the grade of these anatomical changes. We should especially guard against the hasty assumption of extensive ulcerations, as soon as profuse diarrhœa, increasing atrophy, and remittent fever make their appearance. I was often surprised at finding, under these circumstances, merely a moderate intestinal catarrh, and a few follicular ulcers in the neighborhood of the ileo-cæcal valve, or in the colon. We should not fail to examine the mucous membrane of the rectum in such cases, because the catarrh and ulceration may be most strongly developed there, while the upper parts of the intestines present slighter changes. On the other hand, I have repeatedly found the entire mucous membrane, from the lower end of the ileum to the rectum, of a dark red or slate color, and strewn like a sieve with numerous follicular ulcers. Fatty infiltration of the liver is also frequently observed.

Catarrhal diarrhœa in childhood must be carefully treated from the beginning, as it presents more danger than in adults; on the one hand, on account of the greater tendency of the intestinal follicles to hyperplasia and ulceration, and, on the other hand, the tendency of the mesenteric glands to become enlarged and cheesy from repeated or long-continued irritation of the intestinal mucous membrane. I need not refer again to the further possibility of general miliary tuberculosis from this cause.

In the treatment of a recent catarrhal diarrhœa, we must first determine whether it was preceded by constipation, and also whether an attack of indigestion was the exciting cause of the disease. Under such circum-

stances it is best to begin with a mild purgative, a teaspoonful of castor-oil, or a few doses of calomel (0.015–0.03), especially if tenesmus is present and the passages are small in quantity, and mixed with bloody points or streaks. After the purgative has operated the diarrhœa not infrequently disappears after a few days. As almost all primary intestinal catarrhs of childhood have a dyspeptic origin, we may use purgatives at first in almost every fresh case, even if the causative indigestion and dyspepsia cannot be positively proven. But if profuse, thin evacuations have lasted for days, or exposure, or the misuse of a purgative such as tartar emetic, can be proven to be the cause, I would advise you to keep the children warm in bed, to give only farinaceous food, and prescribe inf. rad. ipecac., with a small dose of tinct. opii (P. 29). I prefer this formula to Dover's powder, because it does not produce nausea. Instead of the tincture you may give extr. opii aquosum (0.002–0.005 at a dose). If the diarrhœa resists these remedies, and continues for a week or more, I would recommend subnitrate of bismuth in large doses (0.1 in the first year; later up to 0.3 every two hours, P. 30). In a few days the stools often become more consistent, and assume a gravish green color, but it should be continued ten to fourteen days, in order to prevent a relapse. In very obstinate cases, extr. opii aquos. (0.003–0.005) may be added to each powder. We meet with some cases, however, which become chronic despite the use of this remedy. We then resort to astringents, among which the infusion of colombo or cort. cascarillæ, with small doses of opium (P. 31, 32), are often effective, though difficult of administration on account of their bitter taste. Tannic acid (0.05–0.1, at a dose) may impair the already diminished appetite, but often does very good service, especially in combination with tinct. nuc. vomic. (P. 33). Among metallic remedies you may employ nitrate of silver (0.002–0.003, P. 34), and, if unsuccessful at the end of a week, acetate of lead (0.01–0.015) three times a day, with small doses of extr. opii aq. (P. 35). I have occasionally been successful with the latter remedy in apparently hopeless cases, and have never observed any injurious toxic action.

But all these remedies may prove useless, or even act injuriously, by producing anorexia, nausea, or vomiting. For such cases we may resort to the injection or pouring of medicinal fluids into the intestines, a plan which may produce surprising results if faithfully carried out. We may employ an ordinary irrigator or glass funnel, to which a rubber tube, with an end-piece of bone or ivory, is fastened. The end passed into the rectum should be free in its cavity, so that the fluid can escape. The injections are best performed in the knee-and-elbow position, but it can also be done in right lateral decubitus. As a rule we employ a solution of acetate of lead (5:1,000), more rarely alum and tannin (20:1,000), of which about 300.0–500.0 are introduced each time. If the mucous membrane is very sensitive, a part of the fluid is evacuated during the injection, while otherwise it usually remains five to ten minutes or longer in the intestines. However, my expectations with regard to this method of treatment have not been entirely fulfilled. Although I have met with individual cases in which the first injections produced surprisingly favorable results, in many others no success whatever was obtained, or it was only temporary. But the method is well worth a trial in obstinate cases; it requires persistent use, as a favorable effect cannot be expected in the first few days.

Regulation of the diet, without which recovery is impossible, meets with great obstacles in children. It should be confined to meat-broths,

milk, red wine, soups, eggs, rice, farina and finely chopped meat; all articles which have a tendency to ferment—vegetables, fresh and cooked fruit, leguminous articles, etc.—must be prohibited. The only objection to raw scraped meat is the fact that it may give rise to tape-worms. Whether milk is suitable or not must be determined on trial. I am never afraid to recommend it, and often found the passages grow more consistent at once under milk diet. In older children I would also recommend dried bilberries (vaccinia myst.), which are made into a thick compote, and one or two saucerfuls given daily. This popular remedy has produced surprising results in many, though not serious cases.

I will now add a few words with regard to that form of intestinal catarrh which occurs secondarily in the course of some other disease, and constitutes an important feature of the symptomatology. The infectious diseases, especially measles and typhoid fever, must be chiefly considered in this respect. While circumscribed catarrh of the mucous membrane is scarcely ever wanting, anatomically, in these diseases, it is not always manifested clinically by diarrhœa, and cases of typhoid fever with constipation are not rare. In measles the intestinal catarrh is often present at the height of the affection, and many epidemics are characterized by obstinate diarrhœa. This is more rare in scarlatina, where it may have an unfavorable significance, especially in the first stage; most cases which began with profuse diarrhœa were malignant in character, and soon terminated fatally.

I have also found the anatomical changes of intestinal catarrh in children who have died of various diseases, and who, during life, had suffered little or not at all from diarrhœa. Indeed, hemorrhagic enteritis, of a croupous or diphtheritic character, in patches, is found at times, though no serious symptoms on the part of the intestinal tract were present during life. This was most marked in two cases of chronic nephritis.

A girl, aged eleven, admitted with caries of petrous portion of temporal bone and chronic nephritis, and died in a few days from uræmia, no intestinal symptoms having been noticed during life. At the autopsy the lower third of the mucous membrane of the ilium was dark red from congestion and hemorrhagic infiltration, and covered in patches with a fibrinous membrane.

The cause of this intense irritation of the mucous membrane must remain undecided; perhaps it was partially due to the urea excreted by the latter, and which had undergone decomposition.

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IX. Dysentery.

Dysentery develops more frequently from catarrhal diarrhœa in childhood than in adult life, and during the first two years of life we may remain in doubt whether we have to deal with infectious dysentery or a non-infectious acute catarrh of the large intestines. Mucous and small quantities of blood may occur in the evacuations of every catarrhal diarrhœa, and tenesmus is also not an infrequent symptom. The case is regarded as dysenteric only when the passages are repeated very often, and contain bloody mucus or small quantities of fæces mixed with considerable mucus and blood. Anatomically and clinically this is merely a colitis, the infectious nature of which is shown by its epidemic occurrence in certain seasons, especially in the months of August and September.

The disease almost always begins with diarrhœa; the dysenteric evacuations do not make their appearance until twenty-four to forty-eight hours later, consisting of small quantities of tough, vitreous, brownish mucus streaked with blood, which is discharged very often, perhaps five or six times, or more, in an hour, associated with severe tenesmus. Colic, tenderness and distention of the abdomen, and repeated vomiting are not infrequently observed; the appetite is lost, thirst increased. Fever may be absent or moderate (38.5° to 39° in the afternoon and evening), while more severe cases present a remittent fever, with remissions in the morning (38.0° morning, 39.5° evening). The process may terminate in eight to ten days, but often lasts much longer, in some children fully three weeks. Even in milder cases greater weakness is manifested than in simple intestinal catarrh of equal duration. In severe cases the weakness soon increases to a dangerous collapse; coolness of the extremities, thready pulse, great apathy and somnolence, only interrupted by tenesmus and severe colic, subnormal temperature (36° to 37°), finally paralysis of the sphincter ani, with continual discharge of a foul-smelling mucus, which often contains membranous shreds and clots of blood, gradually terminate in death. When the sphincter ani is paralyzed I have often been able to get a good view of the rectum without introducing a speculum, and have always found extensive losses of substance in the mucous membrane, usually from diphtheritic necrosis:

Max M——, aged seven, admitted July 13, 1877. Severe diarrhœa five days previously from eating too many cherries. On the next day the passages contained mucus and blood, and occurred almost every fifteen minutes, with severe colic and tenesmus; apathy and fever soon developed. On admission, great weakness, eyelids half closed, T. 39.4°, P. 132, small grayish yellow coating on tongue, abdomen depressed, slightly sensitive, flaccid. Constant tenesmus, colic, six to eight scanty evacuations every hour, consisting of brownish green bloody mucus. Death on following night.

Autopsy.—Congestion of mucous membrane of lower part of ileum, beginning 1' above the valve, soon associated with diphtheritic infiltration. Numerous ulcers in ascending and transverse colon, from desquamation of diphtheritic membrane; still more numerous ones in descending colon, in which fresh diphtheritic infiltration again appears. This extends to upper third of rectum, lower two-thirds being free. Other organs intact.

In other cases the disease may last for many weeks or months (chronic dysentery). During this period, the symptoms present a varying intensity, and a temporary feculent condition of the evacuations may lead to illusive hopes. In a girl, admitted September 19, 1876, the disease continued in this manner for nearly eight weeks, the morning temperature being normal or subnormal, the evening temperature rising to 39°–39.7°. Here a striking change occurred from purely bloody and mucous stools to more consistent evacuations, which even contained scybala, but the weakness and emaciation nevertheless progressed steadily despite all treatment; the autopsy showed severe diphtheritic infiltration of the mucous membrane of the large intestine. We can only hope for a favorable termination in mild and moderately severe cases in which, as we may assume, the anatomical changes have not progressed beyond more or less intense catarrh of the colon with swelling and perhaps ulceration of the follicles. The prognosis is very unfavorable and recovery extremely rare when the symptoms reach a severe grade, especially when larger shreds of necrotic mucous membrane appear in the evacuations. Even in the most favorable case, stenosis of the colon or rectum may occur from the retraction of necrotic patches; this may occur even in cases of moderate intensity.

I have several times observed this after not very severe dysentery. Mucous membranous masses, streaked with blood, were passed from time to time without pain or tenesmus, the evacuations being otherwise normal and the general condition undisturbed. These stools occurred perhaps every day for one or two weeks, and then the evacuations became normal for weeks and months. If the passages were spread out in water, they formed delicate, bloody, floating shreds which, under the microscope, were found to be formed largely of a structureless mass, and in part of a fibrinous substance, strewn with blood-globules and leucocytes. I have seen these passages recur from time to time for years after an attack of dysentery. As local examination showed nothing abnormal in the rectum, we may assume that ulcerations or circumscribed inflammatory processes were situated higher up in the colon.

The treatment of dysenteric conditions is the same, in general, at all periods of life. I always begin treatment in fresh cases, which have not lasted more than a few days, with a mild purgative—for example, a teaspoonful of castor-oil or a larger dose of calomel (0.1–0.3), and give an emulsion of castor-oil (P. 36) or calomel 0.03–0.05 every two hours for a few days. After the stools have become feculent, I order inf. rad. ipecac., combined with tinct. theb. or ext. opii aq. (P. 29) if the pains and tenesmus continue. When the abdomen is very much distended and tender, an ice-bag should be applied and nourishment restricted to milk cooled in ice, combined perhaps with oat-meal, gruel, and bouillon. If recovery does not occur under this treatment, I would recommend the previously mentioned injections of tannin, alum, and especially acetate of lead, which should always be preceded by intestinal irrigation with lukewarm water or a solution of salicylic acid (1 : 1,000). These injections may be made twice a day. The inefficiency of these intestinal irrigations in very severe cases is proven by the case reported above (page 206). But they are decidedly preferable to enemata of emollient or astringent fluids which do not pass beyond the rectum and only offer any chance of success when we can assume that the chief changes are found in the rectum. But that all treatment is useless in the most severe cases will be evident to every one who has had the opportunity of observing, on the autopsy-table, the terrible ravages of the intestinal canal produced by this disease.

X. Constipation.

The aid of the physician is not infrequently required on account of constipation in young children, even in nursing infants. Many children remain constipated for days, or, with a great deal of exertion, during which they become red in the face, discharge hard scybala, which clatter in the vessel like stones, and may give rise mechanically to small erosions and hemorrhages from the anus. Traces of blood are therefore often found upon these hard lumps of fæces, which are usually of a pale yellow or whitish gray color, and occasionally look exactly the same as in jaundice, although no trace of icterus can be detected and the general condition is unchanged. The color could, therefore, only be due to deficient secretion of bile, or a paler color of its pigment, but nothing is at present known with regard to the subject. In nurslings the constipation usually ceases with a change of nurses, or at the period of weaning, but may also continue into later childhood, and only disappear after the ingestion of mixed diet and active bodily exercise. Before calling in the physician, the mothers usually endeavor to remedy the evil by the administration of magnesia and rhubarb, pulv. liquor. comp., castor-oil, but they must use constantly increasing doses to produce sufficient action. It is best in

such cases to exclude all internal remedies, and confine the treatment to a daily enema of cold water, to which a pinch of salt may be added in very obstinate obstruction.

Much more rarely than in adults I have observed in children that form of fæcal accumulation in the large intestines which develops gradually, although the patients have an evacuation daily, or at least every other day. In these cases only a small part of the intestinal contents is discharged; the larger part accumulates in the colon, and finally produces morbid symptoms. I do not now refer to the accumulations in the cæcum, which lead to typhlitis stercoralis from distention, but to the enormous distention of the entire abdomen, which I observed in two boys, seven and nine years old. Not only the parents, but the physician also, were seriously alarmed on account of the hemispherical shape, great tension, and partial tenderness on pressure, and I myself could not, at the first glance, suppress the suspicion of chronic peritonitis. Both patients were the children of wealthy and very devoted parents, and were fed with all sorts of dainties (oysters, pastry, etc.), no attention being paid to the insufficient defecation. Thus, an enormous distention of the colon gradually developed from masses of fæces and the gases produced by them. Only after the daily administration, for weeks, of purgatives and strict diet (meat, exclusion of farinaceous and leguminous articles), was it possible to gradually diminish and finally remove the distention of the abdomen; the enormous scybalous and soft, extremely dark and fœtid evacuations, which daily filled entire vessels, excited our astonishment. I would recommend, in such cases, electuar. sennæ, either pure in doses of one to two teaspoonfuls daily, or according to P. 28. In a very few cases it produces severe colic, so that it must be discontinued; usually it may be given continuously for a couple of weeks.

In a few cases, which occurred exclusively in the first two years of life, the constipation was evidently due to pain in the anus. At the moment of defecation a spasmodic, painful contracture of the internal sphincter occurred, and rendered the evacuation of fæces impossible. Every attempt caused crying, and was at once abandoned, so that occasionally several days elapsed before a passage occurred. Upon examination of the anus, one or two narrow red vertical fissures were found in the folds of mucous membrane surrounding the anus, which were extremely tender to the touch, and were perhaps produced by the mechanical action of hard scybala. The painful contracture of the sphincter, which prevented defecation, appears to be a reflex effect from these fissures.[1] But this appearance is by no means constant. In a child, aged eighteen months, I could detect no fissure despite the most careful examination, yet the contracture of the sphincter was so powerful that the attempt to pass through the anus with the little finger met with vigorous opposition. After I had forcibly effected an entrance, during which I distinctly experienced the sensation of a partial rupture, the difficulty was at once relieved. The pain and contracture ceased, and, after castor-oil had been administered for a few days, defecation occurred without any further disturbance. But this mechanical treatment is insufficient when fissures are present. In such cases we must endeavor to produce recovery by the application of lunar caustic or an ointment of tannin (1 : 20), together with the administration of purgatives. We must act with decision under all circumstances, as otherwise the masses

[1] Betz (Memorabil., IV., Lief. 12) observed similar cases resulting from eczema ani.

accumulated in the rectum will give rise to constant tenesmus, and, in a little while, to the secretion of badly smelling mucus.

Under the term ileus is meant the most obstinate and acute form of obstructio alvi, which is soon associated with vomiting of brownish fluids of a fæculent odor. This form of obstruction may occur soon after birth, when congenital stenosis or complete impermeability of the intestinal tract is present in any part of its course. These congenital defects are found most frequently in the duodenum and ileo-cæcal region; I will not enter upon their discussion, because they permit neither a positive diagnosis nor successful treatment.

Nor will the consideration of atresia of the anus detain us, as this belongs to the domain of surgery. I will at once proceed to discuss those morbid processes which produce the symptoms of ileus in the normally developed intestinal canal.

As a rule, we find the same conditions as in adult life, though all are not present with equal frequency. For example, incarcerated herniæ, which assume the first rank among the causes of ileus in adults, are rare in childhood. However, such cases have occasionally been observed during the first weeks of life, and have been successfully operated upon.[1] The examination of the well-known hernial openings should, therefore, never be omitted in cases of ileus. But neither this form of ileus nor that produced by volvulus or obstructing intestinal contents present any differences from similar cases in adults. A peculiarity of childhood is the rare obstruction of the intestinal lumen by a ball of round worms, but in this case we can merely entertain a suspicion with regard to the diagnosis. Only one form of ileus, and one which is the most frequent in childhood, presents characteristic symptoms; I mean

Intussusception.

We do not now refer to the small invaginations found in the small intestines in the bodies of many children who have died from the most various diseases. The absence of all symptoms and the readiness with which the invaginated intestine yields to slight traction with the hand shows that the invagination must have occurred shortly before death. The intussusception now under consideration affects a much larger part of the intestine, and usually in such a manner that the lower end of the ileum, together with the cæcum, is wedged into the ascending colon, and, advancing further downward, forces the latter into the transverse or descending colon. In many cases, however, the invagination is much more circumscribed, and attains only a length of 6–8 ctm.; on the other hand, cases have been repeatedly observed in which the lower end of the ileum, the ascending and transverse colon were forced into the descending colon, and the tip of the intussusception could be felt in the rectum, or was even forced from the latter.

By far the largest number of invaginations in childhood occur during the first year, but the cause of this predisposition is unknown. The diagnosis of this condition depends chiefly on the association of three symptoms, viz., constipation, vomiting, and escape of blood from the anus. As a rule, the disease begins suddenly, in the midst of perfect health, with violent cries, great restlessness, repeated vomiting, and con-

[1] Berl. Klin. Wschr., 1879, S. 488 u. 677 ; Demme's Jahresber. f. 1878, S. 58.

stipation. Purgatives and enemata are ineffectual; the latter are immediately evacuated, and an escape of blood from the anus frequently occurs on the first day, and almost always in the later course of the disease. At first it is mixed with fæces, later with mucus, but may also be discharged pure, in part coagulated, in varying quantity. Occasionally, one to one and a half tablespoonful of blood are evacuated at once. Tenesmus is rarely absent, and five, six, or more passages often occur in the course of the day. Fluids, which are eagerly desired by the patients, almost always produce vomiting. During the first twenty-four to forty-eight hours the abdomen may retain its normal shape and softness, but then usually becomes tense, tympanitic, and tender. As soon as this occurs, we are no longer able to detect distinctly by palpation the tumor produced by the invagination in the course of the colon. This is possible, however, so long as the abdomen is soft and its walls yielding. On two occasions I succeeded, by introducing the finger deep into the rectum, in distinctly feeling the rounded tip of the intussusception. This is very like the vaginal portion of the uterus, presenting a round or slit-shaped opening, situated either centrally or more to one side, and into which the finger can penetrate slightly. This is the markedly compressed and swollen lumen of the invaginated portion of the gut. Under these circumstances, the diagnosis is undoubted. Much more rarely the constant tenesmus presses the intussuscepted portion downward for a few centimetres, and it then lies in front of the anus as a dark red, bloody tumor, with a central opening.

If the invagination cannot be felt either within or without the rectum, the diagnosis cannot be made with absolute certainty, but with great probability, from the fact that, as I have previously mentioned, all other causes of acute ileus in early childhood occur infinitely more rarely, and that the combination of the three symptoms—obstinate constipation, vomiting, and hemorrhage from the mucous membrane of the invaginated intestine—are almost decisive, according to the experience of all authors. The later course of the disease corresponds exactly to that observed in adults. In the unfavorable cases, which unfortunately constitute the large majority, increased meteorism, constant whimpering and crying, which finally give place to complete apathy, cool cheeks and extremities, small and extremely frequent pulse, finally fatal collapse after an average duration of the disease of four to eight days; in favorable cases, restoration of the invagination, with discharge of flatus and fæcal evacuations or necrotic desquamation of the invaginated portion of intestine, with formation of a more or less normal intestinal lumen, and a corresponding shortening of the canal.

C——, aged one year, suffered constantly from constipation, but during last few days passages have been normal. October 15, 1873, great restlessness at night, no sleep, one evacuation, consisting of blood and mucus, vomiting of milk toward morning. Castor-oil and enemata ineffectual. Vomiting and another bloody passage. October 17th, somnolence, increasing weakness, no evacuation. At night a large injection of ice-water, followed in an hour by a brown, fluid evacuation, in which the castor-oil taken on the 16th could be distinctly recognized. Several passages during the night. October 18th, everything normal except weakness.

Ph——, aged one year. During the evening of February 27, 1875, sudden violent crying and vomiting. On the following morning, discharge of pure blood from anus, moderate meteorism. These symptoms continued with increasing severity until March 2d, when I first saw the child. Vomiting had ceased, but no fæcal evacuation could be obtained; beginning collapse. Upon introducing the finger, I could distinctly feel the intussusception in the middle portion of the rectum. On withdrawing the finger, dirty brown fluid escaped from the anus, and, at the same time, a necrotic

portion of the intestine about two and one-half inches long, which belonged to the colon, as the examination showed. The symptoms of ileus then ceased. March 6th, obstruction again occurred, meteorism increased, and the constant cries indicated severe colic. Nevertheless, the general appearance improved, vomiting ceased, and the child took broth, wine, and iced milk. After March 8th, eight to ten fluid brown passages occurred daily, of a gangrenous odor, but containing no shreds of intestines. The meteorism gradually diminished, the appetite improved, the pains became more infrequent, but the diarrhœa continued for some time, and did not disappear entirely until March 30th; complete recovery.

In both cases an invagination of slight extent must have existed, which suddenly resolved in the first child, and in the second was thrown off by necrosis after the extremely short duration of about three to four days. This event was not followed at once by complete recovery, but by severe intestinal catarrh, which kept us in suspense for weeks, and endangered the life of the patient. Whether this was due to the direct irritating effect of the intussuscepted portion upon the mucous membrane or to the gangrenous fragments, which could only be gradually discharged, must remain undecided. The gangrenous smell of the passages favors the latter view.

These spontaneous recoveries are rare at all ages, and in children especially the separation of the invaginated portion of intestine is observed even more rarely than in adults. For this reason, the physician will hardly be satisfied with the expectant plan of treatment. As soon as the diagnosis is made, the administration of purgatives, which can only do harm by increasing peristalsis, is usually discontinued. Many even dread the use of enemata, because they are at once expelled, and favor the further advance of the invagination, in the same manner as purgatives. This view does not appear to me, by any means, to be proven. In addition to many others, our first case, in which a fæculent evacuation occurred an hour after the application of the first ice-water injection, favors the theory that the stimulation of peristalsis may exercise a favorable influence. However uncertain the measure may be, I would advise the trial in all cases of ice-water enemata, applied every hour or two; and still more profuse irrigations of ice-water, even though it be immediately ejected again. By the mechanical pressure of the fluid, this plan may effect the same object which is sought by other mechanical methods of reduction, viz., blowing air into the intestines with bellows, and the introduction of a whalebone bougie, armed with a sponge, with which the attempt is made to thrust the invagination felt in the rectum upward. A small number of cases have been cured by these methods, and no opposition can be made to their cautious employment when they are discontinued forthwith if they do not soon prove successful. The danger lies in the fact that we can never foretell whether the intussusception is still reducible or has become fixed from adhesion of both serous layers (of the intussusception and its so-called sheath). In this event, every forcible attempt at reposition may cause rupture of the adhesions and the serous membrane itself, the effects of which cannot be calculated. I would therefore especially adopt these mechanical methods of reposition (blowing in air, injection of water, introduction of a bougie), if we are able to push upward, by their means, an intussusception which can be felt in the rectum, even though it immediately fall back into its place again. The attempt may then be repeated a number of times without any danger, and it may finally lead to complete recovery. When this cannot be done, it seems to me more advisable to abstain from every forcible measure, and to wait for spontaneous separation, which, as we have seen in

our second case, may occur within a few days. In the larger number of cases in which the intussusception cannot be felt in the rectum, but must be diagnosed from the other symptoms, we may also try the introduction of air, and if this proves unsuccessful, confine ourselves to irrigations with large quantities of ice-water, the application of an ice-bag to the abdomen, and the alleviation of pain by small doses of opium or morphine (P. 10). The diet should consist of wine and iced milk, given in spoonfuls. In a few cases it is said that holding the patient up by the legs, or massage of the abdomen, especially in the situation where the tumor is felt, has produced reduction of the latter. In desperate cases, laparotomy, followed by disentanglement of the intussusception or the formation of an artificial anus, has proven successful in a few exceptional cases; but, after finding the invagination, it is almost always impossible to withdraw it from the lower portion of the intestine.[1]

XI. Rectal Polypi.

Apart from melæna neonatorum (page 32) and invagination, we found that a discharge of blood from the intestinal canal occurs in colitis and dysentery with its sequelæ. Other causes of intestinal hemorrhage, especially gastric ulcers (page 197), intestinal cancer, hemorrhoids, occur very rarely in childhood. Only in a couple of cases have I observed hemorrhoids (which did not bleed) in children. Occasionally we meet with cases of intestinal hemorrhage which remain a diagnostic riddle despite the most careful examination. In another series of cases, which occur quite often from the age of three to twelve years, the hemorrhage is due to the formation of polypi in the rectum. Polypoid proliferations from the size of a pea to that of a bean are not infrequently found, during childhood, on the mucous membrane of the large intestine. I have occasionally observed multiple growths in autopsies upon children who died of various diseases, and presented no striking symptoms on the part of the intestinal tract. But the rectum, especially the portion situated immediately above the internal sphincter, is the most frequent site of the polypi, which are produced by proliferation of the mucous membrane, and the mucous glands contained in it (adenoma) may grow to the size of a cherry or even of a small plum, and finally remain adherent to their base by a more or less long and narrow pedicle.

The first, and, at the same time, only symptom of rectal polypi is a hemorrhage, which rarely takes place spontaneously, but almost always occurs with a passage or immediately afterward, the blood escaping from the anus either in drops or in somewhat larger quantity. The blood is never mingled intimately with the fæces, but lies upon the surface, so that it can only have escaped at the moment of defecation. In every case of this sort you should immediately suspect a rectal polypus. The affection sometimes exists for many months before the parents consult a physician, and I have known cases in little girls in which the disease was regarded as premature menstruation. Upon careful questioning we will often ascertain that "something protrudes" from the anus after every passage, and if older children are asked to bear down during defecation, we can sometimes observe a dark red, round tumor, with a slightly bleeding surface, from the size of a bean to a cherry, or even larger, projecting from

[1] Bell, Marsh u. Hutchinson: Jahrb. f. Kinderheilk., IX., S. 427 u. ff.

the anus, and receding after defecation. But this is always accidental. I have very often been unable to detect the tumor, although the children stated that something emerged from the anus during defecation, and then returned. Digital examination of the rectum is therefore always necessary, and is best performed when the child is in the knee-and-elbow position. But if the polypus is situated somewhat above the internal sphincter, it may slip into the dilatation of the rectum, which is situated in this locality, and may thus escape the finger.

A girl, aged six, said by the mother to suffer from prolapse of the rectum; but the frequent hemorrhages favored the idea of the presence of a polypus. Careful examination failed to reveal the growth, and I dismissed the child. In a few minutes the mother returned, and showed me a bluish red polypus, as large as a plum, which lay outside of the anus, and was connected with the rectal mucous membrane by a narrow pedicle. I grasped the latter between two fingers in order to cut it off; but, before this could be done, the child made a vigorous movement, the pedicle ruptured, and the polypus remained in my hand. From this time on the hemorrhages ceased entirely.

It is evident from this case that polypi with long and narrow pedicles in the rectum may be torn off during the evacuation of hard fæcal masses, and, in fact, this spontaneous elimination of the tumor is not very rare. The treatment is purely operative. If the polypus protrudes from the anus, it is grasped with a pair of forceps, drawn down still farther, and the pedicle cut with scissors. The operation is more difficult when the polypus is not situated outside the anus, and must therefore be grasped within the rectum. Cutting the pedicle is preferable to ligature, as it is more rapid and devoid of danger.

XII. Prolapse of the Rectum.

Prolapsus ani is much more frequent in childhood than polypus of the rectum. It is most common during the first year, though cases are met with quite often from the age of two to six years.

Prolapse constitutes an invagination of the rectum *en miniature*, which is pressed out of the anus. We must take into consideration that above the lowermost muscular part of the rectum is found a wider and more flaccid portion, which passes into a part which is provided with a strong circular, muscular coat situated still farther upward. The latter, by its contracture, can force the middle, flaccid portion into the lower part and extrude it from the anus in the form of a shining red, tense swelling, which surrounds the anus like a ring. Occasionally the folds of the mucous membrane alone project, but these form a small prolapse. The length of the prolapse varies greatly, but averages from three to four centimetres. The surface often bleeds, and is occasionally covered with ascarides. Prolapse only occurs, as a rule, during defecation, after which it returns spontaneously into the rectum. Larger ones not infrequently remain outside the anus, and the mothers are sometimes unable to return them. Every physician knows that this may be extremely difficult to do, because the child, by bearing down, extrudes the gut as soon as the finger has been removed from the rectum.

In very many cases, especially in young children, the cause of the prolapse cannot be determined. Constipation which has given rise to repeated straining occasionally, and obstinate diarrhœa or dysentery much more frequently act as the cause, and, in addition, the catarrhal swelling

of the mucous membrane and the increased flaccidity of the middle portion of the rectum may be regarded as the predisposing factor. Excessive abdominal compression in whooping-cough or violent crying, may also give rise to prolapsus ani, though I have rarely seen this happen. Whether atony of the internal sphincter plays a part in the pathogenesis of prolapse is undecided; this view is favored by the fact that the finger readily enters the rectum, in the majority of these cases, without encountering the vigorous opposition of the sphincter, which is usually felt, and also by the therapeutical experience that remedies which stimulate contractions, act best in this affection.

The effect of repeated straining upon the production of prolapsus ani is also shown by its occurrence in connection with calculus of the bladder. Whenever prolapsus occurs after the period of second dentition, particularly in boys, you should always think of stone in the bladder, especially if other suspicious symptoms—incontinence, tenesmus during micturition, unusual length of penis—are present. In two cases of this sort, in which the gut protruded with every passage and during micturition, an examination showed the presence of a stone in the bladder. The more often the rectum prolapses, the more is the sphincter distended and enfeebled, and thus atony must, on the other hand, favor the occurrence of the prolapse. It is also said that ascarides in the rectum may give rise to prolapse, inasmuch as the irritation produced by them gives rise to severe reflex straining; I am still in doubt with regard to this point.

This affection is always protracted, and may last many months or even years. Temporary improvement and unexpected relapses are not infrequent, but recovery occurs occasionally with surprising rapidity. Thus, I remember a few cases in which a single reposition, after which the finger was left in the rectum for a few minutes, sufficed to relieve the prolapse permanently. But such cases are exceptional and can be explained with difficulty. As a rule, reposition and the use of the tampon are merely palliative measures which do not prevent the return of the prolapse. I will mention that in replacing the prolapse (performed best in the knee-and-elbow position) the central portion, which was last protruded, should be pushed back first, two fingers of the right hand, which is covered with an oiled piece of linen, being placed in the central opening of the prolapse and gently pushed upward. If the children struggle violently, it may be necessary to use chloroform and then introduce a tampon in order to prevent the immediate recurrence of the prolapse.

To secure this object, those remedies to which the effect of causing contraction of the muscular fibres of rectum is attributed are recommended, especially the extract of nux vomica and strychnia. I have employed the former very often (P. 37), but with such uncertain success that I am gradually using it less and less. According to my experience strychnia is also a by no means reliable remedy. I have had much better results from the subcutaneous injection of ergotin in the perineum and in the immediate neighborhood of the anus. In children, one and one-half to three years old, I injected daily 0.02–0.1 of ergotin at once (P. 38), and distinct improvement usually occurred in a week. The prolapse occurred more rarely, remained absent on some days and disappeared entirely in two to three weeks. Although I have repeatedly observed this favorable effect of ergotin, I do not regard it by any means as an absolutely certain remedy, and you will often meet with cases which demand other methods of treatment. I have seen, at the most, a temporary effect from the daily introduction of ice into the rectum, and not much

more from enemata of a solution of tannin, alum, or other astringent decoction (rhatany, oak-bark). When all these methods fail, surgical treatment alone remains, either excision of some folds of skin around the anus (Dupuytren), or punctate cauterization with a fine actual cautery in the immediate vicinity of the anus (P. Guersant), the action of which is intended to penetrate to the external sphincter.

In every method of treatment we should see to it that the children discontinue violent straining. The prolapsus occasionally remains absent if the fæces are passed while lying in bed, as the abdominal pressure acts much less vigorously, and I therefore advise that the children be not allowed to sit on the chamber in the usual manner, with the feet braced against the floor, but that the vessel be placed on a firm chair or table, and the children be held firmly upon it, with the feet hanging loosely. If the patient is constipated, the passages should be softened by purgatives; while, in the event of a protracted diarrhœa or dysentery, the successful treatment of these conditions may suffice to relieve the prolapse.

XIII. Intestinal Entozoa.

Helminthiasis is now justly confined to an extremely small domain. But we should not go too far in this direction, and consider worms under all circumstances as perfectly harmless tenants of the economy. Although comparatively rarely, some cases nevertheless occur in which their influence in the development of certain symptoms is undeniable.

I will not enter into the natural history of intestinal worms, but refer you to the classical works of Davaine [1] and Leuckart.[2] Only three groups of entozoa concern us, viz., oxyuris vermicularis, ascaris lumbricoides, and tænia.

1. Oxyuris vermicularis (pin-worm) is a white, spindle-shaped worm, about nine or ten millimetres long and one-half millimetre broad, especially narrowed at the posterior extremity, and which inhabits the colon, particularly the rectum, in large masses, and derives its nourishment from the fæces. The male worm is found much more rarely than the female (about in the proportion of 1 : 9), is much smaller (two and one-half to three millimetres long) and has a spiral-shaped twisted tail. The oxyures, also called ascarides, are either evacuated with fæces, upon which numerous moving worms, looking like short white threads, are found, or they migrate from the anus at night, independently of defecation, and cause, by their movements in its neighborhood, severe itching, which gives rise to scratching. Upon careful examination we will often find, at this time, a number of moving ascarides in the neighborhood of the anus. In many cases, pain in this region develops nightly, especially when the child is falling asleep. I remember one boy who would throw himself upon the floor, scream, and press the anus forcibly against the floor in order to procure relief. The almost periodical repetition of these scenes every night, which insufficient observation may interpret as convulsive, has induced many an inexperienced physician to make a false diagnosis of intermittent fever. The anal pruritus occasionally occurs during the day or in the middle of the night, and the worms are then al-

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

most always found outside the anus. As they can move upon the moist surface of the mucous membranes alone, and soon become motionless upon the dry cutis, the migration of ascarides is scarcely credible; and though it cannot be denied that ascarides are occasionally found in the vulva of little girls, and may here give rise, by their irritation, to congestion, secretion of mucus, and a tendency to onanism, this occurs much more rarely than is commonly supposed. I would ask you to consider whether, in such cases, a direct transportation of the worms or their ova, by means of the scratching finger, is not more probable than their independent migration into the vulva. This is also true of the not infrequent cases in which not only several children of the same family suffer from ascarides, but also the mother who sleeps in the same bed with one of her children. It has been shown that the majority of ascarides which migrate from the anus, or are discharged in large numbers with the evacuations, are females, and that the fæces of these children always contain numbers of mature ova. By the scratching finger, or by the drying of the fæces—especially in small, narrow rooms, the ova may readily enter the stomach of other persons, where their envelope is dissolved by the gastric juice and the embryo set free. This explains the great obstinacy of these worms, which cannot be destroyed in many families for years, and also their frequency in dirty idiots, whose colon occasionally shows a fur-like coating of ascarides (Vix). The transportation of mature ova or young embryos also explains the rare cases in which the worms are observed in other places far removed from the colon—for example, in a moist eczema of the inguinal folds (Michelson [1]) or even in the buccal cavity (Seligsohn [2]).

2. The round worm (ascaris lumbricoides), a cylindrical annelide of brownish or reddish gray color, and considerable size. The females are nearly 400 mm. long, the males rarely more than 250 mm. (greatest thickness 5.5 and 3.2 mm.). The body is narrowed at both ends, especially anteriorly; the opening of the mouth is surrounded by three lips, provided with extremely fine teeth; the tail end is short and conical. The posterior part of the male is curved toward the belly, like a hook, and the wedge-shaped penis not infrequently projects from the swollen opening of the cloaca. The vulva is situated immediately behind the anterior third of the body or more toward the middle.

The small intestines of the child sometimes contain a scarcely credible number of round worms. I remember one case in which vesselfuls of round worms were passed for a number of days in succession, although no symptoms of such an accumulation had been present. Under these circumstances, the worms may be collected into large balls which cause obstruction of the intestinal lumen and symptoms of ileus (page 211), and they may even form a tumor which is distinctly felt from the outside.[3] In the large majority of cases, however, the worms are present in much smaller numbers. If, as the above-mentioned case shows, an enormous number of worms may be present without any striking symptoms, this will occur still more frequently if only a few are found in the intestines, and I know of only one positive symptom of their presence, viz., their evacuation. At a certain period of its development the worm makes preparations to leave the body, and then migrates from the small intestines into the colon or upward into the duodenum and stomach. In the first event it is discharged with the passage, either dead or alive, or may crawl

[1] Berl. klin. Wochenschr., No. 33. 1877. [2] Ibid., No. 40. 1878.
[3] Jahrb. f. Kinderkrankh., X., S. 298. 1876.

from the anus independently of defecation; in the second event, it is either removed by vomiting or passes through the œsophagus into the pharynx, or even farther. Cases are not rare in which the worms have crawled from out the mouth of the child during sleep. This elimination of lumbrici is the only positive sign of their presence. All other symptoms, viz., pale complexion, dark rings around the eyes, fœtor oris, itching at the tip of the nose, and frequent colic, will at the most arouse suspicion and justify the use of anthelmintic remedies.

We will now refer to the often ventilated question, whether the round worms exercise a local influence upon the portion of intestines inhabited by them. *A priori* this should not be denied, since the presence of numerous ascarides may cause catarrhal irritation of the rectum, and the construction of the buccal opening (the lips provided with teeth) favor the possibility of such an action. Indeed, congestion of the mucous membrane of the small intestine, and even diarrhœa, are said to be produced by the irritation of the worms, but not a single positive case has come under my own notice. We should always remember that in an ordinary catarrhal diarrhœa the round worms which may happen to be present in the intestine may be evacuated, and this is sometimes observed in dysentery and typhoid fever. It is even considered possible that the round worm, by strongly pressing the head against the mucous membrane, may separate the fibres of the walls of the intestines, and pass through the opening into the abdominal cavity. I believe, however, that in all cases in which round worms are present in the peritoneal cavity, they have crawled through an intestinal ulceration which was previously present, and this may communicate with an encapsulated peritonitic process, or may still be covered by the thin serous coat which is ruptured by the worm. Least of all can I imagine that the worms which have entered the peritoneal cavity are capable of producing circumscribed peritonitis with perforation of pus externally. I have never observed such a process in the abdomen, but in two cases of chronic tubercular peritonitis, spontaneous rupture occurred through the umbilicus, or next to it, and after the discharge of feculent pus, one or two round worms also escaped through the opening. I therefore believe that the worms make use of an opening accidentally present in the intestines, either a follicular or tubercular ulcer around which circumscribed peritonitis has developed, for their emigration from the intestine, and then pass immediately into the abscess, toward the formation of which they have in nowise contributed. This view is also favored by the experience that the worms have a tendency to pass through narrow openings. When a child, who is known to suffer from round worms, develops chronic incurable icterus or the symptoms of hepatic abscess, we must think of the possibility of occlusion of the ductus choledochus or hepaticus by a worm, or of an abscess-formation of the liver, caused by the irritation of young lumbrici which have entered the finer biliary passages. Whether the emigration of the worms into the stomach will produce, apart from nausea and vomiting, any serious irritative conditions of the stomach, appears to me as doubtful as their supposed effect on the intestinal mucous membrane. In a boy, aged four, who was suffering from a febrile gastric disorder, I observed emesis not only of a live round worm, but also of some clotted blood; but I have also found it in the vomit of children who have never passed round worms, and in a girl, aged six, who complained of severe colic, eight worms were evacuated from the bowels, and nine were vomited within a week, although no blood could be discovered in the vomited matter. If the worm mi-

grates into the pharynx, it may then pass into the larynx, nares, lachrymal duct, or Eustachian tube.

Thus far we stand upon the firm foundation of fact, but we forsake this position in deciding the question whether worms, either lumbricoid, ascarides, or tænia, can produce certain symptoms on the part of the nervous system. All possible convulsive diseases have been attributed to the reflex irritation of these worms, but personally I have hardly ever been able to detect such a connection, and, least of all, in chorea minor and magna, eclampsia, epilepsy, and related conditions. I will acknowledge, however, that the experience of any one person is insufficient to decide the question, and will therefore not deny the possibility of such a relation, especially as I have repeatedly observed milder nervous symptoms in connection with worms. I include among these, apart from the frequent itching of the nose, unusual dilatation of the pupils, severe colic, slight rigors, and even chills, which were not followed by heat or sweating, headache, vertigo, and, in one case, ecstatic seizures:

A boy, aged twelve, had suffered for some days from severe pains in the region of the descending colon, which was tender on pressure; moderate fever. On the fourth day, subsidence of the pains, but nocturnal ecstatic symptoms occurred, consisting of delirium, great restlessness, tossing about, general tremor, which continued until midnight; complete euphoria during the day. On the tenth day, evacuation of two unusually large, living round worms, after which all the symptoms at once ceased.

Despite my scepticism I cannot, in this case, deny the intimate connection of the neurosis with the irritation of the worms, and must therefore acknowledge the possibility of the reflex production of convulsions in this manner. There is therefore no objection to the use of anthelmintic remedies in such neuroses if we carefully search, at the same time, for other causes of the disease. If no worms are evacuated, we may make a microscopical examination of the fæces, which usually contain a greater or less number of ova in almost all cases of helminthiasis.

The treatment consists chiefly in the administration of santonin. We may give this in doses of 0.05 to 0.1, three or four times a day, in powder or lozenges, for two or three days in succession, and on the third day administer a purgative (castor-oil, inf. sennæ comp.) in order to evacuate the worms which have been rendered motionless by the santonin. Santonin has the advantage over the formerly used semina cinæ, which contain it, that it is more readily taken by the children, but I think that the latter produce better effects. Santonin may also be given in combination with other purgatives—for example, calomel (0.05 to 0.1), or with castor-oil (50.0, with santonin 0.2, in teaspoonful doses). That santonin colors the urine yellow, and occasionally produces yellow vision (xanthopsia), is well known. But as other toxic symptoms, such as urticaria, vomiting, even epileptiform seizures (especially after larger doses of 0.2 to 0.3), have been observed, it appears advisable, for this reason, to combine the remedy with a purgative, in order to remove it from the body as rapidly as possible. In the treatment of round worms, santonin is used internally alone; when ascarides are present, we must associate it with injections into the rectum, either of a solution of corrosive sublimate (0.015 to 100 aq. dest.) or inf. sem. cinæ (10.0:20.0 to 100.0). These injections should be made at night, and retained in the rectum as long as possible. The often used enemata of an infusion of garlic or sapo medicatus have often failed me, as all other remedies, both external and internal, not infrequently do. I have had no personal experience with other forms of enemata (lime-

water; quinine, 0.3 to 1.0 to an enema; terebinth., gum. arab., āā 8.0, inf. chamomile, 120.0). If very severe pruritus is present, I order a couple of inunctions daily of ung. hydrarg. ciner. in the region of the anus, or the introduction of suppositories in the anus (ung. ciner. 2, with ol. cacao 4, or sapo domest. pulv. 3). I may remark, in passing, that pruritus ani occasionally occurs in children independently of worms, but is then present in the day rather than at night. I observed this condition in a healthy boy, aged eleven, who was suffering from obstinate constipation. A couple of wineglassfuls of Ofen bitter-water relieved both the constipation and the pruritus.

3. Tænia (tape-worm). Although much less frequent than round worms and ascarides, tænia are observed almost as frequently in children as in adults. The thirty-three cases, which I have previously reported,[1] have since increased threefold, and not a month passes in which a couple of children suffering from tænia are not present at my polyclinic. The age of the patients varies greatly. Although I have observed no case of tænia in the fœtus,[2] or in a child five days old,[3] I have repeatedly seen pieces of tape-worm discharged in two children who were only a year old. If we take into consideration the well-known experiments with regard to the metamorphosis of the cysticercus into tænia, we can scarcely conceive how a tape-worm can develop in the fœtus or new-born, since we cannot accept the view of an infection of the fœtus from the blood of the mother. In all cases of tænia observed by me in nurslings and children during the second year, it was found that the patients ate raw meat or sausage in addition to milk.

The majority of the children affected with tænia solium or mediocanellata, who came under my treatment, presented no morbid symptoms; only the occasional discharge of single, movable mature sections (the so-called proglottides) or of larger pieces attracted the attention of the mothers. Their evacuation occurs either with the fæces or independently of them, so that they were found in the children's trousers or in bed. In comparatively rare cases, complaint was made of pains in the stomach or abdomen, nausea, accumulation of water in the mouth, occasionally also creeping or numb sensations in the legs, and even difficulty in walking.

I could never, with any degree of certainty, attribute more serious symptoms, such as chorea, epilepsy, catalepsy, etc., to tænia, so that I do not regard them as more injurious than round worms and ascarides to the health of the children. Moreover, I have repeatedly observed that these symptoms were present at the same time with the tape-worm without exercising any worse effects than usual. As in adults, so also in children: the ingestion of herring often resulted in the spontaneous discharge of proglottides or larger pieces, and about this time, ill-humor, colic and disturbed sleep were often observed, especially in small children. Several times, also, I have seen pieces of tænia pass in the fæces during the course of acute or chronic diarrhœa, and once during diarrhœa of typhoid fever. In two cases the tænia, which had been partially protruded from the anus during defecation, were torn by the mothers in the attempt at complete extraction. In such cases all traction of the protruded parts should be avoided, they should be held outside of the anus by means of adhesive

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[1] Beitr. z. Kinderheilk., S. 133; ibid. N. F., S. 327.
[2] Barrier: Maladies des Enfants, II. p. 98.
[3] Oesterr. Jahrb., I. Anal. S. 103, 1873. Jahrb. f. Kinderheilk., V., S. 444; Hirsch u. Virchow, Jahresber. f. 1872, II., S. 701.

plaster, and the endeavor made to evacuate the worms entirely by means of purgatives and injections. I cannot decide from my own experience whether the injection of vapor of chloroform into the rectum, under such circumstances, will facilitate the discharge of the remainder.

I place kousso in the front rank of remedies for tape-worm in children, as it has always seemed to me to act most effectively. According to the age, I give every morning 8.0 to 10.0 in coffee or milk, in two portions, at an interval of half an hour; even if the second dose produced nausea or vomiting, this did not interfere with the effect in many cases. If a spoonful of castor-oil is given an hour later, the thin passages which occur during the day, often, though not always, contain large masses of tænia or the entire chain with its adhesive apparatus, usually termed "worm" and "head." In many cases the "head" could not be found, but only the narrow part adjacent to it, and I then usually repeated the treatment after the lapse of two days. It is advisable to clear the intestines by means of castor-oil on the day before the administration of the remedy, and to let the patients eat herring on the previous night; but on the day of treatment, after purgation has been secured, to give an enema of cold water at least every two hours, in order to give the tænia, which have been stunned by the kousso, no time to adhere again to the walls of the intestines. As a rule, the effects of pomegranate root and filix mas were less certain. I administered them in the following combination: decoct. cort. rad. granat. (30.0) 180, ext. filic. mar. æth. 2.5 to 3.0, syr. cort. aur. 20.0. M. S.—To be taken half-hourly, in three portions. At the end of an hour a spoonful of castor-oil should be given, and, after this has acted, the enemata of water recommended above should be given every two hours.

XIV. Acute and Chronic Peritonitis.

Acute peritonitis is observed much more rarely in children than in adults, and most frequently, in the former, in connection with pyæmic and septic processes in the new-born. The symptoms, tympanites, tension, tenderness of the abdomen and vomiting, are then combined in such a manner with the general symptoms of pyæmia, or so masked by the latter, that a distinct clinical history of the disease is not produced. In older children I observed acute peritonitis, especially as the result of perityphlitis. Whether this inflammation is due to excessive distention of the cæcum by masses of fæces, or to the irritation of a fæcal concretion in the vermiform appendix, it always shows the same symptoms as in later life, viz.: pains which are at first confined to the region of the cæcum, but are apt to spread over a large part of the peritoneum, the frequent formation of a tumor by the exudation, which not infrequently terminates in suppuration, the repeated relapses, etc. I will therefore confine myself to a few remarks on the treatment of this condition with opium. My experience is decidedly in favor of this treatment in children as in adults. I keep the intestines perfectly quiet, and give castor-oil or calomel only when large accumulation of fæces in the cæcum has been determined by the existence of prolonged constipation or by palpation. Otherwise I would advise that you abstain, at first, from the use of purgatives, and apply four to eight leeches (without after-hemorrhage), according to age, when the cæcal region is very tender, and then place an ice-bag constantly over this region. Internally I give an emulsio oleosa (100.0) with extr. opii

aquos. 0.1 to 0.2, and syr. emuls. 20.0; a teaspoonful every two hours, until the spontaneous pains cease and the tenderness on pressure is ameliorated. As soon as this is done, defecation usually occurs spontaneously or after the use of an enema, or the administration of a spoonful of castor-oil. When this treatment was begun early enough, recovery ensued in almost all cases, and if a swelling had been formed by the exudation, its transition into suppuration was prevented, even in children who, in the course of a few years, had been repeatedly admitted to the hospital on account of relapses.

The exudation, which occurs in these cases in the vicinity of the cæcum, forms a hard mass, which can be distinctly felt and determined by the dulness on percussion, and which may even extend across the median line and upward to the level of the umbilicus. If absorption does not occur under the continued use of the ice-bag, but the tumor and its tenderness increase, are associated with constant fever, and present nocturnal exacerbations, warm poultices should be applied night and day, in order to aid suppuration. Occasionally spontaneous rupture of the abscess then occurs externally into the rectum, bladder, or vagina; but this termination is often delayed and the continued fever of suppuration threatens to exhaust the powers. We must then perform artificial opening of the abscess, which has lost a large part of its danger on account of the antiseptic method. Such peritoneal abscesses may also develop independently of perityphlitis in other parts of the abdomen, without any distinct cause, but occasionally as the result of traumatism.

A girl, aged ten, who came to the dispensary on November 11, 1879, had been seriously injured in August by a large dog, who threw her down and trampled on her abdomen. An acute peritonitis developed after this, which reached its end in the middle of September, with the perforation of pus through the umbilicus. In November a large red wound covered with granulations, from which a little pus flowed, was still present at the site of the former umbilicus.

M. L——, aged ten, first examined by me on December 18, 1876. For two weeks symptoms of acute peritonitis, which had developed without any ascertainable cause in the left iliac region (two applications of leeches, ice-bag). I found a diffuse, very sensitive tumor, dull on percussion, which occupied the lower part of the left half of the abdomen to a little above the level of the umbilicus, while the right side was entirely free. Pain during defecation and micturition, remittent fever, great weakness. In the last few days very violent attacks of colic, associated with loud cries and sunken features, with perfectly free intervals. Treatment: warm poultices to the abdomen, emulsio oleosa with extr. op i December 20th, repeated evacuation of mucus and purulent fluid from the rectum, and, during the same night, four to five thin, very profuse, purulent stools of the color of milk and coffee. December 21st, tumor and pains entirely disappeared, continuance of the purulent evacuations, mixed with lumps of fæces. Complete recovery within a few days. As I learned at a later period, two slighter relapses occurred in the same locality in the course of the next few years, which did not terminate in suppuration, but aroused the suspicion that some unknown local cause of the peritonitis was still present.

In these cases the peritoneal abscesses were discharged through the umbilicus and rectum. If we take into consideration that the umbilicus is the most yielding part of the abdominal walls, that the fascia is here absent in great part and the peritoneal cavity merely covered by the tegumentary cicatrix, fat, and the peritoneum, we can understand that in all distentions of the abdomen, whether from pregnancy, firm tumors, or ascites, attenuation and hernial protrusion of the umbilicus occur so readily, and still more so during the age of childhood, when the umbilicus is even less resisting than in adult life. Although it is stated by some au-

thors[1] that the majority of cases of perforation of the umbilicus by pus were not due to peritonitis, but that the suppuration almost always occurs outside the peritoneum in the so-called subserous tissue, and I must acknowledge, from my own experience, the occurrence of these abscesses in the abdominal walls, which usually have a traumatic origin, and may readily be regarded as peritonitis, I nevertheless believe that these writers go too far in their statements. I will soon have the opportunity of communicating a few cases in which, in chronic peritonitis which was demonstrated on autopsy, perforation of pus, and once of ascitic fluid, occurred through the umbilicus, and I see no reason why such an event should not be produced by a more or less acute collection of pus in the peritoneal cavity. Acute peritonitis from perforation of one of the abdominal organs, apart from ulceration of the vermiform appendix referred to above, occurs only exceptionally in children, on account of the great rarity at this period of round gastric ulcers and other diseases of the abdominal organs leading to perforation. Even typhoid fever, as we shall see at a later period, very rarely gives rise to it. But I have, on a couple of occasions, had the opportunity of observing diffuse purulent nephritis in the course of scarlet fever, especially of the secondary nephritis, to which I shall again refer in the discussion of this disease.

Chronic peritonitis, with the exception of the very frequent tubercular variety, is a very rare affection. Even the adhesions of the abdominal organs observed so often in adults, especially of the internal genitalia in females, are exceptional in childhood, while the cases in which a non-tubercular chronic peritonitis presents a well-defined clinical history, are so rare that their occurrence has been denied by some authors. But this view is unfounded, as is shown by the following history.[2]

Anna S——, four years of age, admitted November 14, 1873, said to have been well until a week ago (?). Since then distention of the abdomen has been noticed. Examination showed marked ascites and distinct fluctuation; no pain or tenderness. Liver dulness extends to fifth rib. Respiration, 28-40, labored. Dulness, and feeble respiratory murmur to right side of scapula and inferiorly. Slight œdema of feet, urine normal, no fever. November 16th, 3.900 ccm. of a greenish albuminous fluid removed from abdomen by tapping; it contained numerous pus-cells, fibrin, and some flakes which, under the microscope, showed a fibrous mesh-work filled with cells. Palpation showed that the lower edge of the liver projected downward from 3–5 ctm. Fluid reaccumulated, and tapping again performed on November 24th. Condition unchanged until December 13th: evening temperature, 38.6°; pulse, 96–140; general condition growing worse, repeated vomiting, occasional diarrhœa. The ascites diminished, and on December 13th, partly nodular, partly band-like hard bodies could be felt in the region of the umbilicus, coalescing into a larger tumor near the boundary of the hypogastrium; friction-fremitus distinctly felt occasionally. Condition almost unchanged until December 21st, when fatal collapse occurred. The following notes were taken on the last day: abdomen soft, readily compressible; lower border of liver felt 1–1½ ctm. below the edge of ribs; to the right of the linea alba, and extending from border of liver into right iliac fossa, is a tumor about as broad as the hand, projecting distinctly from the abdomen, and composed of a number of sausage shaped enlargements.

Autopsy.—About 500.0 cloudy fluid in abdominal cavity. The visceral and parietal layers of the peritoneum present quite broad and long bands of recent fibrinous exudation; the loops of small intestines firmly connected with one another by firm peritoneal adhesions. The serous coat of the intestines enormously thickened, friable, cloudy, and in very many places has become organized, in conjunction with the

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[1] Vaussy: Des Phlegmons Sous-Péritonéaux de la Paroi Abdominale Antérieure. Paris, 1875. Gauderon: De la Péritonite Idiopathique Aiguë des Enfants. Paris, 1876.

[2] Berl. Klin. Wochenschr., No. 10. 1874.

subserous tissue and the overlying exudation, into a bluish-white, semi-translucent tissue, which creaks under the knife, and is ¼–1 ctm. in thickness. Greater and lesser omentum very much retracted. The entire intestinal canal shortened, the mucous membrane pale, liver somewhat enlarged, with a bloody deposit upon its convexity (perihepatitis hemorrhagica). Pleura on right side.

This is a case of very extensive chronic peritonitis without a trace of tuberculosis ; as was ascertained at a later period, the affection must be attributed to traumatism, viz., to a kick in the hepatic region, received a few weeks previously. The hemorrhagic inflammation of the hepatic peritoneum, which was still present at the autopsy, probably opened the scene, and from this situation the process gradually spread to the entire peritoneum, and also to the right pleura. It therefore follows that injury to the abdomen may result not only in acute, but also in chronic inflammation of the peritoneum, with strong adhesions of the loops of intestines to one another, and with considerable serous exudation, and this may occur so insidiously and latently that attention is first attracted by the increasing ascites. I also call your attention to the extremely slight tenderness of the abdomen and the usually normal passages from the bowels, despite the intimate adhesion of the loops of intestines to one another, because this is also observed in the tubercular variety.

Special attention should be paid in this case to the very considerable nodular thickening of the walls of the intestines, which, during life, had assumed a tumor form, and had led me to make a diagnosis of sarcoma of the abdomen, as the microscopical examination after the first puncture, and the friction-fremitus also favored this diagnosis. We can, therefore, draw the deduction from this important case, that a nodular thickening of the walls of the intestines may reach such a height in the course of chronic peritonitis as to produce a deceptive feel like that due to a tumor (especially sarcoma), and that, furthermore, the microscopic examination of the ascitic fluid in such cases, may show an alveolar structure rendering possible the assumption of broken-off shreds of sarcoma, though consisting, in reality, of pus-ceils embedded in flakes of fibrin.

This is the only case of chronic non-tubercular peritonitis in children which could be corroborated by autopsy, but I have seen several cases in private practice which presented all the symptoms of beginning or even completely developed chronic tubercular peritonitis, but which, to my surprise, were entirely cured. Some of the children in question had been previously healthy, others were suffering from osteomyelitis, or other scrofulous symptoms. Should these cases be regarded as cured tubercular or simple chronic peritonitis ? I cannot answer this question positively, although I incline to the latter view.[1] Indeed, there is no reason why the peritoneum, like the pleura, should not be the site of a chronic inflammation, with serous exudation independently of tuberculosis.

XV. Tuberculosis of the Abdominal Organs.

In discussing meningeal and pulmonary tuberculosis, I called your attention to the frequent occurrence of miliary tubercles or cheesy products in the abdominal organs. You often find tubercles in the spleen and liver, occasionally in enormous numbers, some large, some small, so that they can hardly be recognized by the naked eye. Tubercles of the

[1] Vide a similar case reported by Stiller, Deutsch. Archiv, XVI., S. 412. 1875.

spleen and liver may also attain the size of peas, and the latter variety often show in the centre a cavity filled with greenish yellow fluid—the lumen of a bile-duct which has been surrounded by tubercular masses. The peritoneum, greater omentum, the serous coat of the liver and spleen, diaphragm, intestines, etc., constitute with almost equal frequency the site of numerous miliary tubercles, which are also found in the kidneys, most rarely in the internal genitalia of little girls. The mesenteric and other abdominal lymphatic glands are usually more or less swollen, and converted, either partially or entirely, into a cheesy mass. All these changes, however, constitute accidental post-mortem appearances, which may be suspected in tuberculous children, but cannot be diagnosed with certainty.

A diagnosis can be made, on the other hand, in those cases which are characterized by the predominant or exclusive development of tubercles in the abdominal organs, while the contents of the thoracic and cranial cavities are affected by tuberculosis either to a trifling extent or only at the end. Although, in all the cases observed by me, tubercles of the liver and spleen escaped positive recognition during life, and cheesy degeneration of the mesenteric glands could only be exceptionally determined with certainty, the diagnosis of peritoneal tubercles is, as we shall soon see, attended with much less difficulty. Before entering upon this subject more in detail, I will say a few words with regard to the affection of the mesenteric glands just referred to.

The period in which degeneration of these glands played such an important part that almost all atrophic conditions were attributed to their enlargement and "stoppage" interfering with the flow of chyle, has long passed. "Atrophia meseraica" is found in most cases to be a more or less general tuberculosis, which is concentrated chiefly in the abdomen, and in which the mesenteric glands have been affected secondarily to the peritoneum or intestinal mucous membrane. They may also become hyperplastic, and, under unfavorable conditions, undergo cheesy degeneration in otherwise healthy children, who suffer from chronic or often repeated intestinal catarrh, as the bronchial glands do after chronic bronchitis or whooping-cough ; but the disease of the mesenteric glands is much more often caused by tuberculosis of the intestines and peritoneum, which produces infection of the glands through the agency of the lymphatics. I have repeatedly traced through the mesentery lymphatics which contained miliary tubercles and which started from tuberculous portions of the intestines. In the majority of cases the enlargement and hardness of the mesenteric glands attains but a moderate intensity, and cannot be recognized by abdominal palpation ; even larger swellings cannot sometimes be felt, this being prevented by the distention of the intestines with gas, and the consequent abdominal tension. For this reason I was unable, for example, to detect a tumor in a girl aged five, who was suffering from chronic tubercular peritonitis, although the autopsy showed a tumor as large as a child's head, composed of an agglomeration of tubercular mesenteric glands. But when the gaseous distention of the intestines is absent or subsides temporarily, we are often able to feel the enlarged glands as movable rounded nodules of variable size, although we must always in such cases think of the possibility of deception by fæcal masses.

A characteristic clinical history is alone given by tuberculosis of the peritoneum, and then only when not alone miliary tuberculosis is present, but, as in tuberculosis of the pia mater, an inflammation is superadded.

In the large majority of cases this pursues an insidious, chronic course, but we must always be prepared to meet a more rapid development, under which circumstances the history of an acute peritonitis may be closely simulated when the previous history is defective or entirely wanting, especially in hospital practice. And indeed, we then have to deal with such an attack which is finally superadded to abdominal tuberculosis which has existed for a longer period, in the same manner as meningitis complicates tuberculosis of the brain and pia mater, or pericarditis and pleurisy complicate tuberculosis of the pericardium and pleura. On the whole, cases of this kind appear to occur rarely.

I remember but one case, in a boy, aged five, who was admitted to hospital January 10, 1879, without any previous history. He was fairly nourished, and examination of the lungs merely showed diminished breathing in the upper portions of both lungs. The inflammatory abdominal symptoms present on admission, the great tenderness, tympanitis and distention, vomiting, fever, were therefore attributed to an acute peritonitis, whose cause remained unknown. Death occurred in collapse, January 18th.

Autopsy.—The distended abdomen contained about one and one-half litre of a somewhat dirty, purulent fluid; both layers of the peritoneum were covered with fibrino-purulent exudation; all the loops of the intestines were adherent to one another. Many submiliary and miliary tubercles were present in the serous coat, partly also in the deeper layers of the intestinal walls; the lower end of the ileum was studded with them so that the peritoneum was markedly thickened. In other situations the tubercles appeared in nests, with hemorrhagic neighborhood. Scattered tubercles in omentum. In the small intestines many solitary tubercles of the mucous membrane as large as sago-grains, and tubercular ulcers as large as tenpenny pieces, extending to the muscular coat; a few cheesy follicular ulcers. A little above the cæcum the entire wall of the intestine was converted into an ulcerated surface, on which cheesy tubercles and fresh gray nodules were visible. Ileo cæcal valve almost entirely destroyed by ulceration. Vermiform appendix dilated threefold, slightly stenosed at insertion into intestine; the dilated portion covered with tubercular ulcers. Numerous ulcers in colon. Fatty liver. Upper lobes of lungs of a slate-gray color; cicatricial retraction; a few cheesy spots.

This case of peritonitis might appear to be due to perforation of one of the numerous tubercular ulcers of the intestines, but none could be found, and the history of the disease, especially its long duration, does not favor this view. The case was probably one of acute miliary tuberculosis of the peritoneum, due to the old wide-spread tubercular enterophthisis, and which was combined with acute inflammatory phenomena. The following case shows that the latter, under such circumstances, may retreat into the background, and the symptoms assume a typhoid character:

J. S——, two and one-half years old, admitted January 23, 1878, said to have been previously healthy, and was taken sick three weeks previously with loss of appetite, diarrhœa, great weakness and fever, increasing paller and emaciation; temperature, 38.2°. Lips and tongue dry and encrusted, thirst; loam-colored, very offensive stools; spleen cannot be felt nor percussed, on account of tympanitis. Abdomen tense, not particularly painful; tympanitic catarrhal rhonchi in lower lobe of left lung. During next six days continued fever (morning, 38.2°; evening, 39.6°); pulse, 120; increasing meteorism without abdominal tenderness. January 29th, death.

Autopsy.—About 100 ccm. of cloudy brownish fluid with tough flakes of fibrin in the distended abdomen. Intestines markedly distended with gas and adherent by fibrinous exudation; serous membrane reddened in these places. Numerous miliary tubercles on surface of intestines and on the parietal peritoneum. Liver fatty. Intestinal mucous membrane unchanged.

This case is noteworthy from the fact that the tuberculosis was confined exclusively to the peritoneum. No other organ, not even the bron-

chial and mesenteric glands, was affected, and the liver alone showed the fatty infiltration so often found in tuberculosis. The most careful examination showed no cheesy focus from which the miliary tuberculosis of the peritoneum could have started. Of clinical importance is the comparatively rapid course of the disease (about four weeks), with symptoms which would lead more readily to a suspicion of typhoid fever than tubercular peritonitis. I call your especial attention to the very slight tenderness of the tympanitic abdomen, which harmonized very little with the appearances presented at the autopsy.

Cases like those just mentioned are, however, much less frequent than those which run a chronic course and present the classical history of tubercular peritonitis. The most prominent feature is the gradual increase in the size of the abdomen, which is usually regarded at first as gaseous distention, but in time arouses anxiety and causes the parents to seek medical aid. Of the numerous children whom I have treated for this disease, the youngest was two and one-half years old; the majority were from three to eight years old. The first glance at the abdomen will arouse the suspicions of the experienced physician; after the distention of the abdomen has steadily increased for a number of months, it finally presents an elliptical curvature; the abdominal walls are very tense, even shining, and the epigastric veins are dilated into bluish cords which show through the skin. The appetite suffers at the same time; the children grow thin, and when the affection is fully developed the contrast between the hemispherical prominent abdomen and the emaciated lower limbs always appeared to me somewhat characteristic. Many children complain from the beginning of colic-pains in the abdomen and tenderness on pressure, but I have much more often found the spontaneous pains, as well as those produced by pressure, absent entirely or confined to a few parts of the abdomen. When the latter is very markedly distended, examination by percussion and palpation very often indicates the presence of an accumulation of fluid (ascites), the sound varying according to the position of the patient, and a feeling of fluctuation is also often present. But this is by no means constant, as very little fluid is often present in the pelvis, and the distention of the abdomen is then mainly caused by the intestines, which are dilated by gas, force the diaphragm upward, and not alone cause a tympanitic percussion-note over the entire abdomen, but also on the sides of the thorax as high as the fifth rib. Occasionally some parts of the abdomen give a dull, others a tympanitic note, without being influenced by position; this is explained by the encapsulation of fluid exudation by peritoneal adhesions.

The distention of the abdomen is so characteristic that I even observed it continue until death in two cases of chronic peritonitis, although, in the latter, as a rule, there is considerable depression of the abdomen. But exceptions to this rule also occur. I have repeatedly observed, during the entire course of the disease, an unusual flatness, or even retraction of the abdomen, with or without tenderness, and the autopsies always showed complete absence of fluid exudation, empty and contracted intestines, and adhesion of the loops of intestines to one another and to the parietal peritoneum. In such cases the peritonitis usually formed only one link in the chain of general tuberculosis, and the symptoms of the latter were so prominent that the diagnosis of the peritoneal affection could be made on the autopsy-table alone, on account of the absence of the characteristic abdominal enlargement. But this enlargement may also be absent in those cases in which the chronic tubercular peritonitis

is the predominant affection. Thus, in a girl, aged six, who had long suffered from a remittent fever, and finally died of diphtheria, I found chronic peritonitis with adhesion of the loops of intestines to one another and to the abdominal walls, and innumerable miliary tubercles in the peritoneum and the new-formed adhesions, although no tubercular deposit was found in any other organ, with the exception of a few lumbar glands. The abdomen of this girl had been unusually flat, firm, and insensible during the entire course of the disease.

Cases like that just mentioned, in which the tuberculosis was exclusively confined to the peritoneum, or at least to the abdominal organs, are by no means rare:

A child, two and one-half years of age. Intestines adherent to one another and to the abdominal walls; collection of bright chocolate-colored fluid in the free spaces. Liver and spleen surrounded by firm fibrous adhesions and firmly adherent to adjacent parts. Numerous miliary tubercles in parietal peritoneum, serous layer of intestines, and greater omentum. Lungs, bronchial glands, and parenchyma of liver and spleen entirely free from tubercles.

A child, aged five. No fluid in abdomen; loops of intestines adherent to one another and to abdominal walls, also greater omentum, which is converted into firm mass, 2″ in thickness. Numerous cheesy masses situated between all these parts. Numerous miliary tubercles on free surface of peritoneum. Spleen small, very firm, its tissue like that of liver, and lungs free from tubercles. Bronchial glands cheesy.

O. T——, two and one-fourth years of age, admitted May 6, 1879, with emaciation, hemispherical distention of abdomen, projection of liver below border of ribs. Affection has lasted six months, without fever or symptoms referable to other organs. March 19th, death from collapse and œdema of lungs. Autopsy.—Loops of intestines adherent to one another and to abdominal walls; numerous miliary tubercles in the pseudo-membranes, the greater omentum and lower surface of omentum. Tubercular ulcers in intestines, with gray nodules on corresponding parts of serous layer. Liver fatty; spleen, lungs, and pleura free from tubercles. Œdema pulmonum. Cheesy bronchial and mesenteric glands.

In all these cases, the thoracic organs, with the exception of the cheesy bronchial glands in two instances, were entirely intact, and the peritoneum, the intestinal mucous membrane and mesenteric glands were alone affected with tuberculosis. This peculiarity of abdominal tuberculosis also explains the fact that such children, during the entire course of the disease, which may extend over six to twelve months, may present no other symptoms except the previously described hemispherical, very tense enlargement of the abdomen (which is traversed by venous plexuses), with or without tenderness, anorexia, increasing weakness and emaciation, irregular nocturnal exacerbations of temperature (up to 39.5°) being usually observed, while the morning temperature may be normal or subnormal (36.7°–35.8°). Death occurs either from an accidental complication or from exhaustion, after œdema of the lower limbs and scrotum has perhaps occurred in consequence of the increasing failure of the heart.

In many cases these symptoms are complicated by diarrhœa, which resists all treatment or returns after short intermissions, and is caused by tubercular ulcers of the intestines. The more extensive the tuberculosis, the more complicated are the symptoms, and the physical examination of the lungs, the obstinate cough and the remittent fever in conjunction with the local symptoms of chronic peritonitis present the picture which I endeavored to draw in describing pulmonary phthisis (page 164). I no longer attach any importance to the enlargement of the inguinal glands, as this is extremely frequent in children, and in some cases of chronic peritonitis was either entirely absent or only present to

a slight degree. A detailed description of the anatomical changes is unnecessary in view of the previous post-mortem reports (page 227). I will merely mention that I have rarely missed a fatty degeneration of the liver, and have also repeatedly observed parenchymatous nephritis as a complication.

In the last stage of the disease, perforation externally sometimes occurs, an abscess forming in the abdominal walls and opening. It gives exit either to the contents of an encapsulated peritoneal exudation mixed with the abscess, or to thin, fæcal fluid. I observed rupture in two cases, both through the umbilicus. In one case the autopsy showed a communication of the external opening of the abscess with a perforated loop of intestine adherent to the umbilicus, in addition to all the signs of severe tubercular peritonitis. In another child, in whom a quantity of fluid was present in the abdomen, very profuse purulent evacuations suddenly occurred a few days before death, the size and tenderness of the abdomen rapidly diminishing at the same time. The autopsy showed no more fluid in the abdomen, but in the posterior wall of the peritoneal sac, corresponding to the right iliac fossa, was an ulcerated opening three lines in diameter, through which the sound passed into a tortuous canal directed toward the rectum. Although a direct communication with the latter could not be distinctly proven, there can be no doubt that the fluid ruptured into the rectum.

As in every other form of tuberculosis, so also in this death may be due to a terminal tubercular meningitis, as in the following case:

A boy, aged eight, treated in the summer of 1878 for pericarditis; re-admitted October 3d. He then presented such marked ascites that puncture was performed, 2,050 grms. of an opaque, albuminous fluid being removed. Examination then showed considerable projection of the liver below the ribs. The suspicion of a hepatic origin of the ascites was so much the more justified as nothing abnormal was discovered in the heart or kidneys, and no trace of œdema was present. Urine free from albumen, scanty. The enlargement of the abdomen had attained its previous dimensions by October 13th, but there was no pain, either spontaneously or on pressure. A second puncture, November 11th, removed 3,800 grms. of fluid, but it soon re-accumulated. The boy became more emaciated, but had good appetite and no fever. Toward the end of February, 1879, clear serum began to escape from the projecting umbilicus. At the end of March, the umbilical region became more prominent, red, and led to the suspicion of an impending perforation, but this did not occur; the discharge of serum continued and the tension of the abdomen diminished. April 16th, fever developed without any distinct cause. The lungs appeared normal. Cerebral symptoms (apathy, somnolence, vomiting, headache) appeared and death occurred May 7th in convulsions.

Autopsy.—Parietal and visceral layers of peritoneum covered with grayish white nodules. Around the liver and in the mesentery these tubercles had coalesced into nests as large as a bean, and formed irregular nodules. The greater omentum contained nodules as large as a pea. About one hundred grammes clear yellow fluid in abdominal cavity. Spleen and kidneys normal. Liver very large, markedly fatty, with a few tubercles from the size of a millet-seed to that of a hemp-seed. The left costal pleura thickly strewn with tubercles, the pulmonary layer to less extent. Lungs congested, but free from tubercles. Pericardial cavity obliterated by old adhesions. Muscular substance of anterior surface of right ventricle in condition of fibrous degeneration. Severe tubercular meningitis of base and convexity. Brain œdematous, ventricles dilated and filled with serum. Tubercles on choroid plexuses.

This case, which is important in many respects, teaches that:

1. A severe chronic, tubercular peritonitis may exist without any pain, and merely manifest itself by the symptoms of ascites, increasing emaciation, and cachexia.

2. That, under these circumstances, a projection of the liver below

the border of the ribs, on account of fatty infiltration, may lead to the false diagnosis of ascites due to hepatic disease.

3. That in very great tension of the abdominal walls, the umbilicus, growing more and more attenuated, may permit the escape of the serum, accumulated in the abdominal cavity, through fine rents.

This case also shows that operative treatment of ascites from chronic tubercular peritonitis need no more be dreaded than in every other form of ascites. We should merely convince ourselves, by careful percussion, of the free mobility of the fluid, in order not to enter adhesions or loops of intestines with the trocar. In this case I convinced myself of the utility of gentle percussion, which gave a dull sound over the situation which I chose for puncture, while more vigorous use of the pleximeter gave an almost tympanitic note. It is scarcely necessary to add that puncture is merely used as a palliative measure to relieve dyspnœa. The constant employment for many weeks of hydropathic fomentations, baths, and applications of iodine to the abdomen, were as useless as the internal administration of cod-liver oil, iodide of iron, and iodide of potassium. Those few cases which improved, or were even cured under this treatment, were, in my opinion, simple chronic, and not tubercular peritonitis. In any event, these rare cases show that we should not satisfy ourselves with the diagnosis of tubercular peritonitis, but should always persevere in our attempts at treatment. The application of iodine should never be made over too large a surface. I divide the abdomen into four parts by two lines intersecting at the umbilicus, and have one of these parts pencilled daily with tincture of iodine. We can then continue for weeks without any bad results, and the albuminuria, said to result frequently from the application of iodine in children, could be discovered in none of these cases.

I previously mentioned that chonic tubercular peritonitis may be accompanied by obstinate diarrhœa, which must almost always be regarded as the result of tubercular ulcers of the intestines. These ulcerations also occur in children when the peritoneum is almost normal and the tuberculosis chiefly affects the lungs. I will here refer only to a few details which affect the age of childhood.

If a few tubercular ulcers are alone present, diarrhœa may remain absent, and a positive diagnosis is then impossible. On the other hand, we know that a chronic diarrhœa, which occurs without any distinct signs of tuberculosis in other organs, but complicated with increasing emaciation, exhaustion, and remittent fever, is often produced by chronic intestinal catarrh, with follicular ulcerations (page 204). Tubercular ulceration of the intestines can, therefore, be diagnosed with any approach to certainty only when a more or less profuse and obstinate diarrhœa is combined with distinct signs of tuberculosis in other parts. But it must also be remembered that tubercular and follicular processes in the intestines are occasionally associated.

Max H——, aged three, admitted April 1, 1873, with distended and tender abdomen; suffers for three months from severe diarrhœa. Examination of chest negative. No fever, but increasing emaciation and weakness. April 6th, œdema of left leg, which disappears in two days. April 16th, œdema of face, associated after a few days with œdema of left leg. Diarrhœa continues; increasing collapse. June 8th, death.

Autopsy.—Numerous small, peri-bronchitic, cheesy spots in both lungs. Cheesy bronchial, tracheal, and mesenteric glands; fatty liver. The next to the lowest Peyer's patch contains an irregular ulcer, in whose edges enlarged follicles are visible, which are cheesy in the centre. Submiliary gray nodules on corresponding part of serous

coat. From the ileo-cæcal valve begins a very considerable enlargement of the follicles of the large intestine and of the entire wall of the colon. Ulcers very soon appear, become more numerous farther down, and finally coalesce, so that a few small parts of congested mucous membrane are left between them in the descending colon and rectum.

The chief affection in this case was a chronic dysentery, with ulceration of the follicles (page 208). As it developed in a child who was predisposed to cheesy tubercular processes, there is nothing striking in its accidental combination with the cheesy ulcer in a Peyer's patch, which had caused miliary tuberculosis of its immediate neighborhood. It is noteworthy, from a clinical standpoint, that this enormous ulceration of the intestinal mucous membrane ran its course with scarcely any fever. In other cases, however, marked hectic occurs, the morning temperature being occasionally, for weeks, two to three degrees lower than the evening (morning, 36.5°–35.8°; evening, 39.5°). The œdema of the left leg and face, which could not be explained by a renal affection, also deserves mention. The local œdema of the left leg could only have been produced by thrombosis in the territory of the left femoral vein, as venous stasis in consequence of heart-failure must have produced œdema of both legs. Unfortunately the veins of the thigh were not examined at the autopsy, but it is well known that "marantic" thromboses are by no means rare in phthisical patients; they may even extend into the inferior vena cava, and, by the prominence of their symptoms, force the primary affection into the background.

In the treatment of tubercular phthisis of the intestines I would recommend the measures advised in chronic intestinal catarrh and catarrhal ulcers of the intestines (page 206). But sooner or later the children die from exhaustion on account of the constant diarrhœa, the tuberculosis of other organs, and the hectic fever.

XVI. Diseases of the Liver.

The liver is much more rarely affected by disease in children than in adult life. Cirrhosis, which is so common in the latter, is exceptional in children.[1] I have never found this disease fully developed in children, but have several times seen moderate hyperplasia of the interstitial tissue, with fatty degeneration of the liver-cells. This could not be determined before death, and therefore presents no clinical interest. Abscesses[2] and tumors of the liver occur still more rarely. West reported a medullary sarcoma at the age of eight months, to which I may add the following case:

A child, aged two and one-half years, presented beginning of February, 1878. Healthy until Christmas, 1877, when the abdomen began to enlarge. The liver was found to be very much enlarged. Rapid increase during the next few weeks; flat, soft, almost fluctuating projections can be distinctly felt upon the liver; dilatation of veins of abdomen and lower part of thorax. Increasing emaciation, slight jaundice.

[1] Unterberger: Jahrb. f. Kinderheilk., Bd. IX., S. 390, 1876. Fox: Ibid., Bd. XIII., S. 404, 1879. Neureutter: Oester. Jahrb. f. Pädiatrik., Bd. VIII., S. 14, 1879.

[2] Abscesses of the liver, due to migration of round worms, have been observed only three times in children. Scheuthauer, who reported the third of these cases (Jahrb. f. Kinderheilk., VIII., S. 63), regards them as cheesy spots, which, in fact, do not contain worms, but merely the ova; whence he concludes that the lumbrici may wander from these spots toward the ductus choledochus.

Death on March 23d. Autopsy.—Liver enlarged threefold, yellow, contains at the periphery and internally numerous yellowish white, soft tumors, from the size of a hazel-nut to a walnut. Gall-bladder dilated; cystic duct compressed by a tumor. All other abdominal organs normal; the other viscera could not be examined.

We have more frequent opportunity of observing echinococcus cysts in children from eight to twelve years. I will merely report one case, which appears noteworthy on account of the apparent good effects of puncture.

A boy, aged eleven, admitted July 15, 1878; nothing abnormal except protrusion of right hypochondrium. The liver projected two fingers' breadth beyond the border of the ribs, and between the umbilicus and xiphoid process could be felt an elastic, hemispherical projection about as large as an apple. July 19th, about one hundred grammes of a clear serous fluid were removed from this tumor with a fine trocar. The fluid contained no albumen, and although it contained no hooklets or succinic acid, it could alone have come from an echinococcus cyst, which was probably sterile. The patient was discharged nine days after the puncture. No trace of the elastic prominence could be detected, and the lower border of the liver could merely be felt to a slight extent. Whether the recovery remained permanent is unknown.

Amyloid degeneration of the liver is much more frequent in childhood than the affections previously mentioned. The smooth enlargement of the organ, which is not tender or very slightly so, sometimes attains such dimensions that it occupies the entire upper part of the abdomen, and extends on the right side below the spine of the ilium. Apart from the large size, the diagnosis depends especially on the implication of the spleen and kidneys in the amyloid degeneration; the tumor formed by the former can be felt in the left hypochondrium, or at least demonstrated on percussion; the affection of the kidneys is shown by albuminuria. But if these complications are absent, the circumstances under which the enlargement of the liver occurred may furnish an explanation, since amyloid degeneration, as is well known, almost always develops in consequence of a cachexia or dyscrasia. Prolonged suppuration of bone frequently acts as a cause in childhood, and I have repeatedly seen waxy degeneration of the liver, spleen, and kidneys in spondylitis, coxitis, and other carious affections of the bones. I cannot, however, agree with those who ascribe a similar influence to rachitis. Despite the enormous number of rachitic children whom I have treated, I do not remember a single uncomplicated case in which waxy degeneration could be proven either clinically or anatomically. When this did occur, other important morbid processes, such as caries or tuberculosis, were present. Amyloid processes may also be produced in children by syphilis, not the hereditary form in its first development, the influence of which on the production of interstitial or gummy hepatitis we have previously discussed (page 45), but chronic syphilis, whether of hereditary origin or acquired at a later period.

A. Z——, aged eleven, admitted January 12, 1875. The mother had an eruption, which disappeared spontaneously at time of delivery. A year ago, swelling of nose, ozæna, extraction of several pieces of bone. During last three months, pain in left arm and both tibiæ, emaciation. Tibiæ covered with hard nodes anteriorly; lower epiphysis of left arm swollen, movement of arm painful, muscles less developed than those of right arm. Glands of neck swollen, uvula absent. Liver dulness begins at upper border of fourth rib, projects 4 ctm. beyond border of ribs in mammary line, 3½ ctm. in parasternal line, and 3 ctm. beyond the base of xyphoid process. Lower border of liver can be distinctly felt, also spleen. Albumen in urine, no casts. Treatment: Iodide of potassium, 5 : 150, a tablespoonful t. i. d. After administration of

10.0 the pains in bones ceased, nodes diminished, movement of arm improved. The nose, liver, spleen, and kidneys remained in same condition, and the girl, who is now fifteen years old, occasionally comes to the dispensary for iodide of potassium, as this alone alleviates the nocturnal pains in the arm and tibiæ.

B. R——, aged twelve, admitted December 2d, 1875; suffered, while a child, from enlarged glands and suppuration around right knee and thigh, scars of which are still visible. No dysentery or protracted diarrhœa. For some years very frequent straining and pain in anus, followed by a thin, occasionally bloody, evacuation. Frequent angina tonsillaris. Opacity of left cornea, thick nose, coryza, grayish yellow, adherent coating on tongue and cheeks, tonsils and uvula. A hemorrhoid as large as a bean. Liver dulness begins at lower border of fourth rib, and extends to level of umbilicus; its surface very hard and smooth. Spleen not enlarged. Urine pale yellow, clear, albuminous, no casts. Passages variable, occasionally normal, but often combined with tenesmus, clay-colored and streaked with blood. December 8th, local examination with finger and speculum showed roughness, swelling and redness of rectal mucous membrane, and an annular stricture above the internal sphincter. These symptoms, together with nocturnal pains and enlargement of inguinal and cervical glands, favored the diagnosis of syphilis, and the administration of iodide of potassium, with application of a one per cent. solution of nitrate of silver to the affected parts of the mouth and pharynx, produced, at the end of a week, a very favorable effect on the latter and on the coryza and pains in the limbs. The rectal disturbance continued undiminished. December 20th, treatment by inunctions was begun, but without effect. March 7th, the child transferred to eye ward on account of keratitis of right eye. On readmission to my wards (May 24th), the cachexia had progressed, the dimensions of liver had increased; pressure on liver somewhat painful. Condition otherwise unchanged; five to six thin evacuations daily, containing pus and blood. Fever, thirst, anorexia, nausea. November 16th, death.

Autopsy.—Extreme emaciation. Lungs normal; heart small and flabby, muscular tissue pale and grayish red. Pharynx normal; a gumma, as large as a hazel-nut, upon the uppermost part of the posterior wall of the larynx. Liver enlarged threefold, waxy degeneration. Spleen relatively small, shows amyloid degeneration of the pulp; similar condition of kidneys and mucous membrane of the stomach and intestines. At the border of ileum and jejunum is an ulcer with irregularly swollen edges and clean base. No tubercles. Peyer's patches prominent; a small ulcer, similar to one described, near the cæcum. Mesentery and loops of intestines thickened, and many of latter fixed by adhesions. Liver adherent to diaphragm. From the splenic flexure of the colon the mucous membrane begins to be swollen and reddened; then ulcers are found down to the rectum, in which only isolated patches of the mucous membrane are intact. Colon markedly thickened and retracted; rectum considerably narrowed down to the anus.

Although the previous history was uncertain in both cases, syphilis must undoubtedly be regarded as the basis of the complicated symptom-complex. The liver, spleen, and kidneys showed waxy degeneration; in the second case, this was also found in the entire intestinal mucous membrane, which was covered with ulcerations and almost entirely destroyed in the rectum. The chronic adhesive peritonitis must be regarded as due to the propagation of inflammation from the mucous membrane to the serous layer, and not as a specific syphilitic affection. Both cases also showed the inefficacy of specific treatment. The amyloid degeneration and the ulceration of the intestinal canal remained unchanged.

Among all diseases of the liver, fatty degeneration is most frequently observed in childhood, although much more often on the autopsy-table than clinically. It is observed in very many cases of severe infectious diseases, especially diphtheria and scarlatina, also in tubercular and phthisical children, or those exhausted by chronic diarrhœa. The liver is thickened, bright or grayish yellow, compressible, and, under the microscope, the cells contain smaller and larger drops of fat. In tubercular children I could distinctly feel the lower border of the liver below the ribs, even in dorsal decubitus, and was surprised at the autopsy to find a fatty liver of normal size, or only slightly enlarged—a fact which

can alone be explained by an abnormally low position of the diaphragm or unusual length and flaccidity of the suspensory ligament. Much more rarely the organ was considerably enlarged, and then occupied a more or less large part of the right hypochondrium and upper portion of the abdomen.

Whether fatty liver may develop in consequence of improper nourishment, as in adults, I leave undecided. At least the causes of this condition, viz., excessive ingestion of fat and alcoholic liquors, with deficient exercise, are found exceptionally in children, and I have had but one case which can be brought into this category:

R. M——, aged two and one-half years, admitted January 10, 1875; had measles (?) some time ago, diarrhœa for some months, also whooping-cough. The child has for a long time drunk a good deal of Bavarian beer. Fine desquamation of integument of trunk and slight œdema of feet and eyelids; urine normal. Some fluid in abdomen; liver enlarged and extending to umbilicus; four to six thin, brown evacuations daily. Depression of temperature, emaciation, feeble pulse, somnolence. January 17th, death.

Autopsy.—Heart-muscle pale, grayish red, fatty. Liver enlarged, grayish yellow; marked fatty degeneration of cells. Intestinal mucous membrane very pale. Epithelium of cortical substance of kidneys very fatty.

Whether the extensive fatty degeneration of the liver, kidneys, and heart were due to the ingestion of beer must remain undecided.

Icterus, due to catarrh of the duodenum and biliary passages, occurs almost as often as in adults, and, at times, in an almost epidemic manner. The majority of the children are over three years old, but I have also seen the affection in two children, aged respectively two and five months. It was constantly accompanied by anorexia, perhaps nausea and vomiting in the first few days, gray or clay-colored fœtid evacuations, which were occasionally frequent and fluid, but more frequently scanty, biliary urine, malaise, tendency to sleep. Fever was almost always absent or present in the beginning to a moderate extent. I have never seen the pulse reduced in frequency to fifty or even less beats per minute;[1] it always varied from one hundred to one hundred and twenty, and I must therefore believe that the greater irritability of the nervous system of the child compensates the inhibitory influence of the gallic acid on the heart. Perhaps the slow pulse in icterus may be observed in very quiet children. A projection of the liver below the ribs, due to biliary stasis, can very rarely be felt, but is more readily detected by percussion. All my cases terminated favorably in one to two weeks; one presented special interest on account of the repeated occurrence of severe febrile movement:

G. K——, aged eight, admitted December 13, 1875, on account of a traumatic necrosis of the right ischium, which had given rise to a fistula in the perineum. January 29th, icterus with high fever, 40.4°; pulse, 132; euphoria. Liver prominent. Icterus increasing during next few days; urine biliary, no albumen, leucin or tyrosin; stools discolored and fœtid. This condition continued seven weeks, during which the wound closed. The fever occurring on January 29th lasted two days. February 2d, fever recurred (morning, 38.4°; evening, 39°), and gradually diminished until February 21st, when it again reached 41°, then diminished and disappeared March 21st. The icterus and enlargement of liver subsided at same time.

The first suspicion that the violent fever and jaundice were pyæmic in character, and due to the disease of the bone, was disproved by the entire absence of chills and the favorable course of the affection. The

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

view that obstruction of the bile-ducts was caused by calculi, which occasionally give rise to such febrile attacks, was negatived by its extreme rarity in children and the entire absence of pain, and it therefore only remains to attribute it to a severe, obstinate catarrh of the biliary passages, which extended far into its branches.

I would recommend the following treatment: in the first three days give purgatives (calomel, 0.06–0.1 every two hours, inf. sennæ comp., inf. rad. rhei (P. 39); then hydrochloric acid (P. 3), which may be given from the start if diarrhœa exists). Absolute rest and strict diet, avoidance of all meat, except bouillon, gruel soup, zwieback, farina, very soft boiled rice, compote; as a drink, a few glasses of Wildunger water daily, in order to rapidly remove the bile-pigment excreted by the urinary tubules. In two very obstinate cases, I saw surprising results from profuse intestinal injections of cold water by means of the irrigator.

Fatal icterus, combined with cerebral symptoms, caused by acute atrophy of the liver, occasionally occurs in children. I have seen it in three cases, in only one of which an autopsy was obtained. It presented no features which are peculiar to childhood.

XVII. Diseases of the Spleen.

The most frequent disease of the spleen in childhood is tuberculosis, but, in my experience, it never gives rise to any definite symptoms, and can therefore not be diagnosticated.

On the whole, affections of the spleen can alone be recognized with certainty when it forms a tumor which can be palpated and extends more or less beyond the border of the left ribs. I expressly say a tumor which can be palpated, because I do not place absolute confidence in percussion alone, least of all in children, who struggle during the examination and readily cause mistakes in percussion on account of the muscular contractions. Palpable tumors of the spleen are especially found, as in adults, in certain infectious diseases, particularly typhoid fever, recurrent fever, and repeated attacks of intermittent fever; more rarely in acute miliary tuberculosis and cerebro-spinal meningitis. I have never observed it in scarlatina, measles, erysipelas, or catarrhal angina.

Among chronic diseases, amyloid degeneration of the spleen, in consequence of caries of the bones and syphilis, may give rise to a tumor, although the amyloid spleen is not infrequently normal in size or even diminished. My previous remarks (page 233) on amyloid degeneration of the liver also hold good with regard to the spleen. I will now turn to the consideration of simple hyperplasia of the spleen, which is by no means rare in children during the first few years of life. This disease can usually be recognized by the peculiar yellowish white color of the skin, especially the face, which can best be compared to that of white wax. The spleen then extends beyond the border of the ribs as a hard, smooth tumor, and not infrequently occupies the entire left half of the abdomen, so that its anterior border extends to the umbilicus, or beyond it, and can be distinctly grasped when the abdominal walls are flaccid. Occasionally the tumor is somewhat movable. Marked tension of the abdominal muscles, especially in crying, may render the palpation of smaller tumors difficult, and we must then wait for the quiet intervals, during which the descent of the diaphragm in inspiration makes the spleen perceptible. The percussion-note is dull over the entire tumor,

but toward its upper border, as a rule, it shows no special change. This is due to the weight of the tumor, which drags it downward and may produce considerable dislocation of the organ. Thus, in a child aged eighteen months, I found that the spleen, which could originally be felt in the left hypochondrium, finally lay in the left iliac fossa, and could be moved quite readily.

The chief symptoms, viz., the tumor and the peculiar color of the skin, are often accompanied by œdema of the feet and eyelids, and especially by small hemorrhages into the skin. Hemorrhages from the mucous membranes,[1] even a fatal hemorrhage from a small vaccination wound,[2] have been observed. Leukæmia was only exceptionally present in my cases; as a rule, the proportion of white to red blood-globules was not appreciably changed. Nothing is gained by placing this hyperplasia of the spleen in the category of "pseudoleukæmia." I should also oppose this view for the reason that in no case have I observed enlargement of the lymphatic glands.

The etiology was obscure in almost all of my cases. In rare cases it was preceded by intermittent fever of long duration and, in one case, the mother stated that she had suffered from chills and fever during pregnancy with the child in question.[3] The disease was occasionally preceded by frequent dyspeptic disturbances, vomiting and diarrhœa, but they were usually absent, and the increasing pallor and enlargement of the abdomen alone attracted attention. The appetite and evacuations from the bowels may be entirely normal, and emaciation does not occur for a long time.

The prognosis must always be considered doubtful, but only absolutely bad in the rare cases in which examination of the blood shows marked leukæmia. Experience teaches that the majority of children with chronic enlargement of the spleen die from progressive anæmia, emaciation, anasarca, and finally from dropsy of the cavities, unless some accidental complication—for example, broncho-pneumonia, causes an earlier fatal termination. Post-mortem examination reveals simple hyperplasia of the spleen. The extremely firm tumor, whose capsule is occasionally thickened and adherent to adjacent parts, is pale red or brownish gray on section, occasionally dark bluish red, with more or less distinct Malpighian bodies. In some cases we find numerous whitish depots, formed by the accumulation of lymphoid cells.

Examples are not lacking, however, of the complete retrogression of large splenic tumors. Nothing can be expected from nature in this respect, but appropriate treatment is required for many months.

M. E——, aged one year and nine months, admitted January 14, 1847, to Romberg's polyclinic, with excessive atrophy, waxy complexion, enormous splenic tumor, œdema of the face, hands, and feet. Treated with iron, salt, and iron-baths. November 2d, the spleen was diminished one-half; May 29, 1848, the spleen could no longer be felt. Complete and permanent recovery. According to the statement of the mother, febrile attacks, combined with sweating, had occurred in the summer of 1846. This was expressly denied in the two following cases:

A. N——, aged eighteen months, rachitic, presented May 8, 1865. Progressive enlargement of spleen for past four months; no leukæmia, waxy complexion, treatment with quinine and iron. Complete diasappearance of tumor, and blooming appearance by end of July.

G. M——, aged eighteen months, presented May 10, 1878; emaciated, waxy complexion, marked enlargement of spleen. Treated with quinine and iron for five months.

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[1] Rilliet and Barthez, II., 34. [2] Pott: Klin. Wschr., S. 655. 1879.
[3] A similar case was reported by Playfair (Schmidt's Jahrb. f. 1858, II., S. 338).

In November, a small portion of the spleen could still be felt below the border of the ribs; in December this also had disappeared.

We should therefore never lose courage, but persevere in the administration of iron and quinine (P. 40) for many months, or even years. Proper nourishment from the breast, and later good milk, broths and wine are indispensable, and lukewarm salt baths (one to two pounds of salt to the bath) may be recommended as effective adjuvants. In some cases this treatment was entirely useless, or, at the most, produced improvement of the general condition without affecting the tumor; but as other remedies of repute (bromide of potassium, iodide of iron, arsenic), also proved useless, I can still recommend to you the treatment with iron and quinine as that which, at least in my experience, has most success. I have had no experience with regard to the use of the induced current employed by Botkin in adults. The parenchymatous injections of a two per cent. solution of carbolic acid and Fowler's solution (1 : 10 of water), recommended by Mosler, must excite some fears, especially in little children.

XVIII. Abdominal Tumors.

Nodular thickenings of the walls of the intestines, in consequence of chronic peritonitis, may constitute tumors of the abdomen. Somewhat more frequently, though still quite rarely, we meet with tumors formed by new-formations, especially of a sarcomatous nature, and which may grow from various parts of the abdominal cavity. This occurs most rarely from the peritoneum, as I observed in a boy aged eleven. In addition to enormous ascites, nodular bands and tumors could be felt in various parts of the abdomen, and proved, on autopsy, to be numerous sarcomata of the omentum, mesentery, mesocolon, and the serous coat of the intestines. Sarcoma develops more frequently from the connective tissue or glands, which are found in the pelvis or behind the peritoneum in front of the spinal column, and may then grow into an enormous tumor.

A boy, aged five; the first striking symptoms were increase in the size of the abdomen and unusual irritability; later, œdema of face, lower limbs and genitalia, pains in the abdomen, diarrhœa, and emaciation. An irregular tumor could be felt in the hypogastric region, and finally extended to the umbilicus. Death occurred from exhaustion about three months after the enlargement was first noticed.

Autopsy.—A grayish white, lobular, hard tumor grew from the pelvis, was somewhat adherent to the right ilium, omentum, and a few loops of intestines; pushed the intestines upward and occupied the entire abdominal cavity below the umbilicus; no ascites. Glands of epigastrium and mesocolon degenerated in a similar manner, and also the lower end of right kidney; a nodule as large as a hazel-nut in the cortex of left kidney. All other organs normal. The tumor was found to be a medullary sarcoma, starting apparently in the retroperitoneal glands.

While the kidneys were affected merely secondarily in this case, we find sarcomata, which may lead to enormous tumors, with comparative frequency in the kidneys and suprarenal capsules of children; they usually appear as medullary sarcoma, to which class most of the cases described as "cancer of the kidney" belong. The striated muscular fibres found by Cohnheim[1] in these sarcomata in two cases, show that these growths

[1] Eberth: Virch. Arch., Bd. 55, S. 518; Cohnheim: Ibid., Bd. 65, S. 64; Landsberger: Klin. Wschr., S. 499, 1877; Kocher u. Langhans: Jahrb. f. Kinderheilk., XIII., S. 152, 1879.

are in part congenital, and this explains their comparatively frequent occurrence in very young children. As the neoplasm rarely affects both kidneys, it may be mistaken for a tumor of the liver or spleen. Examination of the urine, as a rule, affords no clue, as the unaffected kidney usually acts normally, the affected one is more or less destroyed by the new-growth, and the ureter also involved, so that no urine reaches the bladder. Under such circumstances the usually rapid growth of the tumor, the enlargement of the abdomen, the dilatation of the subcutaneous veins, the increasing weakness and emaciation, are the only symptoms useful in diagnosis. We can draw a conclusion with regard to the implication of the kidneys only when the tumor has been observed to grow gradually from the deeper part of one of the hypochondria.

My own experience is confined to a small number of cases. Two affected children in the first year; in a third, which occurred in an older boy, the sarcoma was firmly adherent to the left kidney, but appeared to have started in the retroperitoneal glands.

M. K——, aged six, admitted to hospital, April 19, 1879. On September 28, 1878, he fell from a picket and injured the left testicle, which was finally extirpated October 12th. March 12th, appeared in the dispensary; examination negative. At the end of March, a tumor was felt in left hypogastric region and rapidly increased, and necessitated admission to the Charité. The tumor then extended 2 ctm. to the right of the linea alba and was separated from the border of the ribs by a space of three fingers' breadth; superficial veins dilated; percussion-note dull. The tumor continued to grow very rapidly. General condition fair, urine normal, progressive emaciation. The growth soon occupied the largest part of the abdominal cavity. Finally, dyspnœa, cyanosis, œdema of the lower limbs, diarrhœa. May 19th, death.

Autopsy.—Parietal layer of peritoneum adherent over a large surface to a tumor as large as a man's head, which lies immediately upon the vena cava and aorta and can be removed with difficulty. The left kidney is situated on the upper and external part of the tumor without being continuous with it: its parenchyma, like that of the right kidney, entirely normal. The tumor weighed 3600 grm. and proved to be a myxo-sarcoma. Enlargement of the peritoneal glands down to the genitalia. Numerous diphtheritic ulcerations in the colon. Both ureters dilated to thickness of finger and filled with clear fluid (result of compression). Other organs normal.

Farther examination showed that the tumor of the testicle, extirpated October 12th, was a spindle-cell sarcoma, which had resulted from the injury and given rise secondarily to the tumor of the glands. The rapid growth of the tumor is especially interesting from a clinical stand-point. On March 12th, I could discover no trace of it on palpation, and six weeks later it occupied the larger part of the abdominal cavity.

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PART VII.

DISEASES OF THE UROPOËTIC ORGANS.

I. Inflammatory Affections of the Kidneys.

The most frequent change found in the kidneys during childhood is the condition known as "cloudy swelling," *i.e.*, a moderate increase in the size of the kidneys, the cortical substance of which is more or less enlarged, and of a slightly grayish color. This condition, produced by swelling and granular cloudiness of the cortical epithelium, which may finally lead to fatty degeneration, was observed very often in children who died of various diseases, without presenting any renal symptoms. It was especially frequent in small atrophic children, and in those who died from exhausting diseases attended with profuse loss of water, such as cholera, chronic intestinal catarrh, enterophthisis, dysentery, general tuberculosis, etc. It may also occur in consequence of high fever in severe acute diseases, such as pneumonia, typhoid fever, scarlatina, relapsing fever, and the hepatic cells, and muscular fibres of the heart may then show the same changes. These lesions cannot, however, be diagnosed.

There is an imperceptible transition between cloudy swelling and the higher grades known as acute parenchymatous nephritis. The granular and fatty epithelium-cells of the convoluted tubes degenerate in part into a fatty detritus, which, mixed with desquamated but healthy epithelium, white and red blood-globules and cylindrical hyaline casts, fill the lumen of the tubes. The nutritive disturbance is thus intensified into inflammation, which may also be indicated by an accumulation of lymphoid cells in the interstitial connective tissue and by nuclear proliferation in the glomeruli (glomerulo-nephritis of Klebs). The kidneys present a more or less tense, occasionally cylindrical enlargement, the capsule is readily detached, and the venous plexuses appear more distinctly on the paler surface; on section, the broad, yellowish gray cortex contrasts with the congested, dark red pyramids, the papillæ of which may alone appear pale. More rarely a part of the cortex also shows congestion and larger or smaller hemorrhages. The anatomical changes of acute diffuse nephritis are so alike in children and adults, that I do not feel called upon to discuss the disputed points with regard to the finer changes in the tissues.[1]

[1] Klein: Minute Anatomy of Scarlatina, Lancet, I., 571, 1877. Friedländer (Arch. f. Phys. Jahrg., 1881), like Klebs, considers the changes in the glomeruli as the chief factors in scarlatinous nephritis (increased size and proliferation of the nuclei on the tufts of vessels, thickening and cloudy condition of the capillary walls, with occlusion of their lumen).

It may be boldly maintained that, with the exception of an extremely small minority, all cases of diffuse nephritis in children are of scarlatinous origin, and we shall therefore first discuss this form. My experience, which includes many hundred cases, will enable me to describe the disease in detail.

In the majority of cases the nephritic processes constitute, at least from a clinical point of view, a sequel of scarlatina, which, at the earliest, occurs from the twelfth to fourteenth days, but more often in the beginning of the third week from the appearance of the eruption.[1] I do not agree with the view that they are due to "cold" or "suppressed perspiration;" it is much more probable that the unknown virus of scarlatina exercises its specific influence on the kidneys, which may even be manifested during the first few days.

The mildest form of the disease appears as a rapidly subsiding albuminuria. If we make daily examinations of the urine at the time mentioned and during the entire third week, we not infrequently find a smaller or larger amount of albumen, which may disappear on the afternoon of the same day or on the following morning, but occasionally reappears temporarily without interfering with the general condition. It may therefore be asked whether this is a very mild nephritis or an albuminuria, caused by other influences which favor the passage of the serum of the blood. A further development of the disease from such a beginning is by no means rare, and as, on the other hand, nephritis may be found at the autopsy, although no albumen was present in the urine during life, I would advise you, in these cases of temporary albuminuria, to keep the children in bed, order mild diet, and stimulate the excretion of urine by diuretic mineral waters (Biliner, Wildunger).

This is so much more necessary if the albuminuria remains permanent. Several weeks may elapse without the appearance of any other symptom, except perhaps increasing pallor of the skin. During this time the urine is sometimes scanty, sometimes profuse, often contains considerable uric acid salts, and almost always albumen, a few hyaline casts and desquamated epithelium, which are occasionally found only on repeated and careful examination. In a case of this kind, for example, I saw the albuminuria continue from February 5th to March 10th, the child feeling quite well during this time, with the exception of a dyspeptic diarrhœa. Rest in bed for a month, liquor potass. acetat., lukewarm baths, and finally iron produced complete recovery in this, as in other similar cases. The euphoria may continue one to two weeks, although the amount of albumen is so great that almost half the urine coagulates on boiling. Even when the urine is scanty and contains blood, I have seen the appetite and good humor undisturbed for weeks, and it therefore follows that the urine should be examined daily after the end of the second week in every case of scarlatina.

Certain morbid phenomena are, however, much more often present. The children feel uncomfortable, in bad humor, lose appetite, and complain of headache. The urine becomes scanty and opaque, often deposits a yellowish red sediment, which dissolves on boiling, showing that it is composed of uric acid salts. Occasionally this condition of the urine precedes the albuminuria by several days. At times the scene is opened by complete anuria lasting twenty-four hours, or only a few

[1] On careful examination we will often find, at the height of the affection, a slight amount of albumen and a few hyaline casts in the urine, perhaps only the latter.

tablespoonfuls of cloudy urine are passed in this time. Partial œdema may occur at the same time, though it may also be absent during the entire course of the disease. In the majority of cases, however, a variable degree of œdema becomes noticeable sooner or later. In some only the eyelids, perhaps also the dorsal surfaces of the feet and the ankles are slightly œdematous, its intensity varying from day to day; in many cases, other parts of the skin, especially the scrotum and penis, also take part, or general anasarca occurs, the swollen eyelids can scarcely be opened, and the enlarged thighs are covered with an erythematous redness in those places where they are in contact with one another, and with the distended scrotum. The distended epidermis of the lower limbs occasionally ruptures, and the serum exudes in drops, so that finally the outer layers of the skin become macerated and excoriated. Under these circumstances, which must be regarded as very unfavorable, the skin, especially the face, and the visible mucous membranes, assume an anæmic, waxy color. One-half the face or body is often more swollen than the other, because the child prefers to lie on that side. When the skin is very tense it becomes sensitive, and pressure upon it produces pain.

The condition of the urine is unaffected by the intensity of the œdema. It is almost always scanty, not infrequently diminished to 100 ctm. or even a few tablespoonfuls in the twenty-four hours, while on other days a larger quantity is passed, though it never reaches the normal amount. I have never noticed pain in micturition, though frequent tenesmus is often present, a small quantity of urine being passed at a time. The urine is acid, its specific gravity varies from 1006 to 1024, on the average from 1010 to 1012. The opaque reddish yellow color varies very frequently in the same case, and often changes to a cherry or grayish red, brown, or blackish color, corresponding to the sediment. The reddish or brownish color is due to admixture with blood (nephritis hæmorrhagica). The microscope then shows a much larger number of blood-globules than in the ordinary form of nephritis; but in the darkest, blackish brown urine, they either appear as small, pale rings, or are entirely destroyed, and the urine merely contains free, dark-colored hæmatin (hæmoglobinuria). In addition, nephritic urine always contains a smaller or greater number of white blood-globules, desquamated renal epithelium, and longer or shorter hyaline casts covered with white and red blood-globules or epithelium. We may also very often find uric acid crystals, and, after the disease has lasted for a long time, fatty epithelium, free fat, and granular detritus, which adhere to the casts and testify to the increasing degeneration of the renal epithelium. The quantity of albumen may be slight on one day, and so abundant on the next that almost the entire amount of urine coagulates on boiling. Occasionally the urine is cloudy and brownish red at night, and contains a large amount of albumen and blood, while in the morning it is bright yellow and almost clear.

In one series of cases, the symptoms are restricted to the œdema and urinary changes. The general condition is very little affected, and the morbid phenomena may entirely disappear in two to three weeks. But we should always be prepared for relapses, though they usually have no other serious effects than to prolong the disease for one or more weeks. I would nevertheless advise you, even in these mild cases, to be on your guard and never to give an absolutely favorable prognosis, because dangerous symptoms, especially uræmia, may unexpectedly appear in the midst of apparent euphoria. Experience has also taught me to regard with distrust all cases of nephritis which begin with extensive and rapidly

increasing anasarca, especially if the excretion of urine is very scanty. Even in cases in which merely a few tablespoonfuls of urine are excreted, or complete anuria continues for days, the good general condition may deceive the inexperienced with regard to the gravity of the situation:

C. T——, aged nine. Sudden anuria two weeks after the development of scarlatina; the anuria continued seven days without a trace of œdema; pulse, 80 to 96. A tendency to somnolence, observed during the first few days, soon disappeared under the u e of purgatives. There was almost complete euphoria until the seventh day, when uræmic convulsions suddenly occurred, and death.

In mild cases, also, the dropsy, which is not infrequently entirely absent, and usually develops as anasarca, may occur into the cavities of the body. Ascites is then observed most frequently, while serous effusions into the pleural or pericardial cavities are rarer, and usually do not occur until the last stage of fatal cases. If ascites is alone present, the general condition, as I have often observed, may remain quite good, or, at the most, dyspnœal breathing results from encroachment upon the thorax.

A. R——, aged three and one-half years. Œdema of the face and feet; urine scanty, very cloudy, albuminous and somewhat hemorrhagic. Moderate ascites and marked meteorism, with elevation of the diaphragm. Dyspnœa; respiration, 60 to 70 per minute; no fever; organs of respirati n and circulation entirely normal. Complete recovery in three weeks, under treatment with purgatives and acetate of potash.

The prognosis is much more grave when the ascites is complicated with hydrothorax. The dyspnœa then increases more and more, occasionally occurs in the form of asthmatic attacks, and compels the patient to sit up day and night. I have very rarely seen œdema and hydrothorax develop without ascites, as in the following case, which will also serve to maintain our courage in the treatment of apparently hopeless cases:

M. S——, aged ten. Admitted May, 1877, with scarlatinous nephritis. Urine very scanty, very little albumen, no blood. Severe œdema of face, feet, back, and loins, but no trace of ascites; pale appearance—otherwise euphoria. From the middle of the second week, dyspnœal breathing, 50 to 60 respirations per minute. On examination, dulness and feeble breathing on both sides posteriorly, up to angle of scapula, extending, during the third week, to middle of scapula. Several severe attacks daily of asthma with cyanosis, coolness of the extremities and tip of nose, continuing several hours. No fever. Complete recovery in four weeks, under persistent treatment, at first with purgatives, then with infusion of digitalis and acetate of potash, in addition to repeated application of dry cups and mustard poultices.

Death is produced most rapidly by the sudden development of œdema of the lungs, more rarely œdema of the pharynx, the aryepiglottic ligaments and their vicinity (œdema glottidis). Orthopnœa and cyanosis, accompanied, in the first case, with diffuse crepitant râles; in the second, with a stenotic inspiratory and expiratory murmur, characterize this termination, which may not alone occur in cases attended with extensive dropsy of the skin and cavities, but also in those which run their course without dropsy or with very slight œdema.

More or less frequent vomiting of food and tough mucus, or of a watery fluid, is one of the most frequent symptoms of scarlatinous nephritis. This does not always possess the unfavorable "uræmic" significance which many ascribe to it, but often appears at the very beginning, or during the course of the disease, although the latter does not assume an unfavorable appearance, and must, therefore, be regarded as a reflex

symptom. Under these circumstances, the other symptoms, viz.: headache and somnolence, which give a serious significance to "uræmic" vomiting, remain absent. The bowels are usually constipated; much more rarely there is more or less profuse diarrhœa, occasionally frequent colic. Whether the thin evacuations are an accidental complication, or caused by excretion of constituents of the urine from the intestinal mucous membrane, remains undecided. In consideration of this possibility, however, I have always refrained from rapidly relieving the diarrhœa by constipating remedies. In one boy with ascites and slight hydrothorax, I observed almost constant tenesmus, without diarrhœa. Castor-oil proved useless, but relief was obtained by small hypodermic injections of morphine and the use of extr. opii. (0.005 t. i. d.). The experience that diphtheritic inflammation of the intestinal mucous membrane is occasionally found after nephritis, and may be more or less latent during life (page 207), cautions us to be careful in making a prognosis in these cases.

It is not true that the disease is always apyrexial if no complication be present. I will admit that in a series of quite severe cases fever may be entirely absent or the temperature be even subnormal (37.0°, 36.8°), and that in others more or less high fever may be due to another sequel of scarlatina, such as otitis, necrotic pharyngitis, phlegmons of the connective tissue of the neck or synovitis. But cases are not lacking in which nephritis alone is capable of producing a febrile condition of variable severity and duration. While occasionally there was merely an initial fever of 38°–39°, which disappeared in a few days, in others, an evening temperature of 38.5°–39.0°, with normal morning temperature, continued for two or three weeks, or ephemeral elevations of temperature to 39.0°, 40.0°, or even more occurred, the general course of the disease being apyrexial, and these elevations were sometimes associated with vomiting and increase of albumen or blood in the urine.

It is a noteworthy peculiarity of nephritis to produce inflammatory complications in various organs, which either give rise to fever, or increase it if already present. These complications, which may occur in every case, whether it begins with rapidly increasing dropsy or with apparent mildness, most frequently affect the respiratory organs. Pneumonia, bronchitis and pleurisy occur in many cases, even bilaterally, and were repeatedly the cause of the fatal termination, while milder bronchial catarrh, which is one of the most frequent complications of nephritis, does not influence its course unfavorably in any wise. In a boy, aged four, with extensive hepatization of the right lung, the previously yellow, cloudy urine assumed an exquisitely hemorrhagic color, under the influence of the pulmonary infiltration. In another child, who suffered from synovitis and nephritis after scarlatina, a pneumonia of the right lower lobe first developed, which gave rise to a pleuritic exudation, filling the thorax on that side (pneumo-pleuritis). This became purulent, and was cured in the sixth week by the radical operation. Pericarditis and endocarditis may also occur, and the latter may be so latent that it would remain undiscovered without an examination of the heart (page 182). It may be mentioned in this connection that, during the course of scarlatinous nephritis, the pulse occasionally becomes slow and even irregular, although no definite cause can be found. In one girl, aged twelve, the pulse sank to 48 beats per minute, and was very irregular, although no abnormality was discovered in the heart, and the general condition was not appreciably disturbed. In a few days the pulse rose to 60, then to 96 and became regular, and the nephritis was also cured at the end of a

week. I have repeatedly observed a similar reduction to 84 or 68, with or without irregularity, but in only one case did examination reveal a cardiac abnormality:

A boy, aged ten, admitted with scarlatina; two weeks later, nephritis. Pulse, which varied from 112–124 almost constantly, suddenly sank to 88, and became irregular, intermitting from 10–15 times per minute. A loud systolic murmur, which did not entirely mask the first sound, was heard at the apex of the heart. On the following day this had entirely disappeared, the pulse was 96–100, and entirely regular. Complete recovery.

The symptoms, which only lasted a day, cannot be explained, either on the theory of a material change in the endocardium or on that of anæmia. I would therefore abstain from a hypothetical interpretation, were it not for the fact that, in a number of cases which were associated with more or less distinct signs of uræmia, I have observed similar or more collapse-like disturbances of the heart's action, without any demonstrable structural changes. Thus, in a girl eight years of age, the previously normal pulse became strikingly slow (72–68) and irregular, when headache, nausea, vomiting, and somnolence developed, and again became normal, when these symptoms disappeared. In another child, a uræmic attack, which lasted the whole night, was followed by extreme failure of the heart's action (small, rapid, irregular pulse and heart-beat, cool extremities, frequent superficial respirations, 60–70 per minute), while the local examination merely showed a reduplicated first sound (galloping rhythm), which continued into convalescence. Similar symptoms were observed in a girl suffering from hemorrhagic scarlatinous nephritis, who, on the day before, had passed through a uræmic attack lasting several hours, immediately after which my attention was attracted by the extreme smallness of the pulse, reminding me of the collapse following diphtheria. This heart-failure is especially to be dreaded on account of the tendency of nephritic children to serous effusions, because the venous stasis produced thereby in the pulmonary circulation may readily cause œdema of the lungs. In a boy nine years old, continued slowness of the pulse (68–52), and arhythmia, with repeated vomiting, persisted for almost two weeks before uræmic convulsions occurred, at the beginning of which the pulse immediately rose to 120 and more.

In this case, as in a few others, the autopsy showed moderate hypertrophy and dilatation of the heart, which were perhaps the results of the nephritis, but were certainly not the cause of the slowness and irregularity, or the collapsed condition of the pulse. I therefore believe that these symptoms must be regarded as disturbances in the innervation of the pneumogastric nerve, which should be regarded as serious on account of their relations to uræmia, though they may also occur independently of it. They are not due to fatty degeneration of the heart, because, in the first place, they may disappear very rapidly, and secondly, because in one boy, who died in collapse with a temperature of 35.9°–34.8°, the hypertrophied left ventricle was markedly fatty,[1] although neither slowness nor arhythmia of the pulse had been present during life.

[1] I have repeatedly found hypertrophy of the heart, especially the left side, combined with dilatation. According to Friedlander's investigations (Arch. f. Physiol., 1881), it is rarely absent after scarlatinous nephritis. If this is true, it must gradually disappear, since in a large number of cured cases I have found the heart entirely healthy after the lapse of years, or was, at least, unable to detect the hypertrophy clinically.

The peritoneum is also occasionally the site of an inflammatory complication:

A boy, aged nine, suddenly affected, during the course of nephritis, with high fever, shortness of breath, distention and extreme tenderness of abdomen, nausea, vomiting and constipation. The danger was relieved in a few days by local bleeding, warm fomentations, and mercurials.

A boy, aged eight, with hemorrhagic nephritis and affection of the joints; followed by a remarkable succession of serous inflammations, at first acute hydrocele, then acute peritonitis, finally left pleurisy. At the autopsy considerable clear, yellow, serous fluid was found in the peritoneal cavity, and the serous coat of the intestines was extremely injected.

A boy, aged six, admitted May 24, 1876, with severe scarlatina, followed by nephritis. June 13th, pain and distension of abdomen, with increased fever. Collapse in a few days, and death on June 18th. Autopsy.—General purulent peritonitis.

Uræmia occupies the first rank among the grave symptoms of nephritis. Although generally preceded by marked diminution of the urinary secretion, or by complete anuria, examples are not lacking in which the amount of urine was not sensibly diminished, although uræmia occurred. Epileptiform convulsions occur, either without prodromata or preceded by vomiting, headache, somnolence, slowness and arhythmia of the pulse; the attacks are repeated in rapid succession for hours, complete coma or at least a somnolent condition being present in the intervals. On a couple of occasions I have also noticed violent excitement and delirium in the intervals. The temperature is usually elevated considerably during the seizures, even as high as 40.0° or more, while a marked depression occurs after their cessation, sometimes to 36.2°, combined with coolness of the extremities and extreme smallness of the rapid pulse. Death may occur very rapidly in such cases, as in one boy who became pulseless soon after the first convulsion, which lasted three minutes, and died during the second seizure. The convulsions may be limited to a few groups of muscles, or affect the entire body, but they are always associated with complete unconsciousness. Disorders of the special senses, for example, deafness, but especially amblyopia and amaurosis, sometimes remain after the attack, but they usually disappear in a few hours or days. Much more rarely the uræmic attack is preceded by amblyopia.

P. R——, aged nine, admitted June 6, 1878, with scarlatinous nephritis; irregular and slow pulse for several days; normal temperature. June 18th, vomiting and suddenly epileptiform convulsions, repeated seven times in five hours. First one limited to right side of face and right arm, and immediately followed by complete amaurosis, which soon disappeared. It reappeared after the second attack; in the afternoon sight was normal. Toward five o'clock, repeated epileptiform attacks, delirium, death in collapse. Post-mortem examination showed, in addition to dropsy of the cellular tissue and all the cavities, œdema cerebri, parenchymatous nephritis, moderate hypertrophy and dilatation of the heart, especially the left ventricle.

C. R——, aged eight. Scarlatinous nephritis with severe anasarca. Repeated vomiting, followed on the next day by apathy, headache, and very indistinct vision; pulse, 96–100. Severe convulsions and coma, consciousness returning in two hours. Sight perfectly normal on following day. Death at a later period from pleurisy and peritonitis, without return of uræmic symptoms.

E. K——, aged twelve; nephritis in third week after scarlatina. Uræmic convulsions, preceded by vomiting, in beginning of fourth week. Sensorium unclouded on next day, but almost complete amaurosis. Profuse secretion of urine on following day, and restoration of sight.

The cause of this disturbance of vision is as unknown as that of uræmia. It is not yet settled whether the dangerous cerebral symptoms are

produced by the retention of constituents of the urine, or by œdema of the brain due to increased pressure caused by the left ventricle. I attach no special importance to the discovery of œdema of the brain on autopsy, as this is sometimes found in cases which presented no uræmic symptoms during life. Graefe's conjecture that the amaurosis is due to more or less temporary œdema of certain parts of the brain, therefore, has no positive foundation. The reaction of the pupils in my last case was perfectly normal; whether this was true of the other two, I am unable to say. These cases were not examined with the ophthalmoscope.[1] There is no doubt, however, that the uræmia of acute nephritis is curable, despite the occurrence of amaurosis, and I may add that the uræmic symptoms present a more favorable prognosis in the scarlatinous form than in others. I have even repeatedly observed that the nephritis recovered more rapidly than usual after uræmia had occurred. Another series of cases, however, terminates fatally, or continues after the disappearance of the uræmic symptoms. I have had no personal experience with regard to the occurrence of partial paralysis, aphasia or hemiplegia, which have been observed by some authors after the uræmic seizure.

As a rule, the symptoms described above, the previous scarlatina, the chemical and microscopical constitution of the urine, will render the diagnosis certain. But there are undoubted cases in which, despite repeated examination of the urine, neither albumen nor the microscopic elements of nephritis can be discovered. The latter may be obscured by abundant uric acid sediments, but there is no doubt in my mind that they, as well as albumen, may be entirely absent for a time, and occasionally are only found during the last days of life, especially on the occurrence of uræmic symptoms. The autopsy, however, shows all the signs of well-marked, diffuse nephritis. I have observed at least half a dozen cases in which albuminuria was absent either entirely or at least for a few days. Complete absence of albuminuria has also been observed in the waxy kidneys of adults,[2] and also in a case of hemorrhagic scarlatinous nephritis. In the latter case, no symptom of nephritis (albuminuria, diminution in the quantity of urine, etc.) was present during life. The œdema of the face, and a few hyaline casts which were observed, could alone have aroused suspicion.

I have previously mentioned that there may be variations in the quantity of albumen present in the urine during the course of scarlatinous nephritis. We are scarcely ever able to ascertain a definite cause for these variations, but I have noticed on a few occasions, when the urine contained very little or no albumen, that a decided increase occurred after violent crying or vigorous movements (running, jumping), probably on account of the increased amount of blood in the kidneys. But those cases are inexplicable in which no albumen could be found in the urine for at least several days in succession:

Otto S——, aged twelve, admitted July 22, 1873, with œdema of the face and scrotum after scarlatina. Urine scanty, acid, contains neither albumen nor nephritic elements, merely amorphous uric acid. Negative results on next two days. July 24th, uræmic attacks during the night; next morning, cyanosis, feeble pulse, unconsciousness. The urine, removed by catheter, contained considerable albumen and numerous

[1] Œdema of the papilla has been occasionally found, in other cases, for example, one observed by Selberg and v. Graefe: no abnormality could be discovered. (Hirsch u. Virchow: Jahresber. f. 1867, II. S., 170.)

[2] Berlin. klin. Wochenschr., Nos. 22, 23. 1878.

granular casts. July 27th. death. Autopsy.—Exquisite parenchymatous nephritis, fatty liver, œdema pulmonum, broncho-pneumonia.

Paul S——, aged four, admitted March 8, 1876. Scarlatina two months previously, followed by nephritis. No œdema; a few spots of purpura; moderate diarrhœa. Urine scanty, but without trace of albumen or casts; death on March 11th.

Autopsy: severe hemorrhagic nephritis; fatty liver, chronic intestinal catarrh.

These at present inexplicable cases are calculated to make us careful in the assumption of scarlatinous dropsy without renal disease. Not merely single cases, but entire epidemics of this kind have been described. Legendre believes that the albumen, which may have been previously present in the urine in such cases, had disappeared at the time of examination, and I have often observed œdema and even ascites after scarlatina, in which the urine remained entirely free of albumen. But almost all these cases terminated favorably, and a post-mortem examination was refused in the only fatal one. Moreover, œdema of the feet occasionally occurs, after severe scarlatina which has lasted for weeks, and is entirely unconnected with the kidneys, but must be regarded merely as the result of weakness and anæmia and soon disappears under tonic treatment. Finally, in very severe scarlatinous inflammation of the skin, slight œdema of the face and feet may occur immediately after the disappearance of the redness as a local product of the inflammation of the integument. Such cases must be excluded as well as those in which œdema of the face is produced, during the course of scarlatina, by a phlegmon in the vicinity of the lower jaw or by severe rhinitis.

In the most favorable cases, scarlatinous nephritis continues two to three weeks, and often much longer. In a girl of twelve years, the œdema of the face, albumen and casts in the urine only began to diminish at the end of the tenth week and did not disappear until several weeks later, and in another girl who had scarlatina in January, 1875, albumen was still present in the urine toward the end of May, and on June 2d, fragments of granular casts were found in the urine. The danger of a transition into chronic nephritis is therefore not excluded, though I have observed but few cases of this kind. In a child of eight years, a severe chronic nephritis with marked œdema and characteristic urine could be traced back to a scarlatinous nephritis which had occurred the year before.

The opinions of physicians with regard to treatment vary greatly, an indication that nature effects more than our art. I will therefore describe those measures from which, after numerous trials, I have derived the best results. Above all I would advise you, as soon as albuminuria is discovered, even if it be only temporary, to keep the child in bed, and order a strict diet consisting chiefly of milk and articles made of milk. This diet also appears to me to be necessary when the disease is more developed. I also allow bouillon, but no meat, which would only increase the inflammation. If there is no diarrhœa, I begin treatment with a purgative (P. 7), which should be continued for two or three days, and then give acetate of potash (P. 41) which may be combined with decoct. chinæ (P. 42), if the patient is very weak and anæmic. In addition, three to four wineglassfuls of Wildunger water should be taken daily. I have never observed an unfavorable irritation of the kidneys from these remedies, if the potash salt is not given in too large doses. This is also true of digitalis, which I have very often used with good effect, either alone or in combination with acetate of potash (P. 22), in febrile as well as in apyrexial cases.

I have comparatively rarely been called upon to apply six to ten dry cups, or even wet cups to the region of the kidneys, and only when the quantity of urine was very small, or anuria was present, and high fever had developed. Venesection (about a cupful of blood) was formerly recommended as the best "diuretic" in such cases, and I remember some of my earlier cases, especially those complicated with inflammation of internal organs, in which this method produced surprising results. Perhaps I would have saved many a child, had I not been infected with the dread of venesection now prevalent. I acknowledge this openly, because I am resolved to employ it again in suitable cases, and will not be deterred by the presence of œdema or dropsy of the cavities.

Warm baths (at least 28° R.), followed by the pack in woollen blankets, enjoy great popularity. I have also employed them very frequently, and will not deny their efficacy, if they produce copious perspiration. In marked œdema, however, diaphoresis usually remains absent, or is insufficient, and even when there is no dropsy the baths are not infrequently entirely useless. Indeed, in many cases of hemorrhagic nephritis I observed increased blood in the urine after each bath. I therefore regard them merely as an experiment, which should only be made in uncomplicated cases. This is also true of the wet pack. Nor can I give an unqualified recommendation of pilocarpine. In order to produce profuse diaphoresis, it was sometimes necessary to increase the dose from 0.01 to 0.02, and repeated vomiting was then almost always observed; occasionally also when the dose was 0.01; in a couple of cases dangerous symptoms of collapse occurred, although a spoonful of strong wine had been previously administered. I have been repeatedly compelled to desist from this treatment, from which I feared dangerous depression of the heart's action. In other cases, in which the injections could be continued for a week or longer without any danger, and always produced copious diaphoresis, but usually very slight salivation, I have seen the dropsy rapidly diminish and the quantity of urine increase, though the amount of albumen remained almost unchanged. I do not believe that I have succeeded in curtailing the course of the disease by means of pilocarpine, but have often produced increased urinary secretion and rapid disappearance of the dropsy, results with which we may be satisfied.

The remedies recommended must be employed persistently for at least ten to fourteen days, and then alone would I advise you to use astringents if recovery does not progress. I first employ tannic acid, giving the preference to ergotin if the urine contains considerable blood. Both remedies appear to me rather to favor the elimination of water by the kidneys (P. 43 and 44). If they prove ineffectual after having been used eight to ten days, I administer liq. ferri sesquichlorati (P. 45), which is especially appropriate in the hemorrhagic form, but may also be recommended like the other preparations of iron, to relieve the anæmia which persists after recovery.

Inflammatory complications are treated according to their character. In threatening or developed uræmia I have repeatedly had excellent results from wet cups to the neck, two to six leeches behind the ears or on the temples, the bites of which should not be allowed to bleed after removal of the leech; an ice-bag to the head, combined with an active purgative of inf. sennæ comp. and syr. spin. cervin. (P. 7); or, if this is rejected, enemata of equal parts of water and vinegar. When the uræmic convulsions are very severe and prolonged, inhalations of chloroform should be tried, as in other epileptiform attacks (page 66), and the col-

lapse which occasionally occurs after the seizures requires the abundant administration of wine and subcutaneous injections of camphor (P. 14). Since the recent recommendation of pilocarpine in uræmia by Prætorius,[1] I have employed it successfully in two cases (two injections daily of 0.005 to 0.01). But as I have repeatedly been successful with antiphlogistic measures, I am not at present very enthusiastic with regard to pilocarpine, and reserve my judgment.

Scarlatinous nephritis, as I have remarked, almost always occurs as a sequel of the exanthem, at least in its clinical appearance. Although in autopsies upon scarlatina patients, who died in the first and second weeks of the disease with malignant symptoms, cloudy swelling of the renal cortex or higher grades of parenchymatous nephritis are not infrequently found, their symptoms usually disappear in the general terrible symptomatology. An examination of the urine can alone give any information in such cases. For example, in two children aged respectively six and nine, I found, on the fourth day of scarlet fever, which was associated with "diphtheritic" pharyngitis and typhoid symptoms, cloudy, very scanty urine, containing abundant albumen and lymph-corpuscles, and after death severe parenchymatous nephritis was found present. In a girl, aged eleven, marked œdema and rapidly increasing ascites, with abundance of albumen in the urine, developed on the fifth day of scarlatina, coincidently with broncho-pneumonia, and terminated fatally in the beginning of the second week. In such a case we must assume a rapid and severe exacerbation of the "cloudy swelling," which develops in consequence of very high fever (page 240), but usually resolves, and by no means justifies a fear of a nephritic sequela. I have seen convalescence undisturbed in several cases of scarlatina which presented very high temperatures and albuminuria lasting a few days during the height of the disease.

In very severe cases of scarlatina, which begin with the symptoms of heart-failure, the albuminuria may be attributed, in my opinion, to stasis of the renal veins produced by weakness of the heart.

P. P——, aged seven, taken sick with scarlatina, January 24, 1873. January 26th, pulse 140, very small; on the next day irregular and scarcely perceptible; hands and feet cool, eruption present, and buccal mucous membrane cyanotic. Urine scanty and albuminous. January 28th, pulse more perceptible and eruption redder under use of stimulants (wine, musk). January 29th, pulse stronger—120, eruption of normal red color, urine abundant, and no albumen. The albuminuria and cyanosis of the eruption and buccal mucous membrane must be regarded as the result of venous stasis in the kidneys, since all these symptoms disappeared when the circulation became normal.

Acute diffuse nephritis of children is a sequela of scarlatina with such overwhelming frequency that you should always think at first of this disease, even though it be denied by the family of the patient. Mild cases of scarlatina, with slight, temporary redness of the skin, are quite often overlooked; and not until a later period, when nephritis develops, do the parents remember, on being questioned by the physician, that the child, two to three weeks previously, had slight fever for a few days, complained of the throat, and perhaps had a few "red spots." In such cases the remains of desquamation, especially on the hands and feet, often furnish proof that scarlatina had really been present.

However, scarlatina is by no means the only cause of nephritis in

[1] Jahrb. f. Kinderheilk, XV., S. 375. 1880.

childhood. Next to it, diphtheria plays an important part. It often produces nephritic symptoms during its course, more rarely during convalescence; but I shall reserve its discussion until my remarks on diphtheria. Rubeola more rarely acts as a cause of nephritis. Although cloudy swelling or parenchymatous nephritis is occasionally found in autopsies upon rubeola patients, as in all severe infectious diseases (Reimer found them twelve times in 51 cases), their clinical manifestation during the course, or as a sequela of measles, must be regarded as extremely rare.[1] I have had but one positive case which I have observed from the beginning of the measles. It has, however, been proven by the observations of Malmstein, that nephritis, even of a hemorrhagic character, may occur during the first few days of an attack of measles.

In very rare cases nephritis is due to intermittent fever. In a girl, aged six, who was cured by quinine after three paroxysms of intermittent fever, the urine, a week afterward, contained abundant albumen, hyaline casts, and blood-corpuscles, but its normal character was restored after the continued administration of quinine for a week. I have also observed two other similar cases.

In one case I observed similar symptoms as the sequel of angina parotidea.

C. S——, aged six, healthy, suffered from parotitis in the diminishing stage of whooping-cough. A week after its disappearance œdema of the face was observed, with bloody, albuminous urine. On my first examination these symptoms still continued. Slight fever at night, with complete euphoria; urine abundant, greenish brown, with scanty sediment, containing some blood and considerable albumen, blood-globules, and epithelium; no casts. Complete recovery in a week.

As I observed œdema of the face and feet, with albuminuria, in a child during whooping-cough, the question arises whether the albuminuria, in the case reported above, was due to the whooping-cough or parotitis. The marked venous stasis, which occurs in all parts, and therefore also in the kidneys, during severe coughing paroxysms, may undoubtedly predispose to dilatation of the vessels and exudative processes; but as the whooping-cough had very markedly diminished when the renal symptoms appeared, the dependence of the latter on the parotitis is rendered almost certain. I will therefore examine the urine after every attack of parotitis in order to shed some light on this question.

Nephritis occurs much more often in children as the result of a severe cold or wetting. Thus, I observed it in a girl, nine years old, who fell into the water while her body was overheated, and in a boy, eight years old, who was caught in a heavy rain. Probably other cases, in which I could find no cause, must be attributed to similar circumstances, which have either remained unknown or have been intentionally concealed.

Finally, I must direct your attention to a form of "artificial" nephritis, which develops as the result of certain therapeutical measures. It is well known that the internal use of powerful diuretics, especially turpentine and cantharides, may produce albuminuria, and even nephritis, and this effect is also attributed by some, with what justification I do not know, to large doses of chlorate of potash. Much less consideration is paid to the fact that an analogous effect may be produced by the external use of these remedies. In an epileptic girl, aged ten, who had had a cantharides plaster applied daily for a month, I found albumen and hya-

[1] Kassowitz: Oesterr. Jahrb. f. Pädiat., I., 80. 1874.

line casts in the urine, which disappeared a few days after the cantharides was discontinued. But special attention should be paid to those cases in which balsams or tar are employed as ointments in chronic diseases of the skin. The more carefully the examination is made in such cases, the more frequently will we find albumen and formed elements in the urine after a certain lapse of time, usually after several weeks, or even later. This was observed in several cases of chronic eczema, which had been treated with tar ointment (pix liquid. 1 : vaseline 10.0).[1] On the other hand, applications of tincture of iodine, which are said by French authors[2] to exercise a similar influence very rapidly, are almost always harmless in this respect if confined to small portions of the skin. In one case, in which tincture of iodine had been applied four times to a quite extensive surface, severe nephritis developed at the end of two weeks, associated with œdema, considerable albumen, casts, and epithelium in the urine, and threatening uræmic symptoms, but it must be added that treatment with tar ointment had been previously used. On two occasions, also, I found nephritic symptoms develop one to two weeks after the cure of scabies by Peruvian balsam. In addition to blackening of the urine, albuminuria and true nephritis may also occur after the external application of carbolic acid. I have observed but one case of this kind, and this sufficed to render me cautious in the use of this article during childhood.

A. S——, aged six, admitted June 14, 1879, with chronic eczema of left forearm. Fomentations to arm of five per cent. solution of carbolic acid, applied continuously for four days. June 19th, inunctions with vaseline; later, application of plaster-of-Paris splint to prevent scratching. July 7th, extremely scanty excretion of urine, abundant albumen, a few red-blood globules, hyaline and partially granular casts. July 10th, œdema of the feet and abdominal walls; œdema of face in next few days. Treatment with purgatives, then tannin and wet packs, which were always followed by profuse diaphoresis. Complete recovery did not occur until the beginning of August.

In a series of cases, however, it will be impossible to determine positively the cause of the nephritis, despite all our efforts. The etiology is entirely unknown in the few cases which I have observed in the new-born and in very young children. I have called attention, at a previous period, to the fact that albumen may be found in the urine of the new-born, at least temporarily, and that it was undecided whether this was due to the irritation of the uric acid concretions in the uriniferous tubules. Although this extremely small quantity of albumen usually disappears after the first ten days, cases occasionally occur in which nephritis, attended with serious consequences, develops during earliest infancy. I do not refer to the cloudy swelling of the cortex observed very often in marasmus, and which must be regarded as a nutritive disturbance of the epithelium, but to a clinically recognizable disease, as, for example, in the following case:

C. K——, aged five weeks, admitted March 24, 1874; moderate marasmus. March 25th, severe œdema of face and extremities (temperature, 36.4°). Normal passages. Urine exceedingly scanty. March 27th, dyspnœa, cyanosis, dulness over lower part of thorax on both sides. March 29th, death. Autopsy.—Diffuse parenchymatous nephritis and serous effusion into the pleural, pericardial, and peritoneal cavities.

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[1] Jacubasch: Charité-Annalen, Bd. VI.

[2] Badin: De l'Albuminurie Consécutive aux Applications de la Teinture d'Iode chez l'Enfant. Thèse. Paris, 1876.

The cause could not be ascertained in a few other cases of chronic nephritis, which occurred during the first and second years of life. No connection with syphilis could ever be demonstrated, and in one suspicious case, which was treated antisyphilitically, the treatment remained entirely unsuccessful. I would, however, advise you to keep the possibility of syphilitic origin in mind, in view of a case of this kind successfully treated by Bradley.[1] Even at this early age, œdema constitutes the chief symptom of chronic nephritis. On the whole, the chronic form presents no noteworthy differences in childhood from that observed in adult life. But I do not doubt that many cases of chronic nephritis, which are said to occur in tuberculous and scrofulous children, or those exhausted by malaria or syphilis, have been falsely interpreted, and were really examples of amyloid degeneration. The diagnosis of the latter is easy if the liver or spleen is distinctly swollen and is associated with marked cachexia from syphilis, suppuration of bone, tuberculosis, etc., œdema of various parts of the body, and albuminuria. If these symptoms, especially the albuminuria, are absent, as sometimes occurs, a suspicion may be entertained, but no diagnosis can be made.

I will make use of this opportunity to add a few words concerning the dropsical symptoms, which may occur in children, without the presence of albumen or of the microscopical characters of nephritis in the albumen. In the description of œdema neonatorum (page 27) we became acquainted with a series of causes which produced it. The same conditions hold with regard to the œdema occurring in older children. During the first two years of life I have seen, with special frequency, œdema of the dorsal surfaces of the hands and feet, the legs, cheeks, and eyelids. It has been already stated that such cases may be due to nephritis or to waxy degeneration of the kidneys. In my cases, however, the urine very rarely presented such a character; it was much more often entirely free from albumen, though very scanty and often containing abundant uric acid salts. I would therefore entirely exclude the assumption of nephritis, did not my experience with regard to the absence of albuminuria in this disease (page 247) make me suspicious. In fact, in a case of this kind, in which there was ascites and extensive œdema of the abdominal walls, but no albumen had ever been present in the urine, both kidneys were found very firm, and the cortex indurated from hyperplasia of connective tissue. Dickinson[2] also observed nephritis in two very young dropsical children, whose urine had never contained albumen. We must not be satisfied with the gross appearance of the kidneys, but resort to a microscopical examination.

In addition, œdema not infrequently occurs in children, as in adults, which is entirely independent of any renal affection, and is due to exhausting diseases, especially phthisis, protracted diarrhœa and dysentery, or severe affections of the blood, leukæmia and pseudo-leukæmia. In a large proportion of these cases, the immediate cause of the œdema is the weakness of the heart and the venous stasis dependent thereon; this may be restricted by "marantic" thrombosis of the larger veins to individual parts—for example, to a lower limb. The numerous atelectases of the pulmonary tissue, which occur so readily in exhausted children, as the result of the diminished power of inspiration and the complicating bronchial catarrh, must also favor the development of œdema by the stases in

[1] Hirsch-Virchow: Jahresber. f. 1871, II., S. 176.
[2] Ibid., II., S. 175.

the veins of the body to which they give rise. This category also includes the dropsy which accompanies diseases of the heart in children as frequently as in adults.

It has been previously mentioned that inflammatory diseases of the skin, especially erysipelas (page 27), may give rise to œdema, and I have occasionally observed it after urticaria or erythema. The preceding disease of the skin may have been entirely unnoticed, and only the subsequent swelling of the eyelids or other parts arouse the fears of the parents. Occasionally all etiological factors, to which the œdema could be attributed, may be absent, and we then usually accept the idea of a cold:

G. S——, aged nine, admitted October 8, 1878, with œdema of the face, scrotum and foreskin, which is said to have been present for twenty-four hours. Complete euphoria; cause entirely unknown. Urine normal. October 28th, patient dismissed cured.

Occasionally these œdemas occur periodically. I observed this, for example, in a four-years-old girl, who presented œdematous swelling of the dorsal surfaces of the feet, the hands and face three or four times in the course of about three months. It lasted about a week, and was associated with general malaise, ill-humor, once with vomiting; but repeated examination of the urine and of all the organs showed nothing abnormal. As the etiology was entirely obscure, and the pallor of the child suggested anæmia, iron and quinine were ordered (the latter on account of the possibility (?) of malarial infection) and produced rapid recovery.

Among the local causes of œdema must be mentioned compression of some of the veins, as occurred, for example, in the following case:

A child, aged eighteen months; autopsy, June 7, 1873. During life, marked œdema in front of and behind the right ear, pushing it away from the head, as in carious disease of the petrous portion of the temporal bone. Autopsy showed miliary tuberculosis of the serous membranes, spleen, liver and lungs, cheesy degeneration of the bronchial glands, and compression of the right external jugular vein by an enormous collection of glands. The œdema disappeared immediately after death.

II. Disturbances of Urinary Excretion.

Apart from acute diffuse nephritis and tumors of the kidneys (page 238), diseases of this organ, in children, present nothing characteristic. I can therefore confine myself to a few remarks on the morbid disturbances of urinary excretion, which we meet with in children with special frequency. I will refer at first to congenital hydro-nephrosis, which is produced by obliteration of the ureters, is almost always unilateral, and is only exceptionally recognizable clinically. Among the most rare cases belongs that of a child, three weeks old, who was admitted to my wards with two large fluctuating tumors, which were dull on percussion in the hypochondriac spaces. No urine was passed in about ten days, but it is said to have been excreted during the first period of life. By puncture I removed from the tumor on the left side a quantity of hemorrhagic serous fluid, and post-mortem examination revealed bilateral hydro-nephrosis and cicatrical obliteration of both ureters at their origin from the pelvis of the kidneys. This obliteration appeared to be quite recent on the right side. The fluid removed by puncture was not derived from the hydro-nephrosis itself, but from a cyst filled with bloody serum, surrounding the kidney.

I will require your attention a little longer with regard to the disturbances produced by lithiasis.

The uric acid infarct, which is constantly present in the new-born, is washed away, as a rule, in the first few weeks of life, without producing any disturbances, but occasionally this occurs very slowly, and I have even found remains of the infarct in children seven to eight weeks old, partly in the lumen of the tubules, partly upon the papillæ, or perhaps lying in the pelvis as small reddish crumbs. The irritation of these small concretions may very early give rise to disorders of micturition, which at first remain unnoticed, and at all events are difficult to interpret, because examination of the urine is extremely difficult at this age. Children a few months old have not infrequently been presented, who cried violently either before or during each act of micturition, only passed a few drops of urine, despite severe straining, and evinced great restlessness, so that there could be no doubt of the existence of pain, while the general condition remained undisturbed in the intervals. The spots left on the diaper by the urine are often darker than in the normal condition, the edges are somewhat reddish, a few yellowish red crumbs, like grains of sand, being occasionally found. Under these circumstances the acidity of the urine may not alone cause burning in its passage through the urethra, but also cause redness of the meatus and the inner surface of the labia and their vicinity. Similar symptoms are not infrequent in older children, in whom we do not have to deal with the delayed excretion of uric acid infarcts, but with new-formed uric acid concretions (gravel), which must generally be regarded as the result of perverted nutrition.

In all cases of dysuria in young children, we are readily embarrassed on making an attempt to explain them. The diagnosis is only undoubted when small uric acid concretions are found in the diaper, or in the urine of older children; and then, as in adults, there is occasionally catarrhal irritation of the pelvis of the kidney, which, in its turn, favors the formation of uric acid gravel, and may lead to symptoms which correspond exactly to those of calculous pyelitis of later life.

F. R——, aged five months, presented January 16, 1874. Two weeks previously, vomiting and repeated twitchings in the limbs. Crying before and during micturition, the urine being passed in drops, despite severe straining. Urine pale yellow, very cloudy, abundant albumen, no casts, but a large number of pus-globules and quite numerous uric acid concretions. Further course unknown.

I have previously (page 73) reported the similar case of a child, five months old, who discharged small concretions and suffered at the same time from reflex eclampsia and contracture of numerous groups of muscles. If this condition lasts for a long time, the catarrh of the pelvis may gradually spread through the ureters to the mucous membrane of the bladder, and as vesical catarrh notably favors the formation of calculi, may also give rise to the development of vesical lithiasis. In comparison with later life, vesical calculi occur by no means infrequently in children, even during the first years, and it is therefore necessary to examine the urethra and bladder under chloroform in every case of chronic dysuria, whether associated with catarrh of the bladder or not. The discharge of urine is occasionally stopped entirely, and only a few drops are passed despite the most severe straining, during which prolapsus ani not infrequently occurs. I have even observed retention of urine for thirty-six to forty-eight hours, the bladder being enormously distended and extending above the symphysis, and evacuation by the catheter being neces-

sary, while at other times there was constant trickling of urine from the urethra. In this event, the vicinity of the genitalia was continually wet, and the decomposing urine not alone produced a disagreeable smell, but also caused an inflammatory, œdematous condition of the foreskin, penis, and scrotum. In older children the penis was found to be unusually long and developed, probably in consequence of the numerous manipulations to which the children resort, and there is also often a tendency to prolapsus ani, which I regard as a symptom of vesical lithiasis at this age which should not be undervalued. Occasionally a concretion which has passed into the urethra, and there remained, produces complete retention of urine and œdematous swelling of the genitalia.

A. L——, aged two, admitted November 28, 1877. Quite well-nourished, but pale. Complete retention of urine for two days; slight redness and marked œdema of penis, scrotum, and perineum. Foreskin cannot be retracted on account of phimosis. Abdomen distended, hard, and sensitive—the dilated bladder extending a few fingers' breadth above the symphysis. In order to introduce the catheter, it was first necessary to operate upon the phimosis, during which a calculus, which completely occluded the meatus, was removed. The catheter, when introduced into the bladder, removed a quantity of cloudy urine. The œdema rapidly disappeared under applications of lead-wash, but November 29th vomiting and diarrhœa occurred during the night, with rapid collapse. December 1st, death. Autopsy.—In the bladder a sulphur-yellow stone, as large as a hen's egg, completely filling the organ. Similar calculi, from the size of a pea to that of a bean, in the pelvis of the left kidney; right kidney normal.

But dysuria also occurs in childhood, independently of any calculous formation in the kidneys or bladder. The passage of very acid, concentrated urine through the urethra—for example, in high fever—may cause pain during micturition, which is manifested by crying, and, in older children, by definite complaints. Urine loaded with uric acid salts, may also give rise to attacks similar to those of renal colic in adults. In two children, three and four years old, I observed violent attacks of pain in the abdomen, occasionally accompanied by chill and fever, which lasted for several hours, at times recurred for days in succession, and always ended with the secretion of a cloudy urine, which was loaded with urates and was distinctly albuminous; while in the intervals between the attacks, which often lasted months, the general condition was undisturbed, and the urine entirely normal. As nausea and constipation were also present during the attacks, the diagnosis of intestinal colic was first made, until finally the condition of the urine attracted attention and led to an examination. A discharge of gravel, which may give rise in adults and children to inflammatory processes in the pelvis of the kidney, and perhaps in the parenchyma, was not noticed in these cases, and this renders so much more noteworthy the occurrence of temporary albuminuria in attacks of pain, which could only have been produced by the irritation of morbidly changed urine. The continued administration of Vichy, Wildunger, Bilin waters, or a solution of bicarbonate of soda (3 : 100), produced the same good results as are usually obtained in later life under similar circumstances.

I need scarcely add that an examination of the external genitals should never be omitted in any case of dysuria during childhood. You will not infrequently discover a phimosis which interferes more or less with the discharge of urine, and retains portions of the latter behind the foreskin, where it may decompose and give rise to an inflammatory condition of the prepuce, with painful dysuria. I have also observed, in a child three years old, a fully developed urethritis with severe dysuria,

which appears to have been produced by manipulations on the part of another boy. Anomalies of the meatus are found much more rarely. Thus, in a boy aged seven months, I found, instead of the meatus, a shallow groove, while the urine emerged in three thin streams from three punctate openings which were situated alongside the groove. As a matter of course, operative treatment can alone be considered in such cases. This is also true of the occasional adhesion of the labia minora in little girls, like the similar adhesion of the foreskin in boys. It is almost constant in the first period of life, but sometimes persists to the end of the first year, can usually be torn by the handle of the scalpel, and rarely requires an incision. In a few cases this adhesion appeared to me to be the cause of dysuria, which disappeared after the separation of the labia from one another; in others, examination showed inflammatory redness of the introitus and meatus, with increased secretion of mucus, which rendered the excretion of urine painful.

But your assistance is much more often required by another affection; I refer to enuresis, especially the nocturnal variety. Not only children a few years old, but those who have passed the second dentition and are approaching puberty, not infrequently suffer from this complaint, concerning whose causation we know so little that there is a certain justification for the doubt whether this is a diseased condition or the result of habit. "Wetting the bed" occurs almost as often in girls as in boys, and either during the first hours of sleep or later, perhaps toward morning, sometimes every night, sometimes with intervals of days, or even weeks, which are especially observed during the course of acute diseases. The various views of physicians with regard to the cause of this disorder, but especially the variety of remedies recommended, indicate that it is not always due to one and the same cause. I would advise you never to omit an examination of the urine, because cases of diabetes mellitus and chronic nephritis are known, which were first manifested by nocturnal incontinence. Such a case, however, has never come under my observation, and I must therefore regard this cause of enuresis as extremely rare. The immediate cause of the disorder is either atony of the sphincter vesicæ, or a spasm of the detrusor urinæ, which is capable of overcoming the contraction of the sphincter, which is less vigorous during sleep. The first form appears to me to be rarer, and, although not constantly, is usually associated at times with enuresis diurna. When this is so, we should suspect vesical lithiasis, and an examination of the bladder should not be omitted. In otherwise healthy children, the assumption of atony limited to the sphincter vesicæ is always somewhat forced, and only appears to me to be justified in individual cases in which there is a condition of marked weakness, for example, after typhoid fever or some other severe disease, or in an organic disease of the spinal cord. A characteristic example of the latter variety, which had existed from early childhood, was presented by a boy, aged thirteen, who had a flat, doughy tumor, about as large as a hen's egg, at the lowest part of the dorsal vertebræ, in the middle of which a defect of the spinous process could be distinctly felt, and was probably the sac of a spina bifida[1] filled with fat and granulation tissue. This boy also suffered from involuntary evacuation of fæces whenever the latter were not solid. Treatment with tonic

[1] Similar cases are reported by Blake (Amer. Journ. of Obstet., p. 146. 1878). In one of them the euphoria was undisturbed, with the exception of the diminished function of the bladder and rectum.

remedies (iron), with ergotin and strychnine (internally or subcutaneously), is advisable in these atonic cases alone, though I have never observed any good effects from their use. The recently recommended use of electricity, which is applied directly to the rectum, in order to produce a reflex effect on the sphincter vesicæ,[1] acts, if at all, through its psychical influence, to which I will soon refer.

In nocturnal enuresis the urine is always discharged in a stream, and is either passed during sleep or in a half-waking condition. A reflex irritating effect upon the detrusor urinæ appears to be produced, which acts so much more vigorously because the action of the will on the sphincter is diminished during sleep. The physician should endeavor to determine the situation from which this reflex stimulus arises, and occasionally congenital phimosis, stricture of the urethra, irritation of ascarides, fissure of the anus, onanism, vulvitis, can be detected, after the removal of which the enuresis ceases. Renal and vesical calculi, and even an excess of urates or phosphates in the urine may exercise such an irritation, and these abnormalities must then be treated. In the majority of cases, however, none of these morbid conditions can be discovered. We then assume a hyperæsthesia of the neck of the bladder or its entire mucous membrane, and sedative remedies, especially belladonna (extr. belladonnæ, 0.05–0.01) are then recommended. Others advise the frequent introduction of bougies into the urethra, or cauterization of the neck of the bladder. I will not deny the possibility of success from the use of these agents, but it should not be forgotten that the psychical impression produced thereby must also be taken into consideration, especially as the possibility of an acquired habit cannot be excluded. This is shown by a couple of cases of incontinence of fæces, which recovered with surprising rapidity after one or two hypodermic injections of ergotin near the anus, and in which the effect could have been merely of a psychical character. Many cases of rapid recovery of nocturnal incontinence by painful measures, or by faradization, must be interpreted in the same manner. Under all circumstances, drinking should be restricted before going to sleep, and dorsal decubitus avoided as much as possible, for which purpose we may apply a brush, the hairs of which will come in contact with the skin of the back. Many cases finally recover spontaneously, some not until the development of puberty.

III. Diseases of the External Genitals.

I will first refer to a circumstance which, despite the articles of Bokai and Schweigger-Seidel,[2] is still unknown to many physicians. I refer to the adhesion of the inner layer of the prepuce to the glans by a more or less firm tissue, which permits only partial retraction of the foreskin, even if no trace of phimosis is present. This adhesion, which must be regarded as normal during the first period of life, may be present in children four to five years old; it is composed of ordinary polyhedral epidermal cells, and is probably due to the fact that the cells of the rete Malpighii which pass to the surface do not become horny in the normal manner, but remain filled with protoplasm and secure adhesion of the two surfaces. The occasional adhesion of the labia minora, which I referred to above

[1] Ultzmann: Central-Ztg. für Kinderkr., I., No. 22.
[2] Virch. Arch., Bd. XXVII., Heft 2; Jahrb. für Kinderheilk., Bd. V., Heft 1.

(page 257) as a cause of dysuria, must be regarded, on account of its rarity, as a pathological process, which develops in the same manner as adhesion of the prepuce, but only when the labia are in contact with one another along their entire length.[1]

Cryptorchia, *i.e.*, the absence of one, more rarely both, testicles from the scrotum, is observed about ten times among one hundred new-born. In the normal condition the testicle passes through the inguinal canal in the ninth month of fœtal life, but occasionally this does not occur until after birth. Unusual narrowness of the inguinal ring, or inflammatory processes which produce adhesions of the testicles, may prevent the descent of the latter, so that they may remain in the abdomen or in front of the inguinal ring. The diagnosis of cryptorchism is easy: the scrotum is found to be small and empty on one or both sides, while the testicle (which is generally of normal size, but occasionally enlarged to the size of a pigeon's egg from serous effusion into the vaginal sheath) can be felt more or less distinctly either in front of or behind the external inguinal ring, and is somewhat movable if loosely fixed. The right testicle is more frequently absent from the scrotum than the left. If the descent is delayed until the end of the first year, the formation of an inguinal hernia is always to be feared. Strangulation of the testicle in the inguinal canal, associated with severe pains and inflammation, which necessitate absolute rest and antiphlogistic measures (ice-bag, leeches), rarely occurs in cryptorchism. Finally, it may be mentioned that the testicle may be drawn up so far by the cremaster muscle, when the scrotum is firmly contracted, that it is felt in front of the inguinal ring, and only after the scrotum relaxes can we convince ourselves that cryptorchism is not present. Nothing can be done to relieve this condition.

With the exception of hydrocele, diseases of the testicle are rare in children. I would remind you of the previously mentioned (page 45) enlargements due to syphilis which appear to be more frequent than medullary sarcoma and enchondroma, which are occasionally observed. Tuberculosis and cheesy degeneration of the testicle are rare in comparison with their enormous frequency in other parts; I have only observed a few cases in children between fifteen months and seven years old. The swelling usually affected one or both epididymes, which were hard and nodular, the testicle being rarely involved; they attained the size of a walnut or apple, and enlarged still further from the development of hydrocele; from time to time the tumors became inflamed and discharged cheesy pus. Tuberculosis of other organs or caries of bones was almost always present. Local treatment (incision and poultices) is alone appropriate when suppuration occurs, while otherwise we must confine ourselves to the administration of cod-liver oil, iodide of iron, and salt baths.

In little girls, vaginal hemorrhages are occasionally observed during the first days of life, are apparently due to the epithelial desquamation going on at this time, and are usually borne without any detriment. Early menstruation at the age of eleven or twelve years is not rare, but its occurrence before this time—for example, at the age of three or nine years,[2] is exceptional. But every hemorrhage from the genitalia of little girls must not be regarded as an evidence of premature menstruation; it is occasionally due to papillomata of the vulva and vagina, or to polypoid

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[1] Bokai: Jahrb. für Kinderkrankh., S. 163. 1872.

[2] Oesterr. Zeitschr. f. Päd., VIII., Bd. I., S. 26, 1877. Deutsche Zeitschr. f. prakt. Med., S. 487, 1878.

thickening of the urethra. A dark red, easily bleeding swelling then projects from the meatus, and may become so large as to separate the vulva. The vesical tenesmus which is present may be readily overlooked, and the hemorrhage first attracts the attention of the mother. In two cases of this kind, recovery was effected in a few weeks by cauterization of the swollen and prolapsed mucous membrane of the urethra.

Vulvitis is undoubtedly the most frequent affection of the genitals in little girls. As a rule, they come under observation several days or weeks after the discharge has been noticed, and, on examination, you find a purulent fluid flowing from the genitals, often dried into thin crusts on the inner surface of the labia and thighs, and producing stiff, greenish yellow spots on the underclothing. The mucous membrane of the introitus vaginæ is reddened to a variable degree, and the labia are not infrequently somewhat swollen and sensitive; dysuria is often present, and many children refuse to walk on account of the pain produced by the friction of the parts.

We are at once inclined to suspect an attempt at rape in such cases, and I have seen a number of children from the age of four to ten years, in whom either a forcible attempt had been made to introduce the penis, or lewd manipulations with the fingers had been performed. Rupture of the hymen rarely occurred, as the smallness of the parts prevented the complete entrance of the penis. Nevertheless, I would advise great caution in making such a diagnosis, as it not infrequently happens that the mother, who mentions this cause, either deceives herself or wilfully misinforms the physician; only in undoubted cases should this be accepted, as there are also other causes of vulvitis. Even uncleanliness, and the constant accumulation of sebum between the labia, may produce a condition of irritation; a still greater effect is produced by frequent manipulations of the genitalia, either by the children themselves or by their playmates, and by the irritation of ascarides in the rectum, which cause scratching of the anus and its neighborhood (page 218). When all these causes are absent, we may think of the possibility of a local cold, which is, however, scarcely ever demonstrable with certainty.

The previously mentioned symptoms of vulvitis are not infrequently associated with erosions and ulcerations of the labia minora or majora, which increase the suspicion of syphilitic infection. But even when rape had been performed, the affection usually produced the impression of a traumatic inflammation, and there are only a few exceptional cases of true chancre in girls eleven to thirteen years old, who had already worshipped at the shrine of Venus. Occasionally groups of herpes vesicles are observed bilaterally, extending along the perineum to the anus.

The treatment consists of absolute rest, frequent fomentations with lead-wash, and, when this proved unsuccessful, vaginal injections of tannin or alum (5 : 100), sulphate of zinc (2 : 100), or nitrate of silver (1 : 100). In order to prevent the children from scratching the genitalia, it is not infrequently necessary to bind their hands. Nevertheless, several weeks may elapse before the affection is entirely cured.

In another series of cases, a more or less hard, inflammatory swelling occurs in one labium majus, which rarely involves the other side or spreads to the mons veneris. The mucous membrane of the introitus and the external integument of the labia may be entirely intact and the general condition be undisturbed, and in only one case did I observe urticaria at the same time. I could never positively ascertain the cause of these swellings. They usually resolved in one or two weeks under continued

fomentations of lukewarm lead-wash, or they suppurated and required early incision.

Another variety of inflammatory swelling presents a marked tendency to gangrenous destruction. Gangrene of the vulva may occur from a simple, very hard swelling of the labium, the mucous membrane and cutis being intact; but it is more often the result of an ulceration which penetrates more or less deeply from the surface of the labium, and is covered with a diphtheritic deposit. The following case belongs to the first category:

M. K——, aged two, admitted February 26, 1874. Hard swelling of left labium majus for last four days; it is dark red, and, on inner surface, shows a deep loss of substance, with black edges; high fever, otherwise everything normal. Application of actual cautery to necrotic parts and vicinity; on the next day, fever almost gone. March 4th, desquamation of the gangrenous tissue, leaving a clean surface. Complete recovery in a short time.

The rapid effect of the actual cautery favors the view of a local, perhaps traumatic cause of the process. The second form is observed more frequently; a dark, livid redness of the labium and adjacent mucous membrane first appears, the epidermis is raised in vesicles or desquamated in shreds, and finally, dirty gray or bluish red ulcerations are formed, especially on the inner surface of the labia. The latter are œdematous, or hard as stone, infiltrated and swollen. The ulcers are soon covered with a yellowish gray or blackish green gangrenous layer, and rapidly grow deeper, so that they not alone erode large portions of the labium majus, but may also spread to the labium minor and introitus vaginæ. It thus presents a perfect analogy to noma of the cheek (page 191), and its causes are similar, viz., general severe cachexia, previous infectious diseases (especially measles, scarlatina, and typhoid fever), and gangrenous processes in other parts of the body (pulmonary gangrene). Under such conditions the prognosis is very bad, but hope should not be abandoned when the circumstances are less unfavorable. The gangrenous parts should receive applications of carbolic acid (two to three per cent.), vinum camphoratum, chloride of zinc (0.3 : 150.00); in case of necessity the actual cautery should be applied. When the labium is very hard and swollen, deep incisions should be made in order to relieve the tension and prevent necrosis.

LEEDS & WEST RID
MEDICO-CHIRURGICAL SOCIETY

PART VIII.

INFECTIOUS DISEASES.

In the consideration of infectious diseases, I will confine myself to those features which present practical importance. In my opinion the "bacteria" doctrine has a very insecure foundation with regard to infectious diseases: on the one hand, because microscopical examination furnishes negative results in the most important of these affections; and, on the other, because even in relapsing fever the more recent investigations have at least cast a doubt upon the part played by spirilli in the production of the morbid symptoms. The positiveness with which many physicians speak of the bacteria of diphtheria appears to me unfounded. Apart from the doubtful fungoid character of all the finely granular masses which have been described as diphtheritic germs, it must be acknowledged that the germs floating in the atmosphere will develop and multiply most rapidly in parts which are impregnated with the products of decomposition, and that they may be carried thence to more deeply situated, even remote parts, by the current of lymph or blood. But these processes appear to me to stand in no connection with the nature of the disease.

I will first refer to the simultaneous occurrence of various acute infectious diseases in one individual. Most frequently we find acute exanthemata, especially measles, much more rarely diphtheria, develop during the course of whooping-cough. A number of cases have also been observed in which two acute exanthemata occurred in the same patient.

F. K——, aged nine, admitted March 4, 1875, with varicella, which had been present for two days; the entire body covered with characteristic vesicles. March 7th, no fever, but complaint of pain in swallowing, angina. At night, development of scarlatina, with temperature of 40.0°; on the next day there was marked redness of the skin, on which the varicella pustules, partially dried, partially filled with pus, were also present. Further course normal. Discharged April 13th.

The infection with scarlatina had undoubtedly occurred in the hospital, where the boy lay for thirty-six hours in the ward for infectious diseases, which was partly filled with scarlatina patients.

O. W——, aged seven, admitted October 31, 1876, with spondylitis and cold abcess on right side of lumbar vertebræ. November 29th, development of scarlatina. December 5th, beginning desquamation; on the next day, dulness on right side above, crepitant râles. December 7th, appearance upon the face, arms and legs of a fresh maculo-papular eruption, with all the characteristics of measles, coryza, restlessness, somnolence, croupy cough. Death during the night. Autopsy.—Diphtheritic pharyn-

gitis and laryngitis, croup of the trachea and large bronchi, fibrinous pneumonia of right upper and middle lobes.

A. S——, aged four, admitted February 16, 1877, with prolapsus ani. February 27th, eruption of scarlatina, with moderate pharyngitis; March 4th, beginning desquamation and disappearance of eruption. March 5th, renewed fever, and papular eruption of measles, with cough and catarrhal rales. Continued high fever in next few days, dyspnœa. March 15th, death. Autopsy.—Capillary bronchitis, multiple broncho-pneumonia, fatty liver.

That measles occurred in both these cases and not merely relapsing scarlatina, is proven not only by the character of the eruption, but still more by the affection of the respiratory organs. As measles has an incubation stage of at least twelve days, the infection must either have occurred at the same time as that of scarlatina, or even prior to it.

Those cases are much more frequent in which two or more acute infectious diseases occur successively after comparatively short intervals. Patients convalescing from measles were often affected, after the lapse of a few days, with scarlatina, or vice versa; children who had undergone tracheotomy for diphtheria and croup, were affected with scarlatina, etc., and occasionally three or four of these diseases occurred in rapid succession. Cases of this kind occur very rarely in private practice. In hospitals for children they can only be prevented by the erection of pavilions in which children suffering from measles, scarlatina, and diphtheria, are placed in three separate compartments, which are provided with special nurses.

I. Scarlet Fever.

This disease is one of the most dangerous enemies of childhood. Every child in whose family a case of scarlatina has occurred should be prevented from attending school, and the parents and attending physician should be compelled to report it to the sanitary authorities.

Scarlatina usually attacks children in the midst of perfect health. The usual prodromata of every febrile affection, ill-humor, anorexia, drowsiness, more or less severe headache, are almost always associated with single or repeated vomiting, and soon afterward with pain in the neck on swallowing, which is rarely absent, increased heat of the skin, and thirst. The duration of the prodromal symptoms varies, but is much shorter than in all other infectious diseases, as the eruption appears within twenty-four hours from the appearance of the first symptoms, and often much earlier.

Occasionally the scene is opened by a chill, which I have even observed in a child two years old, or a sudden syncopal attack, immediately followed by high temperature; the disease is much more rarely ushered in by an epileptiform attack. The view that the shorter and more violent the prodromal stage, the more severe will be the future course, I consider unfounded.

The eruption usually appears first on the neck and thorax, a few hours later on the arms, especially around the elbows, or perhaps on the face, and spreads over the trunk and lower extremities in the course of a day. It does not advance so regularly from the face downward as measles and small-pox. In general, the moderate grades of eruption prevail; the skin, when seen from a distance, presenting a diffuse, more or less scarlet redness, while, on closer inspection, it is found that this redness is composed of innumerable red points closely situated together, and separated

from one another by very small, paler portions of skin. The dark red points appear to correspond to the hair-follicles; at least I could observe, as in measles, that when the eruption had passed the abdomen, a darker color and slight swelling of the roots of the hair of the lower extremities was noticeable. In the face only the cheeks and forehead are reddened, while the nose and its vicinity, the upper lip and chin, are strikingly pale, often merging into a yellowish color. As in all exanthemata, the parts subject to pressure, especially the back and nates, present the most marked and diffuse redness; often also the chest and abdomen, while the redness is less marked on the extremities, or appears in large patches, separated by brighter parts. The skin appears rough, if the hair-follicles are more markedly swollen. The redness disappears on pressure, but returns immediately. If we rapidly scrape the reddened skin with the finger-nail, a corresponding white mark is produced, which is distinctly visible for several minutes. These "raies scarlatineuses" are not characteristic of scarlatina, as they also occur in urticaria, or even on the healthy skin. The intensity of the exanthem varies, is more marked at night, but is also different on different days.

The fever increases with the appearance of the eruption, and continues uninterruptedly as long as the exanthem remains on the skin, *i.e.*, from four to six days on the average. A high, continued fever was observed in almost all cases, the nocturnal exacerbations reaching 39.8°–41.0°, the morning remissions showing, at the most, a fall of one degree. I have very rarely observed, for a few days, the inverse type, the morning temperature being one to one and one-half degree higher than that in the evening.[1] The high fever often contrasts with the light color of the urine, though this may also be dark, if urates are present in abundance. As the exanthem grows pale, gradual defervescence occurs, the evening and morning temperatures diminish, and reach the normal with the disappearance of the eruption on the sixth or seventh day. We often find, however, that the evening temperature for several days reaches 38° or 39°, even after the eruption has entirely faded. At this time profuse diaphoresis, especially at night, often occurs, or eruptions of herpes labialis or nasalis. The general condition depends chiefly on the febrile movement. In the milder forms, in which the maximum nocturnal temperature does not pass beyond 40.0°, the children may feel tolerably well, apart from anorexia and increased thirst, but in higher temperatures great restlessness or a tendency to somnolence and delirium is almost always noticeable. The frequency of the pulse generally corresponds to the temperature, but we should not be alarmed by a frequency of 130–140 beats per minute, even in older children, if the quality of the pulse is good.

Most children complain from the beginning of pain in the neck, especially in swallowing, and examination shows a bright or dark redness and swelling of the tonsils, velum, and posterior wall of the pharynx, by which the isthmus faucium is more or less narrowed (angina, s. pharyngitis scarlatinosa). In some cases I observed, even during the first few days, small extravasations of blood on the mucous membrane of the palate, and the sputa, discharged by hawking, were then tinged with blood. The reddened parts are much more often covered here and there with muco-pus, which can be readily removed, or small, purulent points, to which I have previously referred (page 192), are observed on the tonsils. I again warn

[1] Charité-Annalen, III., S. 513. 1878.

you against mistaking them for diphtheritic patches. The buccal mucous membrane is almost always reddened, usually bleeds on firm contact, and the tongue, which, during the first few days, presents a yellowish white coating, with red edges, generally exfoliates this after the third day, and then has a more uniform dark red color, the more or less swollen and prominent papillæ increasing the resemblance to a strawberry (scarlet fever tongue). This condition, though not constant, is so frequent as to present a characteristic significance. As the eruption fades, the inflammation of the buccal and pharyngeal mucous membrane gradually disappears, and only a few slightly swollen glands remain below the angle of the jaw.

From the end of the first week the child is convalescent. At this time desquamation of the epidermis begins, so much earlier the more intense the redness of the skin has been; I have often noticed it in the face as early as the fourth or fifth day. The epidermis is sometimes desquamated in branny scales, sometimes in larger patches and shreds. While the desquamation on the trunk and thighs often appears like a covering of dirt, large flakes are often exfoliated from the fingers and the edges of the hands and feet. I have also observed this on the nates, abdomen, soles of the feet, and in one child the external auditory canal was occluded by the desquamated epidermis, so that it became necessary to remove it by injections of lukewarm water. Desquamation usually lasts several weeks, and during this time temporary albuminuria is not infrequently observed (page 241).

Variations from the normal course of the disease described above are extremely frequent, and they are much more rarely of favorable than of unfavorable significance. We will first consider the former. The febrile movement may be moderate, or at least present considerable remissions —even as much as two degrees—in the morning. The eruption is then usually pale red and the angina slight. Among a number of cases of this kind observed by me, I will choose the following:

Child, aged three, with scarlatina. Began April 9th.

	Morning.	Evening.	Pulse.
April 9th	....	38.5°	144
" 10th	38.0°	39.1°	152
" 11th	38.0°	38.3°	132
" 12th	38.0°	38.0°	...

From April 13th, eruption and fever disappeared.

In rare cases the disease runs an apyrexial course after severe initial fever. Cases of this kind probably occur more often than we imagine, but are overlooked on account of the ephemeral character of the fever, and the pale, rapidly disappearing eruption, until nephritis or the traces of desquamation indicate that the child has really had scarlatina.

In the rarest cases the high fever (40.0° or more) suddenly falls, after a few days, in the form of a crisis, and the further course is apyrexial, despite the continuance of the eruption; or the exanthem appears without any fever, and the latter does not appear until after the full development of the eruption.

The character of the eruption not infrequently presents variations; for example, the redness is not diffuse, but occurs in large patches, or yellowish or white miliary vesicles, which are sharply contrasted with the red skin, make their appearance; these may either be limited to patches,

or appear over almost the entire body. The vesicles are occasionally larger, as in herpes, or assume a varicella or pemphigoid form, the latter being always isolated. Thus, in one boy with very marked eruption, I observed but one vesicle. In other cases dark red nodules, the size of a pea, appear on the diffusely reddened skin, and become flattened after a few days, or itching wheals occur, as in urticaria, and rapidly disappear. All these forms are merely the result of increased dermatitis, and have no unfavorable prognostic significance. On the other hand, a very irregular diffusion of the exanthem—so-called scarlatina variegata—usually appears in unfavorable cases. If, under these circumstances, papular elevations occur, such as I have repeatedly observed, it may lead to confusion with the confluent form of measles. When the redness is very marked, slight œdema sometimes occurs in the hands, feet, and eyelids, the skin feeling hard and tense. The patients complain of pruritus only in these severe grades of dermatitis.

The continuance of fever after the disappearance of the eruption is of more prognostic importance than the variations in the exanthem. This is usually due to some complication or sequela. According to my experience, the continuance of the fever is generally caused by three morbid conditions:

1. Continuance of the pharyngitis, which assumes a necrotic and ulcerative character. This local process may last two to three weeks, and give rise to febrile movement, with marked morning remissions.

2. The development of a glandular and phlegmonous inflammation below the jaw, which is one of the most frequent complications. The submaxillary glands are swollen in the first few days of the disease; in many children they resolve entirely, in others they increase during the second or third weeks, and are converted into a diffuse, hard infiltration of the connective tissue. The swelling may become considerable from collateral œdema, spread upward to the ear and downward along the neck. Almost all these phlegmons suppurate, as a rule, toward the end of the second or in the third week, or even later, and give rise to a remittent fever, with not inconsiderable evening temperatures. The fever disappears rapidly or gradually after the removal of the pus.

These submaxillary abscesses, which affect almost all children in certain epidemics, may become the source of other dangers. If incision is delayed too long, the pus may burrow along the entire side of the neck down to the clavicle, and in a few cases I was compelled to make counter-openings over the pectoralis major muscle.

In one case the pus burrowed to the apex of the right pleural sac, and in another it perforated the pharynx. Although recovery is not impossible in such cases, the prolonged suppuration will weaken the patient very much, even in the most favorable event. Others die from marasmus or from complications, occasionally from hemorrhages due to ulceration of the jugulars or carotid. Not less dangerous is the transition of a phlegmon of the submaxillary tissue into a rapidly progressive, diffuse, extremely hard infiltration of the region of the lower jaw down to the thyroid cartilage (angina Ludwigi). This rigid infiltration, which renders the head almost immovable, I have seen but once at the end of the first week of the disease, more often in the second week, and it has always been associated with other threatening symptoms, somnolence, mild delirium, small and very frequent pulse. The tendency to suppuration is extremely slight, but is very marked toward a gangrenous destruction like that occurring in a carbuncle. Not alone these rigid infiltrations of the

connective tissue of the neck, but also the phlegmons previously described, may endanger life by spreading to the immediate neighborhood of the larynx and producing inflammatory infiltration of the entrance to the larynx, which may terminate fatally, with symptoms of œdema glottidis (page 139).

In rare cases the inflammation appears to spread from the pharynx to the muscles of the neck and throat. I observed pain and stiffness in them, with impaired mobility in three cases, two of which recovered in two weeks under the application of warm poultices and mercurial inunctions, while the other terminated in suppuration and necessitated incision.

3. Otitis is the most frequent cause of protracted fever. The dermatitis may extend from the concha to the external auditory canal, and produce otitis externa, with furunculous abscesses, but this is much rarer than otitis media due to extension of inflammation along the Eustachian tube. In some epidemics this occurs in more than half the cases, and may run a very latent course, especially in young children. Even older ones do not always complain of difficulty of hearing and pains in the ear.

With the appearance of purulent otorrhœa, the pains, but not always the fever, terminate, and examination with the speculum reveals perforation of the drum-membrane. The majority of these perforations usually cicatrize within a few weeks under simple treatment. Much more rarely the inflammatory process is so violent that complete deafness is present by the end of the second week of the disease, and the discharge is extremely fœtid. Even under the best of care, the disease may spread from the cavity of the tympanum to the bones, and cause caries of the petrous portion of the temporal bone. Not infrequently you will find swelling of the mastoid process, which is traversed by fistulous openings, necrotic sequestra in the auditory meatus, and paralysis of the facial nerve. Exceptionally I have observed an abscess behind the external ear, the connection of which with an internal otitis could not be demonstrated. Abscesses in the immediate neighborhood of the external auditory meatus may perforate into the auditory canal, not alone in scarlatina, but also in typhoid fever and simple phlegmons, and the cases observed by me were unattended with permanent bad effects, if free escape of pus was obtained. This is best effected by a counter-opening in the deepest portion of the abscess.

The fever may also be protracted by various complications. Attention should first be directed to the serous membranes. Pericarditis or pleurisy are occasionally found on autopsy, the symptoms of which were either latent or concealed by those of a malignant character, to which we shall soon refer; but, as inflammation of serous membranes also occurs in non-malignant cases, the physician should repeatedly examine the respiratory and circulatory organs, even if no subjective symptoms are presented. We are occasionally surprised to find the physical signs of endocarditis, which was only manifested by the continuance of fever. Chronic cardiac disease may be due to scarlatina, which has long since recovered with an endocarditic complication, which had been overlooked at its acme. This is also true of pleurisy and pericarditis, which rapidly became purulent in a few cases under my observation. The local examination is much the more necessary if the synovial membranes of the joints are affected during scarlatina—a quite frequent complication, which is more appropriately termed synovitis scarlatinosa than rheumatismus scarlatinosus. This affection, which occasionally occurs at the end of the first, but usually during the second week of the disease, is manifested in its

mildest form by pains in the joints, without swelling or appreciable disturbance of mobility, at times limited to a few joints, at times spreading to a number, especially those of the hands and feet. In another series of cases swelling and impaired mobility of the joints are associated with the pain, almost always combined with high fever, and usually, though not always, with other unfavorable symptoms, necrotic inflammations of the mucous membrane of the mouth and pharynx, threatening collapse, and inflammatory affections of other serous membranes—the pleura, pericardium, endocardium, and even the peritoneum. These inflammations, especially those of the pleura and the membranes of the heart, may run such a latent course that they are alone revealed by physical examination.

Although scarlatinous synovitis is usually acute, terminating in a few days or a week, it may also become chronic, and even terminate in suppuration of the joints.

I have had no personal experience concerning the implication of the cerebral meninges in the scarlatinous process. The marked cerebral symptoms which appear in severe cases of the disease are not due to meningitis, so far as my experience goes; at the most I have found congestion or slight œdema of the pia mater and cerebral substance. These appearances are usually due to stasis, in consequence of weak heart's action, which may also give rise to thrombosis of some of the sinuses. However, in view of the observations of others, especially of Reimer,[1] I will not deny the possibility of the occurrence of meningitis. In one case alone did the character of the pulse and the rapid effect of antiphlogistic measures justify the suspicion of beginning meningitis:

A child, two and one-half years old; scarlatina in March, 1865. Normal course until the fourth day; then pallor of the eruption, somnolence, from which the child could be readily roused, slow and irregular pulse, repeated vomiting, constipation, very scanty urine without albumen. Application of three leeches to the head, calomel 0.06 every three hours. Patient more lively on the following day; syr. spin. cervin. to keep the bowels open. On the eighth day, headache alone remained; disappearance of apathy; pulse, 120, regular. Complete recovery.

The mucous membrane of the bronchi and the pulmonary parenchyma are much more often subject to inflammation in scarlet fever, than is usually supposed. Not only catarrhs, but more or less extensive broncho-pneumonias occur during the first and second weeks of the disease, but are often overlooked, as they are masked by other severe symptoms of a "typhoid" character. Bronchitis and broncho-pneumonia were found in almost all cases on which a post-mortem was obtained, and were repeatedly detected during life.[2] The fibrinous form of pneumonia was observed more rarely.

The complication of scarlatina with broncho-pneumonia or pleuro-pneumonia is always dangerous, but not absolutely fatal, as I have seen several cases recover. The prognosis depends especially upon the condition of the heart's action. We here touch upon that characteristic of this many-sided disease, which is most important with reference to pathology and prognosis, and is usually termed "malignity." However manifold the manifestations of this dangerous condition may be, two features predominate in its symptomatology, viz., the marked tendency of the disease to necrotic inflammations and the specific action of the virus upon the heart.

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[1] Jahrb. f. Kinderkrankh., X. 1876.

[2] Vide the cases reported by me in Charité-Annalen, Bd. III., S. 539.

1. Necrotic inflammations. I prefer this term to the word "diphtheritic," which is ordinarily employed, because in my opinion nothing has so interfered with the correct appreciation of the nature of these processes as this term. After Bretonneau,[1] under the term diphtheria, had given an almost exhaustive description of this infectious disease, pathological anatomy introduced new confusion by converting the clinical conception into an anatomical one, and designated all processes as diphtheritic which were characterized by fibrinous exudation into the mucous membranes or the external skin, with subsequent necrosis. Thus it came about that physicians recognized a complication with diphtheria in the most different diseases in which the above-mentioned processes were present. This is especially true of scarlatina, though physicians do not consider whether the specific disease, known as diphtheria, really exists in those cases which are even now generally termed "scarlatina and diphtheria." Necrotic inflammation, as I term it, may occur in various morbid processes, usually in true diphtheria and scarlatina, then in measles, typhoid fever, dysentery, pyæmia, cholera, etc. But the similarity of the anatomical products does not prove the identity of the morbid processes. We are therefore not justified in speaking of diphtheria whenever this form of inflammation is present, and it is better to reserve this name for the specific infectious disease which bears it since the time of Bretonneau. This view is supported by the fact that the form of scarlatina in question by no means affords protection against an early infection with diphtheria.

The necrotic inflammation in scarlatina first attacks the pharyngeal mucous membrane. Between the third and fifth days of the disease yellowish or grayish white patches of variable size are observed upon the reddened and swollen tonsils, and a swelling of the corresponding lymphatic glands. Similar streaks may also form on the borders of the velum palati and on the uvula, or spread from the tonsils toward the root of the tongue. The disturbances of deglutition are not necessarily greater than normal, but correspond chiefly to the intensity of the inflammatory tension and swelling of the pharyngeal tissues. I have observed this mildest form of pharyngeal necrosis in a large number of cases of scarlatina, which presented no other marked deviations from the normal course. After five to six days, or perhaps not until the second or third week, the remains of the patches are exfoliated, and leave shallow, readily bleeding losses of substance, which soon cicatrize. But this affection often becomes more intense and is then almost always associated with serious anomalies and complications, which endanger life. Not only the tonsils and the velum palati, but also the posterior wall of the pharynx, become coated with lard-like patches, the mucous membrane is covered with tough mucus, and, on opening the mouth, extends in tough threads from the tongue to the palate. There is marked fœtor oris, the submaxillary enlargements are more extensive and harder than usual, and the process almost always extends to the nasal cavity and produces coryza. A serous, gangrenous, often bloody secretion flows continuously from the excoriated nostrils over the macerated upper lip and the nose, and surrounding parts not infrequently become œdematous. Occasionally the conjunctiva, probably through the medium of the lachrymal passages, is implicated, either in the form of catarrhal inflammation with profuse secretion and adhesion of the edges of the lids, or more rarely as a necrotic

[1] Traité de la Diphthérie. Paris, 1826.

process with swelling of the lids. In the most severe, and, fortunately, very rare cases of this kind, necrosis of the cornea and complete destruction of the eye may occur. In a number of cases I saw pseudo-membranous shreds in the coryzal secretion. Repeated and severe hemorrhages occasionally occurred from the ulcerative losses of substance left over in the nares and pharynx. When the necrosis penetrated deeply into the tonsils, large portions of them were exfoliated, and floated at first in the pharynx as blackish brown, stinking masses. In a number of cases, bilateral total perforation of the velum palati occurred, usually above the tonsils.

All these grave phenomena may also occur in true diphtheria. In both cases there is fibrinous infiltration with abundant nuclear proliferation and cell-production in the deeper layers of the mucous membrane, by which the vessels are compressed and the tissues become necrotic, and bacteria are also found in both cases. But this does not prove the identity of the morbid processes, in opposition to which it may be mentioned that paralyses rarely occur after scarlatinous necrosis of the pharynx. Another important difference consists in the fact that the latter affection shows very little tendency to spread to the upper air-passages. Hoarseness or even aphonia are, indeed, not infrequent, but these suspicious symptoms gradually subside in the majority of cases, and appear to depend on a catarrh of the vocal cords, which, however, occasionally spreads downward and terminates in broncho-pneumonia. Nevertheless, I have reported eight cases of scarlatina,[1] in which the pharyngeal affection spread to the larynx, and was demonstrated on autopsy in seven cases. But in none did the croup pass the vocal cords, and only in the case in which an autopsy was not obtained, did the discharge of pseudo-membranous shreds from the canula introduced after tracheotomy favor the view of a croup of the trachea or bronchi. But it should also be considered that bronchial croup, under these conditions, may develop from the aspiration of infectious shreds from the pharynx, although the process has not spread continuously through the upper air-passages.

In the last few years a number of similar cases of laryngeal croup in scarlatina have come under my observation:

E. H——, aged five, March 2, 1877; scarlatina with high fever and simple pharyngitis. March 5th, beginning desquamation in the face. In the next few days, hoarseness, increased by March 8th to aphonia, associated with noisy inspiration. March 10th, dyspnœa; temperature, 40.4°; evening, 40.7°. March 12th, bilateral broncho-pneumonia detected posteriorly; March 17th, death in collapse. No membrane was discovered in the pharynx during the entire course of the disease. Autopsy.—Diphtheritic pharyngitis, croupous laryngitis, bilateral broncho-pneumonia, left pleurisy, enlargement of spleen and mesenteric glands.

H. S——, aged eighteen months; February 14, 1877, scarlatina with angina and small points of pus on the tonsils. Bronchial catarrh, moderate fever. February 24th, hoarseness, suspicious cough, increasing fever. During next eleven days, development of dyspnœa, bilateral broncho-pneumonia, almost complete aphonia, slight stenotic sound in breathing; March 7th, death. Examination had never revealed a membrane in the pharynx. Autopsy.—Diphtheria of pharynx and œsophagus, croup of larynx, bilateral broncho-pneumonia, cheesy degeneration of bronchial glands, tubercles of liver and spleen.

In both cases the necrotic inflammation of the pharynx had not been recognized, despite repeated examination—a circumstance which can be explained partly by the concealed situation of the patches, partly by the

[1] Charité-Annalen, III., S. 529. 1876.

impossibility of carefully examining all parts of the pharynx in such children. In these two cases also the necrotic process did not spread beyond the larynx, and such extension must therefore be regarded as extremely rare. The necrotic process in scarlatina may also spread to the œsophagus, and even to the gastric mucous membrane; I have observed the latter in but two instances, while the former occurred more frequently. But there are no definite corresponding symptoms during life, and even the implication of the larynx is manifested by comparatively mild symptoms, hoarseness, aphonia, noisy breathing, which do not attain the intensity of true croup symptoms. In some cases, the malignant symptoms of scarlatina were so predominant that the laryngeal signs were entirely overlooked.

The dyspnœal symptoms occurring in scarlatinous necrotic inflammation do not always depend on an affection of the larynx, but may be caused by enormous swelling of the tonsils and adjacent portions of the pharynx. Co-existing severe coryza may increase the symptoms by narrowing the nares, and they will reach their greatest intensity if œdema glottidis occurs. It is impossible, however, in all such cases, to ascertain positively the condition of the larynx. In a number of cases of this kind, in which the stenotic symptoms had been very severe, the autopsy showed, in addition to necrotic pharyngitis and coryza, large tonsillar abscesses, and, in several cases, retropharyngeal abscesses, while the larynx was entirely free, with the exception of moderate infiltration of the aryepiglottic ligaments. Such cases undoubtedly indicate tracheotomy, but only once have I seen a successful result.

The pharyngeal affection is often combined with an analogous affection of the buccal mucous membrane (stomatitis scarlatinosa), in which the lips, tongue, more rarely the hard palate, appear infiltrated with grayish yellow or grayish white patches. The stomatitis may develop as early as the fifth day, but I have seen it appear more often during the second week, or even later. It is often so painful that the children cannot protrude the tongue, or even eat. Grayish yellow patches often form from the bleeding of the angles of the mouth and the lips, extend over the mucous membrane of the mouth and tongue, and leave more or less deep losses of substance, after exfoliation of the necrotic tissue. Even in several cases in which these ulcerations were quite superficial, I have seen such large hemorrhages ensue, that life was seriously endangered by exhaustion, and the bleeding could only be stopped by the persistent use of liq. ferri sesquichlorat., either applied by means of charpie or brushed upon the tongue. In many cases this affection is so mild that it presents the appearance of ordinary aphthous stomatitis (page 186), while in others the mucous membrane is covered with whitish, croup-like deposits, which can be removed quite readily, and leave superficial, bleeding erosions. The use of a mouth-wash of chlorate of potash (5 : 200), but especially a few applications daily of sulphate of zinc (0.5 : 30.0) often gave me excellent results.

The labia majora, mucous membrane of the vulva, and accidental excoriations of the external skin, may also be covered with fibrinous exudation.

I have remarked above (page 269) that the pharyngitis is at first a simple inflammatory one, and the necrotic character does not usually appear until the third to fifth day. There are exceptional cases, however, in which suspicious patches may appear in the pharynx even before the development of the eruption. The disease then begins with moderate, occasionally high fever (39.5°–40.0°), and diphtheritic angina, and

the scarlatina eruption does not appear until two to three days—in one case five days later. Since my earlier publication [1] I have had repeated opportunity of observing this unusual beginning. In such cases we must ask ourselves whether the first affection was really connected with the second, or whether the former was a true primary diphtheria, rapidly followed by scarlatina. This opinion is favored by the fact that I have only observed such cases in the hospital, in which infection with various forms of contagion can scarcely be avoided.

The malignity of scarlatina depends not only on the tendency to necrotic processes, but much more upon the specific action of the virus upon the nerve-centres, and of these upon the heart. Before entering in detail upon this grave peculiarity, I will direct your attention to certain symptoms which may excite apprehension during the first few days of the disease. At the very beginning of the high fever, and when the eruption is diffuse and dark red, many children fall into a somnolent condition, from which they can be readily roused. Many are delirious and toss to and fro; others are apathetic, do not answer questions, and do not appear to recognize distinctly those around them. No other serious complications are present; the urine is free from albumen, the angina moderate, the pulse not too frequent and of good quality. With the fall of temperature on the fourth or fifth day, the cerebral symptoms disappear, and the disease then pursues the ordinary course.

These apparently threatening cerebral symptoms are undoubtedly due to the continued high fever, and I have obtained good results from the use of lukewarm baths of 26°–25° R., lasting about ten minutes. In many cases I have given two baths daily. In addition, an ice-bag is applied constantly to the head, and another to the throat if the pharyngitis is severe. Large doses of quinine (0.5–1.0) or salicylate of soda (2.0), given at mid-day, were occasionally effective. But all these antipyretic measures prove useless if the high fever, delirium, and somnolence were from the beginning the precursors of truly malignant symptoms. The temperature then remained at the same height, or even rose, and I regard the inefficacy of antipyretic measures as an unfavorable prognostic factor, which indicates that we have to deal with a malignant affection, though we know almost nothing concerning the nature of this virulence.

In many simple cases of scarlatina the skipping character (pulsus celer) and unusually high frequency of the pulse (150 beats or more) evince this effect of the virus, which I regard as paralysis of the pneumogastric centre. Although similar collapse symptoms are found in severe forms of other infectious diseases, they are by far the most frequent in scarlatina, and constitute the chief danger. They are not due to the molecular changes so often found in the heart-muscle on autopsy, as they appear before this degeneration could have occurred. Every physician occasionally meets with cases in which, in the midst of perfect health, sudden vomiting, perhaps diarrhœa, appear, together with enormous frequency and smallness of the pulse and such rapid collapse, that the pulse disappears within eight to twelve hours, the face and extremities become cool, and death occurs in coma, rarely in convulsions, without any eruption having appeared. The diagnosis remains obscure until scarlatina develops in one of the family within a few days. More frequently the eruption appears, though only partially and irregularly, and death occurs in twenty-four to forty-eight hours.

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[1] L. c., S. 525.

The paralyzing influence of the virus on the brain and heart is most marked during the first few days of the disease, most frequently in children under three years. We then often find, from the beginning, the pulse extremely rapid (170, or more) and readily compressible; the hands, feet, and nose are cool, although the temperature of the body is high, and the eruption is cyanotic and livid on account of the venous stasis produced by the weakness of the heart; the little patients are extremely feeble, delirious, rapidly become comatose, and die in this condition, the pulse becoming continually feebler and more rapid. All these "foudroyant" cases are absolutely fatal.

The prognosis is somewhat more favorable when the symptoms of heart-failure do not appear until after the complete development of the eruption, during the first or even the second week of the disease. As they progress less rapidly under such circumstances, they are more readily overlooked by the inexperienced observer. The children are more or less somnolent, completely apathetic, and toss to and fro. The eyes are entirely or half closed, the conjunctiva usually markedly injected, the patient dreads the light; the eruption varies, appearing either as scarlatina variegata (page 266), or like measles, or has a coppery color. The face is swollen, especially around the nose, the implication of which in the necrotic inflammation is manifested by coryza, snuffling, and snoring; the tongue, lips, and gums are dry, covered with brownish sordes; the pharynx is almost always "diphtheritic," and exhales a strong fœtor. Otitis and phlegmons of the submaxillary regions (page 269) may also be present; the urine may show signs of early nephritis, and inflammation of the respiratory organs or serous membranes may also be observed. These symptoms may continue eight to ten days, or longer. During this time the fever persists until death, with a temperature of 39.5°–40°, or more. In a few cases I have even found it 40.2°–42.5° shortly before death, although the pulse could scarcely be felt and the extremities were cool, while in other apparently similar cases the temperature varies considerably, and falls on the last day even as low as 36.5°. More or less profuse diarrhœa is observed quite often in malignant cases, and is occasionally so sudden and severe as to give rise to choleraic collapse. The chief prognostic significance must be attached to the character of the pulse. So long as the pulse is only moderately frequent (120–130 beats, according to age), and retains almost its normal tension and fulness, we should not give up hope, however grave the other symptoms may appear. The prognosis is fatal, however, if the pulse becomes very small, readily compressible, dicrotic, irregular, but especially extremely frequent (180, or even 200 and 240), and if the extremities grow cool, the eruption cyanotic, and the coma more profound.

Post-mortem examination reveals nothing characteristic in this disease. In addition to the numerous complications, you will find, at the most, the albuminoid and fatty degeneration of the muscular fibres of the heart, the liver-cells, and renal epithelium, which is common to all severe infectious diseases, and, in addition, moderate swelling of numerous lymphatic glands, Peyer's patches, the solitary follicles of the intestines, and the mesenteric glands.

As various sequelæ may develop after the acute process has terminated, I would advise to inform the parents at the beginning of the disease that you cannot, before the end of the fourth week of the disease, give a guarantee of a favorable termination. Among the sequelæ, nephritis, which I have previously described (page 241), occupies the first

rank. Then we must fear the sequences of otitis media, its spread to the bones, finally implication of the sinus and meninges, or paralysis of the facial nerve, and permanent deafness. In two cases I observed the development of pneumonia during convalescence, and I have also seen diphtheria occur during this period. But I have very rarely met with gangrene of the skin or mucous membranes, a few times as decubitus over the sacrum or other parts of the body, which are subject to pressure, once as necrosis of the nasal cartilage, but never in the form of noma of the mouth or female genitalia, such as has been occasionally observed by others. Among the frequent sequelæ must also be mentioned abscesses of the neck, back, hands, eyelids, and the immediate vicinity of the joints, which finally led to marasmus from the continued suppuration, and in a couple of cases perforated into the joints lying in their vicinity. Eczematous and pustular eruptions, especially in the face and on the ears, occurred not infrequently during the first few weeks or months after scarlatina, but I have only observed pemphigus once at this time. In a few cases leucorrhœa developed immediately after scarlatina, probably from extension of the dermatitis to the mucous membrane of the genitalia. According to my experience, the nervous system suffers least of all. In two children I observed an ataxic gait, which lasted in the first case for a few days, and in the second for several weeks; chorea occurred twice during the acute period of the disease (associated both times with pains in the joints), but never as a sequel. Finally, I may mention the manifestation of a hemorrhagic diathesis in the form of purpura, which I have repeatedly observed as a sequel; these cases always terminated favorably, though other authors have reported rapidly fatal cases. All of my cases (five in number) developed in the third or fourth week after the eruption; with one exception, they were all associated with nephritis. Rapid recovery from the purpura, as well as the nephritis, ensued under the administration of ergotin. The cause of the hemorrhagic diathesis after scarlatina is unknown; it is probably due to molecular changes in the walls of the small vessels.

As in typhoid fever, relapses of scarlatina also occur, though much more rarely. After the fever has disappeared for a few days, or even several weeks, and desquamation has progressed in a normal manner, the eruption reappears with a sudden exacerbation of fever, and the disease runs its course a second time. I have had repeated opportunity of observing these relapses.

As there can be no question of a second infection, I can only explain these cases on the theory that the scarlatinous virus was not entirely eliminated during the first attack, and that a relapse therefore occurred. With more careful attention, relapses will probably be observed more frequently. But we must guard against mistaking simple erythema or urticaria for a relapse, which must always be followed by renewed desquamation.

This fact appears to me of special importance in deciding upon so-called scarlatina *sine* exanthemate—*i.e.*, scarlet fever without eruption. The occurrence of such cases is undoubted. Some members of the family may be affected with scarlatina which runs a normal course; while others, especially the parents and servants, but occasionally the children also, merely suffer from more or less severe pharyngitis, with fever and considerable disturbance of the general condition, without presenting any eruption. In a few adults I have observed nephritis as a sequel of scarlatina *sine* exanthemate, and it may even be attended by pain in the joints.

The susceptibility to scarlatina is present during all periods of childhood, but is least marked in children under two years. The period between three and eight years is chiefly affected. In general, more people escape scarlatina than measles. Concerning the character of the infection, we possess no certain information. There is no doubt that inspiration of the air surrounding a patient produces infection most readily; probably, though not certainly, the virus may also be carried in clothing and other articles; perhaps also in food. In England, milk is especially regarded as a carrier of scarlatina infection, as it is also of diphtheria and typhoid fever.

I have repeatedly confirmed the fact that individuals with open wounds possess an increased susceptibility for the scarlatina contagion.[1] Children with fresh wounds (phimosis, tracheotomy, etc.) were often affected with scarlatina, usually from five to seven days after the operation. This also corroborates the fact that scarlatina has an extremely short period of incubation compared with other infectious diseases. Repeated personal observations have shown that the period of incubation is often not longer than four days, occasionally only thirty-six to forty-eight hours, while Trousseau, Murchison, and others have observed a still shorter duration (twenty-four to twenty-eight hours).

At what period of its course scarlatina is most infectious we do not know, and must therefore isolate the patient during the entire period of the disease, until the termination of desquamation. The possibility of infection during the period of incubation induced me to advise the strict precautions mentioned above (page 263) with regard to attendance at school.

If we except relapses, a second appearance of scarlatina in the same individual is very rare. I have observed but one undoubted case of this kind, in which the second attack occurred a year after the first.

I finally come to the treatment of the disease. No drugs are required in normal cases which are free from complications. The child should be isolated as much as possible from the brothers and sisters, or the latter be removed from the house. Pure air and cool temperature (13°–14° R.) should be urgently recommended; the windows should therefore be repeatedly opened, at least in the adjoining room, the child covered lightly, and the room darkened only when photophobia is present. Cool drinks, milk, soup, pigeon or chicken broth, should constitute the diet during fever days. An enema or a mild purgative every other day—for example, a teaspoonful of magnesia or a laxative effervescent powder; a wineglassful of bitter water, etc., may be given in constipation.

If the fever continues high and the apparently malignant symptoms described above (page 272) develop, the head should be covered with an ice-bag, a large dose of quinine (0.5–1.0) or salicylate of soda (1.0–1.5) given between four and six o'clock P.M., and the child placed in a lukewarm bath (not under 25° R.). I decidedly oppose cooler baths, because in scarlatina, which presents a tendency to heart-failure, cold may produce an unexpected rapid collapse more than in any other affection. But I strongly recommend washing the entire body every three hours with a sponge dipped in cool water and vinegar. If something must be prescribed, hydrochloric acid (P. 3) is the most suitable.

In truly malignant cases, however, antipyretic treatment proves entirely useless. I have never seen any effect from large doses of quinine,

[1] Charité-Annalen, I., S. 599.

and I consider salicylate of soda in such cases as a dangerous remedy. In several of these cases I have seen dangerous, and in one case fatal, collapse develop during a bath. In this, as in every other intoxication, the termination almost always depends on the quantity of poison which has been absorbed. In all severe cases of scarlatina the paralyzing effect of the virus on the nervous system of the heart is the chief factor to be combated by the physician. If we can sustain the action of the heart by the use of powerful stimulants until the organism has overcome the other severe consequences of the infection, we may still hope for a favorable termination, unless grave complications are present. In a large number of severe cases I have tried quinine, carbolic acid, salicylic acid, benzoate and subsulphite of soda, with entire failure. I have therefore completely abandoned these remedies, and now confine myself to the administration of stimulants, which, at least, have a palliative effect. Among these remedies, I assign the first rank to alcohol (wine, brandy), coffee in large doses, and camphor. Under the persistent use of these remedies, I have seen decided results in a number of severe cases, though never when the symptoms of collapse had reached an extreme grade.

Wine (Tokay, sherry, champagne) should be given in doses of one to two spoonfuls every hour; coffee in quantities of half a cup several times daily; camphor, 0.06–0.1, and musk, 0.05–0.2, according to age, every two hours. When deglutition is impaired by swelling of the pharynx, a nutritive enema of pepton should be given twice a day, or a small cup of bouillon with yolk of egg and a spoonful of wine, and a hypodermic injection made every three hours of sulphuric ether (a hypodermic syringe full), or camphor, either in the form of camphorated oil, or, better still, as a solution of camphor, 0.06, in spirit. vini and aq. dest. āā 5.0. These injections occasionally produce circumscribed, yellowish infiltrations with red borders, which afterward become necrotic and suppurate. I consider carbonate of ammonia and valerian too weak to sustain the action of the heart. Lukewarm baths (27°–25° R.), with cold affusions over the neck and back, are more effective, but their action should be carefully watched, as the latter have occasionally appeared to me to increase the collapse, and more active stimulants were then required to elevate the depressed temperature.

If the malignant form of scarlatina runs its course without any dangerous symptoms of heart-failure, as may happen for a number of days, I would advise the constant employment of decoct. cort. chinæ (5.0–8.0 : 120) with aq. chlori (15.0), which may be exchanged for tinct. valerianæ (2.0–3.0) if the pulse is sinking. Disinfection of the buccal, pharyngeal and nasal cavities should be made by injection of these parts every two to three hours with a solution (two per cent.) of carbolic acid, or with permanganate of potash (0.5 : 100). I have also had success in injecting the nose with sulphate of zinc (1.0 : 100.0), or brushing it with a five per cent. solution of chloride of zinc.

In synovitis, the painful or swollen joints should be wrapped in cotton. Warm poultices, free incision when fluctuation is felt; antiseptic dressings are advisable in phlegmons of the submaxillary region.

Lukewarm baths should be taken as soon as desquamation begins. The patients should not be allowed in the open air before the fourth week.

II. Measles.

Although measles is not, by any means, always a mild disease, it is much less serious than scarlatina.

From certain experiences in localities which were not attacked by measles for a large number of years, we know that the period of incubation is about twelve or thirteen days, and that several days further elapse before the appearance of the eruption. If measles appears in one member of a family, all the children are generally attacked, whether isolated or not. The period of incubation is usually entirely free from symptoms. I could rarely confirm the observation of Thomas, Rehn, and others, that ephemeral febrile movement develops during this period.

The beginning of the prodromal stage is usually shown by general malaise, bad humor, loss of appetite, and slight catarrhal symptoms. The edges of the lids are slightly reddened, the eyes somewhat dull and readily overflowing; frequent sneezing, perhaps epistaxis, and a short, dry cough, are usually present. Some complain of pain in swallowing, and examination shows mild angina tonsillaris. These catarrhal symptoms may be so insignificant that the general condition appears very little affected; but the thermometer shows more or less rise of temperature, occasionally at night, to 37.8°–38°, in other cases reaching 38° or 39° in the morning, especially of the first day; it always presents variations, so that it may be normal, or nearly so, on the second day, and rise again on the third. From the end of the second day a more diffuse or occasionally dark, patchy redness is observed over the hard and soft palate, or there are punctate and stellate red patches on the otherwise pale mucous membrane, a symptom which almost always precedes the eruption. The duration of the prodromal stage is, on the average, three days, more rarely four to six days. We can compare the following tables:

Child, aged eighteen months.

	Morning.	Evening.	
Dec. 2d	..	39.6°	Conjunctivitis.
" 3d	38.1°	39.4°	Cough.
" 4th	39.4°	39.5°	
" 5th	38.3°	39.8°	Eruption.

Child, aged four years.

	Morning.	Evening.	
Aug. 16th	..	39.3°	Angina.
" 17th	38.3°	38.2°	
" 18th	37.7°	38.3°	Euphoria.
" 19th	38.1°	39.4°	Cough.
" 20th	38.4°	39.6°	Eruption.

Child, aged three years.

	Morning.	Evening.	
Mar. 28th	..	39.3°	
" 29th	37.6°	38.4°	Catarrh.
" 30th	38.2°	39.2°	Conjunctivitis.
" 31st	38.6°	39.8°	
Apl. 1st	39.8°	39.8°	
" 2d	39.1°	40.0°	Eruption.

Child, aged three and one-half years.

	Morning.	Evening.	
Nov. 15th	37.6°	38.1°	Catarrh.
" 16th	39.3°	39.6°	Exacerbation.
" 17th	38.8°	40.4°	
" 18th	38.5°	39.6°	
" 19th	40.3°	40.2°	Pneumonia.
" 20th	39.5°	40.2°	Eruption.

In these four cases it is only the last in which the delay in the appearance of the eruption is associated with a pneumonia developing during the prodromal stage. Such cases occur quite often, and we must assume that the early serious complication delays the appearance of the eruption. The prodromal stage is also apt to be delayed in children who are feeble or otherwise sick. During this time the skin, as a rule, presents no abnormal appearances; very small, pale red papules, exceptionally a tem-

porary erythema (rash of the English writers), are rarely found in the face.

The beginning of the eruption, except in very feeble children, is generally marked by a decided increase of fever. The temperature rapidly rises to 39.5°–40.5°, and the exanthem appears first in the face, usually on the forehead and in front of the ear, in the shape of bright red, very flat papules, from the size of a pin's head to that of a lentil. This is usually associated with great restlessness and a short, often almost constant cough. The eruption rapidly spreads downward over the face, neck, chest, and limbs, so that the whole body is covered in twenty-four hours. The papules, which are at first small and situated around the roots of the hair, become surrounded with a hyperæmic zone, and form round or half-moon-shaped spots with irregular borders, from the size of a pea to that of a bean, which disappear temporarily on pressure, and whose papular origin is more perceptible to the touch than to sight. The patches may be discrete over the entire body, separated from one another by normal integument, or they are confluent in some parts, especially on the cheeks, chin, back, and nates; the face presents an entirely changed appearance by its turgescence and the swollen lids, which are usually closed on account of photophobia. Occasionally the eruption is very sparse over the entire body. The poorly developed forms are especially observed in enfeebled children. The development of a serious complication does not always exercise an inhibitory influence on the appearance of the eruption. When the exanthem appears first on the chest or the back, and then spreads irregularly, the course of the disease is often unfavorably influenced by complications or by a general weakness previously present. However, this is not always the case, and the eruption may be extremely scant in favorable cases of measles.

The fever usually continues undiminished for thirty-six to forty hours from the appearance of the eruption, so that the morning temperature reaches 38.5°–39.6° (occasionally even 40.3°), and that at night 38.8°–40.5° (occasionally even 41°.); exceptionally I have observed the typus inversus (morning, 39.5°; evening, 38.5°). It is always associated with great restlessness, thirst, and severe cough. On the second day after the beginning of the eruption the temperature begins to fall; I have often seen a critical depression at this time to 37.2° and 37.6°, in the evening 37.9° at the most, while in other uncomplicated cases the evening temperature reached 38.8°, or even 39.4°. In the majority of cases, however, the fever disappears at the end of two days, and the temperature in the morning may even be subnormal (36°–37°); a smaller number present nocturnal exacerbations to 38.0° and 38.5°, occasionally until the fourth day. Night-sweats and severe pruritus are usually observed at this time, and the skin often shows numerous sudamina. If febrile movement continues beyond the fourth day, you should always be on your guard; this betokens the presence of a complication, generally bronchial catarrh or pneumonia, and examination of the chest is therefore necessary. During the third and fourth days the eruption rapidly pales; yellowish or yellowish gray patches remain several days later giving the skin a marbled appearance, and then disappear to give place to slight branny desquamation, its intensity varying according to that of the eruption.

Apart from the fever, the symptoms during the eruptive stage are essentially the same as in the prodromal period. Cases of absolute euphoria occasionally occur; but, as a rule, we find conjunctivitis and blepharitis, more or less severe photophobia, coryza, epistaxis, a fre-

quent, short, somewhat hoarse cough, anorexia, a grayish white coating on the tongue, the papillæ of which at the apex are occasionally reddened and somewhat prominent, and perhaps anginal disturbances, pain in swallowing, redness and swelling of the pharynx, especially of the tonsils. The gums and entire mucous membrane of the mouth are occasionally reddened and sensitive, and covered here and there with desquamated epithelium. Many children suffer on the first day of the eruption from repeated vomiting, more often from diarrhœa, which may be very profuse, and show small quantities of blood. When the fever is very violent, the little patients often lie in a half-doze, are occasionally delirious, especially at night, and the lips readily become dry and covered with thin brownish crusts. Pruritus of the skin is not infrequently complained of. On examination of the chest we usually hear a rude respiratory murmur, and sonorous, later mucous râles on the dorsal surface ; in many cases nothing abnormal is discovered. The catarrh of the trachea and large bronchi is then only manifested by the painful cough produced by pressure on the trachea. The frequency of the respiration (30–40) corresponds to the high temperature and the pulse, which usually rises to 132–144 per minute. On the fourth or fifth day from the beginning of the eruption the majority of the patients may be regarded as convalescent, merely a slight catarrhal cough and perhaps slight desquamation being noticeable. In not a few cases the traces of the eruption may persist for some time, even two to three weeks, in the shape of bluish red, discrete or confluent patches, which do not disappear on pressure. These are due to hemorrhages in the skin, caused by the hyperæmia, or perhaps merely by the passage of blood-globules through the walls of the small vessels (hemorrhagic measles), and do not interfere with the mild course of the disease. This form of measles has no connection with the "hemorrhagic" diathesis, and the latter may even be present, although the eruption of measles does not present a hemorrhagic character.

This simple normal course of measles is not observed in all cases. In the first rank belong the inflammatory affections of the respiratory organs. Even in mild cases the cough sometimes has a rough or barking character, and in children with a tendency to pseudo-croup the disease often begins with such a seizure. In other cases the voice and cough become hoarse during the first few days, and the children complain of pain in the neck, increased on swallowing and on pressure upon the trachea. When these symptoms are present in vigorous children I would advise antiphlogistic measures (page 144), and the application of a few leeches to the manubrium sterni, as croup may otherwise develop from the laryngeal catarrh. This complication occurs not infrequently in measles, and may be unattended with any diphtheritic affection of the pharynx.

The bronchi and pulmonary tissue are more frequently the starting-point for dangerous complications than the larynx and trachea. The bronchitis and broncho-pneumonia occurring in measles do not vary in their symptomatology from that previously described (page 139). Croupous pneumonia is much more infrequent than broncho-pneumonia. Fibrinous pleurisy is often found over the inflamed lobes of the lung, while fluid exudation is rare, and purulent collections in the pleura very exceptional. Broncho-pneumonia occurs most frequently when the eruption fades, or after it has entirely disappeared. An exacerbation of fever, after normal temperature has been observed for a few days, or the continuance of fever after the eruption has disappeared, should induce you to examine the thorax forthwith. If you merely find dry or moist rhonchi of bronchi-

tis, the prognosis should be guarded, as dyspnœa, moaning expiration, stertor, and the other symptoms of broncho-pneumonia may develop in 24–36 hours. The broncho-pneumonia occurring in the diminishing stage of measles is undoubtedly the most frequent cause of death in this disease—the prognosis is so much more grave the younger the children. During infancy I have found this complication begin with epileptiform convulsions on a number of occasions.

The fever may also be prolonged on account of complications on the part of the mucous membrane of the digestive tract. I have frequently seen angina tonsillaris continue into the second week, or only develop at this period. Stomatitis similar to that described in scarlatina (page 271) may develop on the tongue, occasionally on other parts of the mucous membrane. Diarrhœa is a much more frequent complication ; it may occur in the first few days, is a constant symptom in some epidemics, and is often combined with severe bronchial catarrh or broncho-pneumonia. The evacuations may become very profuse, often as many as 12–20 daily, are often accompanied by severe colic, and at times assume a dysenteric character which may lead to fatal collapse. Although many cases of moderate diarrhœa terminate favorably, the tendency of measles to intestinal catarrh should always be taken into consideration, and requires especial caution in the administration of purgatives. In order to relieve constipation we should therefore merely order enemata or mild laxatives.

Otitis media, with perforation of the membrane and fœtid otorrhœa, may also develop as a sequel of measles, and agrees in all respects with that occurring in scarlatina, except that the latter is much more frequent.

Among infectious diseases, whooping-cough is associated with measles with special frequency, and sometimes in entire epidemics (page 175). The former has usually lasted for some weeks, and the occurrence of measles must then be regarded as unfavorable, since the tendency to broncho-pneumonia common to both is thus increased. Although many cases of this kind run their course without any serious pulmonary affection, the prognosis is always doubtful, especially if broncho-pneumonia has developed as the result of whooping-cough before the occurrence of the measles. Under such circumstances I have seen the eruption very scanty or cyanotic in color, while the dyspnœa was enormously increased, the pneumonic signs became more widespread, and the pulse more rapid and smaller. Death almost always occurred in 36–40 hours in intense cyanosis. This rapidly fatal course is to be apprehended in all children who are attacked by measles after having suffered for a long time from exhausting diseases. In many cases of this kind the high fever at the outbreak of the eruption remained absent, and this even occurred when the primary disease, for example, a chronic ulcerative intestinal catarrh, had been apyrexial.

A child, aged nine months, very feeble and emaciated from chronic diarrhœa.

	Morning.	Evening.
December 17th	.....	38 4° Catarrh.
" 18th	37.5°	36.6° Respirations 60.
" 19th	35.7°	39.7° Eruption of measles.
" 20th	37.9°	39.9°
" 21st	36.9°	39.2° Collapse and pneumonia.
" 22d	38.3°	38.8° Death.

True diphtheria is also a not infrequent complication of measles. Occasionally a child admitted to the wards with diphtheria became affected

with measles, but the reverse obtained much more often. The diphtheria usually developed during the second week of the disease, and was rarely limited to the pharynx. The majority of cases proved fatal from extension of the process to the larynx and bronchi, so that tracheotomy was almost always useless. In a few cases, the diphtheria occurred at an earlier period, for example on the fourth day—once, indeed, at the same time as the eruption of measles, so that it became necessary to perform tracheotomy at this time. But as these children had been in the wards for several weeks on account of other diseases, we may assume that the diphtheritic infection occurred at the same time or soon after that with measles, and thus produced an almost simultaneous development of both diseases.

Affections of the nervous system are the rarest complications of measles. Until the age of two years, eclamptic attacks sometimes occur at the beginning of the eruptive stage. Older children frequently complain of headache, especially in the forehead, which is partly due to the fever, partly to the coryza. The somnolence not infrequently observed at the height of the affection need not cause uneasiness, as it rapidly disappears with the subsidence of the fever. The following is the only case in my experience in which serious nervous symptoms developed:

C. J——, aged three, measles in beginning of November, 1876. In the middle of the second week after the eruption, sudden somnolence, rigid contraction of the muscles of the neck, moderate fever, irregular pulse. Antiphlogistic treatment; rapid improvement. Relapse after a few days. December 1st, rigid contraction of the neck, left internal strabismus; sensorium unclouded. December 7th, head freely movable; strabismus diminished; euphoria. At the end of another week, complete recovery.

There is no doubt that severe cerebral symptoms, deep coma, delirium, tremor, may be caused by the malignant character of the disease, but such cases are extremely rare and not a single one has come under my observation. As in malignant scarlatina, these cases are accompanied by severe hemorrhages into the external skin and various other organs, and always terminate fatally. Nor have I observed any relapse of measles, though I have sometimes found the stage of eruption considerably prolonged.

The sequelæ of measles are usually nothing more than the complications which have run a chronic course. Thus, in some cases we observe chronic blepharitis, conjunctivitis, keratitis, otitis, while in others there are ulcerative processes in the laryngeal mucous membrane, which may even lead to perforation of the cartilages and abscesses on the anterior surface of the neck; furthermore, chronic broncho-pneumonia and diarrhœa. In the latter event, intestinal ulcers may develop, which may be cured by local measures if favorably situated in the rectum. Chronic broncho-pneumonia is undoubtedly the most frequent sequel. In a series of cases it terminates fatally after a number of months, under increasing emaciation and hectic, and we then find, on autopsy, either chronic broncho-pneumonia with dilatation of the bronchi and small pulmonary abscesses, which have been formed by the destruction of the alveolar walls and coalescence of the pulmonary vesicles which were filled with pus, or more frequently cheesy degeneration of the lungs and bronchial glands. The opinion that measles has a special tendency to the production of tuberculosis depends, as I believe, upon the fact that this disease, like whooping-cough, on account of its frequent complication with broncho-

pneumonia, especially in predisposed individuals, may give rise to cheesy processes in the lungs, followed by miliary tuberculosis.

Among the sequelæ of measles are also included diseases of the skin, eczema, impetigo, ecthyma, but especially gangrenous affections, which occur even more frequently than after scarlatina. Cases of noma, gangrene of the pharynx and lungs, are not infrequently observed a few weeks after the eruption in feeble children under poor surroundings. I have observed but a single case of noma, more frequently gangrene of the skin, the subcutaneous connective tissue, the cartilages of the ear and nose, which were followed, in case of recovery, by small defects in these parts.

As in other infectious diseases, cloudy swelling of the cortex of the kidney and fatty liver are by no means infrequent, but nephritis, analogous to that occurring in scarlatina, is of rare occurrence. Experience has taught me that many cases of nephritis which were regarded as due to measles were really caused by scarlatina. I have observed but few cases of nephritis, one of which was verified on autopsy. I have observed purpura in one case as a sequel of measles (page 274). Three weeks after the eruption, which had been associated with bloody diarrhœa, hemorrhages suddenly occurred from the mouth, nose, ears, and intestines, numerous petechiæ appeared upon the skin, and the left palpebral conjunctiva was suffused with blood. There was complete euphoria at the time; the further course is unknown.

There is no doubt that an individual may be twice affected with measles, though the number of these cases is greatly over-estimated.

A boy, aged thirteen, had measles in 1872 (under my observation). In November, 1876, a second attack, with the characteristic prodromata; severe fever, with crisis on the third day; catarrh, photophobia, etc. Eruption strongly marked on the face and trunk, slightly on the extremities.

But such cases are decidedly exceptional. The majority of those spoken of by the laity depend upon confusion with similar exanthemata, described as false measles (morbilli spurii). But we must remember that this term refers to a number of affections, a common feature of which is a papular eruption like measles, appearing in small patches. I will merely call your attention to the erythemata, especially common in the spring, and which are known as roseola vernalis, autumnalis, æstiva, infantilis; these eruptions are not always purely macular, but may appear with flat, central elevations, and are often mistaken for measles. The differentiation depends chiefly on the characteristic temperature curve and the catarrh of the respiratory and pharyngeal mucous membrane—two factors which are absent in erythema and roseola.

This appears to me to be the most appropriate place to say a few words with regard to the affection long described under the term "röthein." While some physicians regard it as an independent infectious disease, occurring epidemically, or more frequently endemically, which is characterized by scarcely perceptible fever (occurring, as a rule, only at the beginning), occasionally by mild catarrhal symptoms, but especially by an eruption consisting of small red dots—their opponents maintain that these cases are nothing more than very mild, almost apyrexial forms of measles, or even of scarlatina. My own experience does not justify me in giving a decisive opinion concerning this question; but epidemics

or endemics of this kind have been described by Steiner, Thomas, Nymann, Roth.

The susceptibility to measles is present at all ages, most marked between two and six years of age, least marked in the new-born and infants. Other existing diseases afford no protection against infection with measles; some, like varicella and whooping-cough, appear to create a predisposition to infection with this disease. The prodromal and eruptive stages of measles may give rise to infection. While the majority of children are infected on the first or second contact, some children are not affected until an association of three or four weeks with those suffering from measles. I have never observed absolute immunity against infection, such as sometimes occurs in scarlatina, nor have I ever seen an example of morbilli sine exanthemate.

The disease merely requires, in the way of treatment, that the patient be kept in bed, the room being at a temperature of 15°–16° (warmer than in scarlatina), with light covering, and a diet consisting of milk, soup, and cool drinks, as long as the high fever lasts. The room should merely be darkened to an extent agreeable to the children. I do not consider it advisable to keep them separated from the brothers and sisters, on account of the slight danger and the certainty that the children will become affected sooner or later; I would only endeavor to secure very young or sick children against infection. No medicinal treatment is required in simple cases. If the cough is violent, we may order infus. rad. ipecac., with aq. laurocerasi (P. 16), perhaps a fly-blister about an inch long over the trachea. If diarrhœa becomes profuse, and the passages occur four to six times a day, or even more frequently, we may relieve it with inf. rad. ipecac. and opium (P. 29), or subnitrate of bismuth (P. 30). Even if the course is entirely normal, I would advise you to keep the children in bed for a week, and in the room for three weeks in summer, and a month in winter.

With regard to the treatment of the complications, I refer you to the previous remarks on croup (pages 139–142), broncho-pneumonia (page 149), and diarrhœa (page 206). The following case shows the rapid success of local treatment in an ulcerative process of the rectum, which had been left over after measles:

M. S——, aged eighteen months; admitted January 30, 1877. Measles during Christmas, with bloody diarrhœa, lasting three weeks. Since then, one to two quite normal soft evacuations daily. At times tenesmus, prolapsus ani, and discharge of one to one and one-half teaspoonful of pus, mixed with blood; occasionally pure blood or muco-pus. Moderate emaciation, pallor, otherwise euphoria. Examination of rectum negative. Diagnosis.—Ulceration of upper part of rectum. Treatment.—Enema of argent. nitr. 0.1 to 60.0. Recovery after five injections.

In gangrene of the skin, the parts should be covered with charpie soaked in vinum camphoratum, a two per cent. solution of carbolic acid, or chloride of zinc, and wine and infusion of bark (P. 23) given internally.

III. Chicken-Pox.

Varicella is one of the few diseases which occurs exclusively during childhood. There can be no doubt concerning its infectious character. Although attempts at inoculation with the contents of the vesicles have been successful in but a small proportion of cases (Steiner), daily experi-

ence affords the most convincing proof of its infectious nature. The period of incubation is thirteen to fourteen days, and the eruption then appears, as a rule, without prodromal symptoms. Headache, vomiting, and fever are sometimes observed before the eruption, or conjunctivitis or angina is at times noticed; but I regard these symptoms as accidental. In one case alone I saw a diffuse erythema precede the appearance of varicella by several hours, and continue during the first day.

The eruption appears simultaneously in various parts of the body, in the shape of round, red patches, about as large as lentils, in the centre of which a vesicle, as large as a pin's head, at once forms. The enlargement of the vesicles occurs so rapidly that, after the lapse of an hour, we find them the size of a lentil or pea, filled with clear serum, and surrounded by a narrow red zone.

In a few cases the eruption consisted of red, round spots, almost all of which showed a miliary vesicle in the centre. The number of vesicles varies; at times they are isolated, at times situated closely together, especially in parts which are subject to irritation from pressure or tension of the skin. A few vesicles are very often found on the buccal mucous membrane, especially on the hard palate and the inner surface of the lips, and isolated cloudy vesicles, with injection of surrounding vesicles, are occasionally found on the conjunctiva bulbi and the genital mucous membrane of little girls.

Fever is usually present during the eruption, *i.e.*, on the first day, and occasionally on the second. As a rule, the temperature on the first day was 38.3°–38.8° (evening), and normal on the second day, or only elevated at night (38.5°, or even more); the fever was rarely higher and more protracted. Unusually high temperature is generally associated with a widespread and dense eruption of vesicles, the efflorescence surrounding which is occasionally connected by erythematous redness, and which usually undergo purulent change on the second or third day. More frequently the vesicles remain translucent, and on the third day they begin to dry and are converted into correspondingly large, brownish or blackish, thin crusts, whose red border soon disappears, fall off in one to two weeks, and leave red spots, but no cicatrices. The latter are not formed unless the intense pruritus has led the children to scratch off the crusts, as this readily causes superficial ulceration, and occasionally ecthymatous pustules and erythema in the vicinity. The eruption is not always complete on the second day, but exacerbations not infrequently occur at irregular intervals.

I must again refer to the appearance of pus in a few vesicles or in a larger number, observed in a series of cases, as these have given rise to the dispute concerning the relation of varicella to the variola group. In my opinion these affections are unconnected with one another, and I base this view on the following reasons: first, the anatomical differences in the eruption, that of varicella being vesicular from the beginning, while variola starts with red papules; furthermore, the varicella vesicles are monocular, those of variola being multilocular. But these data are not decisive, as varicella vesicles are occasionally found which are multilocular, present central umbilication, and finally become purulent. The chief difference is found in the fact that varicella presents no security whatever against variola; that varicella may develop immediately after successful vaccination, and the latter may be performed successfully shortly after the occurrence of varicella; that, finally, the cases of varicella, which appear externally like varioloid, always give rise to varicella in other individ-

uals. I have never observed a spread of the affection to the adult members of the family, which would undoubtedly, in some instance, have occurred in small-pox.

Varicella may also occur during the first few months of life, but is not more dangerous then than at a later period. I have never observed any serious complication, or death, in consequence of this disease. When combined with other infectious diseases (scarlatina, measles, diphtheria), the latter may give rise to serious consequences. Varicella is best suited, in my opinion, to show the simultaneous occurrence of two acute exanthemata, because its characteristic vesicular form contrasts strikingly with the diffuse or papulo-macular eruptions of measles and scarlatina. I have twice observed varicella in children suffering from congenital ichthyosis: once in extensive psoriasis, and once complicated with tertian intermittent fever.

Even in the mildest cases, the patients should be kept in bed for a few days, and in the room for a week.

IV. Diphtheria.

As I have previously remarked (page 269), we adhere to the theory of the specific nature of diphtheria, and cannot be forced from this clinical standpoint by purely anatomical considerations.

Had Virchow foreseen that medical opinion would again become undecided, on account of the strict separation of croupous from diphtheritic exudation—the former being situated upon the mucous membrane, the latter infiltrated into its tissues—he would perhaps have avoided the term "diphtheritic." It would be preferable to give the disease under consideration an entirely different name, for example, that of "cynanche contagiosa," proposed by Senator, because the clinical independence of the disease is clearly expressed in this manner. We shall soon see that the anatomical differentiation between the two forms of exudation often loses its significance here, and that both varieties frequently appear in combination. But, as Senator's proposition has not met with approval hitherto, we shall adhere to the term "diphtheria" for the present, and reserve the word "diphtheritic" for the infiltrated exudation.

The following description is chiefly based upon 209 carefully observed and reported cases in my wards; but I will also make use of many others occurring in private practice, but of which I only possess short notes. Among the 209 cases there were 97 girls and 99 boys (in 13 the sex is not noted). The following table shows that all ages are represented:

Age.	Number of Cases.	Age.	Number of Cases.
3 months	2	8 years	15
8 "	4	9 "	8
9 "	3	10 "	3
1 year	19	11 "	2
2 years	35	12 "	4
3 "	27	14 "	1
4 "	27	Unknown	15
5 "	25		209
6 "	13		
7 "	6		

The greatest frequency, therefore, occurs from the age of two to eight years, which agrees with the experience of others. The twenty-eight cases occurring during the first year are explained in the main by the almost unavoidable infection in the children's wards.

I could not arrive at any positive conclusion with regard to the influence of the seasons. My cases are distributed almost uniformly throughout every month. Like scarlatina, diphtheria never disappears entirely in Berlin, but sporadic cases occur at all times, their number increasing more or less at indefinite periods, and occasionally assuming a more epidemic spread. Endemics are met with especial frequency in single houses, or in a number of them, and may be attributed either to mutual infection or to a common cause. This category also includes the not infrequent endemics or epidemics in neighboring villages. We possess no positive knowledge with regard to the character of the infectious substance, which is undoubtedly communicable from one person to another, and constitutes the true cause of the disease. I will not repeat my previously expressed (page 262) objections to the bacteria theory, which is now accepted with absolute positiveness, nor trouble you with an enumeration of other poorly founded causes: for example, sewer-gas, infected water or milk. We also know practically nothing with regard to the period of incubation. From a few observations made in my wards, I am inclined to believe that the average duration is about a week; but I was never entirely certain that infection had not occurred before admission to the hospital. I therefore prefer to proceed at once to the clinical symptoms of the disease after this confession of our complete ignorance of all these factors.

In a large number of cases the diagnosis is easy from the beginning, because the symptoms at once point to an affection of the pharynx. Older children make decided complaints concerning the throat, with regard to pain in swallowing, which attract the attention of the parents. The physician finds the entire pharyngeal mucous membrane reddened to a variable degree, the tonsils swollen, and, especially on their inner surfaces which are presented to one another, covered with white or grayish white patches, which are firmly adherent and can with difficulty be removed by the brush or spatula, this being usually followed by a slight hemorrhage from the denuded mucous membrane. Exceptionally I have found the tonsils free from exudation—the velum, or perhaps the posterior wall of the pharynx, most rarely the mucous membrane of the hard palate, more or less coated from the beginning. As a rule the children have fever, but the temperature scarcely reaches as high a grade as in the initial fever of ordinary follicular angina (page 192), but usually varies from 38°–39°, with nocturnal exacerbations. Cases are not wanting in which the fever is entirely absent, at least in the beginning. Even in the first few days we can usually feel an enlargement of one or two lymphatic glands under the angle of the jaw, which may, however, be present in simple catarrhal angina, and be absent in diphtheria. The latter is rare, but I observed it in two brothers, one of whom died from croup, the other in collapse.

The differentiation of beginning diphtheria from catarrhal angina is by no means easy, and is occasionally impossible in the first day or two, so that it is wise to be cautious in giving a decisive opinion, though the patient should be isolated from the other children. The grayish yellow color and round shape of the small, isolated plugs of pus scattered over the red and swollen tonsils, are indeed quite characteristic of the catarrhal angina under discussion, as is also its appearance on one side, and

later on the other; but cases often occur in which diphtheria begins unilaterally in the same manner and with very small patches, and does not spread to the other tonsil until the following day. The differentiation becomes still more difficult if, instead of the plugs of pus referred to, grayish white shreds form upon the tonsils in catarrhal angina; they may be very similar to those of diphtheria, but are distinguished from them by the fact that they are loosely adherent to the mucous membrane, like a croupous exudation, and are chiefly composed of desquamated epithelium bound together by an amorphous, fibrinous mass. I think that these deposits are often the product of a simple inflammation of the mucous membrane, because I have not infrequently observed them at the same time or alternating with the ordinary yellowish plugs in children and adults who were predisposed to catarrhal pharyngitis. The microscope affords no positive criterion, because epithelium, amorphous matter and bacteria are found in both cases.

The diagnosis will become positive when not alone the tonsils, but the edge of the velum palati, the uvula, the angle between them, and the descending pillars of the palate, are covered in places with white patches. Other symptoms then usually occur which are often absent in the mildest form—that is, limited to the inner surface of the tonsils. But even in such cases the disturbances in swallowing may be absent or very slight, the fever moderate, general euphoria very little disturbed. I cannot lay too much stress upon this tolerance on the part of many children for diphtheria, because it often causes the disease to be entirely overlooked, and I would strongly advise to carefully examine the pharynx of every child who has fever or is changed in his manner, even though no local signs be present. My hearers have often seen that this examination revealed a previously unrecognized diphtheria. The disease then remains latent, until the sudden occurrence of dangerous symptoms or certain sequelæ proves our carelessness. While the local pharyngeal and the general symptoms may be slight, at least during the first few days, the affection of the nasal mucous membrane—diphtheritic coryza—is an important symptom in many cases. The children snore unusually during sleep, or breathe audibly through the nose; while awake, a thin, purulent secretion escapes or can be readily pressed from the nose. This discharge is especially observed during crying or other expiratory acts, and it gradually reddens and erodes the nostrils and upper lip. Diphtheria of the nares has, in my experience, the same unfavorable significance as the similar necrotic process in scarlatina (page 269). The seropurulent discharge from the nose is often mixed with blood, and even severe hemorrhages may occur. In little children who are unable to speak, this coryza may first arouse suspicion of diphtheria. The snoring during sleep may become so loud that it is similar to that occurring in croup; but it diminishes on opening the child's mouth, and thus prevents mistake.

The affection may begin with diphtheritic coryza, but the diphtheria is then rarely confined to the nasal mucous membrane, and usually extends to the pharynx, and even into the larynx. We learn that the children have suffered from coryza for eight to ten days, before the further spread, or perhaps even croupal symptoms, excited alarm. In the beginning we cannot distinguish this form of diphtheritic coryza from the ordinary form, because it is rarely possible to distinctly see the exudation. We should therefore lay special weight upon the fever, œdema of the outer part of the nose, unusually marked snoring, sero-bloody discharge from the nose,

general apathy, and a pale, exhausted appearance—a complex of symptoms which strongly favors the diphtheritic character of the coryza. Certainty is alone secured by the passage of the disease into the pharynx, or the exfoliation of membranous shreds from the nose (page 133). Much more rarely than in the nose, diphtheria begins on the mucous membrane of the lips, in the form of grayish white patches, very similar to those of aphthous stomatitis (page 186), and which I have observed thirty-six to forty-eight hours before the development of pharyngeal diphtheria. As I have not observed this form of development except in the hospital, I am inclined to suspect a diphtheritic infection of erosions of the lips previously present. In a number of cases the disease began with diphtheritic conjunctivitis, in others with diphtheritic exudation into eczematous spots upon the face or ear. Most rarely the affection began on the genitalia of little girls, and then infected the entire organism, though it may also be confined to those parts, and either run a favorable course or prove fatal.

The diphtheritic infectious material can therefore enter the organism at various places (nose, lips, conjunctiva, genitalia, external skin), but it occurs most frequently from the pharynx.

I will now return to a description of the typical form of the disease, viz., nasal and pharyngeal diphtheria. You should always be very guarded in your prognosis, however mild the disease may appear to be. In general, we may distinguish three forms—a mild, moderately severe, and severe form, though the first may be transformed at any moment into the second or third.

1. *The mild form.*—The exudation is restricted to the tonsils, the nasal mucous membrane is free, the submaxillary lymphatic glands moderately swollen. Fever is usually present, but the temperature rarely rises above 38.5°–39°, with marked remission in the morning. The frequency of the pulse varies from 120 to 144, and its quality remains good. The general condition may be very little disturbed. After an average duration of eight to twelve days, after the exudation has been gradually exfoliated, the superficial losses of substance become filled up, and dark red patches are soon the only traces left. But, after the exudation has been exfoliated, other patches may appear in its place, and the entire process thus be prolonged to two to three weeks. The urine may contain albumen, although the prognosis is not rendered sensibly worse on account of this fact.

2. *The moderately severe form.*—In addition to the tonsils, you also find the velum, uvula, and not infrequently the posterior wall of the pharynx, coated with grayish white patches. Muco-purulent secretion, flowing from the posterior nares upon the posterior wall of the pharynx, is sometimes mistaken for diphtheritic exudation, but it can be readily removed. The nasal cavity is more affected, the discharge more profuse, grayish yellow, bloody, often fœtid. There is considerable swelling of the mucous membrane of the pharynx and nose, the snoring is louder, the isthmus faucium more narrowed, though disturbances of deglutition are sometimes entirely absent. Although the fever is moderate (38°–39.5°), the general condition is much more affected, the apathy greater, and a moderate degree of somnolence is often noticeable. The appetite is lost, the tongue has a grayish yellow, rarely deep red coating, with prominent papillæ at the tip. Vomiting is frequent during the first few days. The urine is scanty, and often contains albumen, epithelium, and pale casts; but these symptoms have no prognostic significance. The voice may become more or less hoarse and even be lost, and the cough may be similar

to that of croup. We must then always be prepared for severe croup-symptoms and for tracheotomy. In quite a large number of cases, however, I have seen these symptoms gradually subside after having lasted a number of days; I have learned from post-mortem examination that serious laryngeal changes cannot be excluded under these circumstances. In a child who had presented the above-mentioned laryngeal symptoms for four to five days, but without dyspnœa, and had recovered the normal voice, and was almost entirely free from cough, death suddenly occurred in collapse, and the autopsy showed a thin, croupous exudation upon the mucous membrane of the larynx and trachea. We are therefore not always justified in diagnosing a simple catarrh, if the hoarseness and croupous cough disappear. Laryngoscopic examination, which would afford the best evidence, can rarely be performed satisfactorily. In an older boy, who could open his mouth very widely, I distinctly observed, without the use of the laryngoscope, that the free border of the epiglottis became gradually covered with a white diphtheritic membrane, and complete recovery occurred after its exfoliation.

This form of diphtheria lasts, on the average, about two weeks, but it is not infrequently prolonged to three or four weeks, in consequence of repeated exacerbations and ulcerations which heal with difficulty. The healing process, after the exfoliation of the exudation, may run an apyrexial course, but a remittent fever not infrequently continues for weeks and causes increasing weakness and emaciation. The enlargement of the submaxillary lymphatic glands usually resolves as the ulcerations heal; phlegmons and abscess-formation are much more rare than in scarlatina.

During the period of recovery, necrotic shreds are occasionally observed, partially adherent to the mucous membrane, or the children expectorate larger pieces. Even large parts of the infiltrated and necrotic tonsils may be exfoliated in this manner, and more or less profuse hemorrhages are thus occasionally produced. In a few cases I have seen the entire uvula, or at least a portion of it, destroyed by ulceration, and deep cicatricial depressions left on the free border of the velum; in three cases complete perforation of the velum palati occurred (twice unilateral, once bilateral).

3. *The severe form.*—The danger in diphtheria is chiefly due to the paralyzing influence on the cardiac nervous system, and the tendency of the process to spread from the pharynx into the air-passages. It is by no means rare to find that within a few days after the beginning of the disease, which had hitherto apparently presented no danger, the pulse suddenly becomes very frequent and small, more rarely slow and irregular, and rapidly fatal collapse occurs, or, after the pharyngeal affection has begun to resolve, croupous symptoms suddenly develop.

More rarely the diphtheria is very severe from the beginning, as shown by the high initial fever (40°) and an unusually rapid pulse (140–160), associated with apathy, somnolence, and complete anorexia. The pharyngeal affection is always combined with intense coryza and profuse fœtid secretion, œdematous swelling of the outer part of the nose, often of the eyelids, and snoring, the children breathing with open mouth. The voice is nasal, and often understood with difficulty. There is an extremely fœtid odor from the mouth, and occasional salivation; the submaxillary lymphatic glands are markedly swollen, and twice I have observed infiltration of the entire connective tissue of the submaxillary region. Hemorrhages from the nose and the ulcerated portions of the pharynx are especially severe in this form, and may hasten death by exhaustion. Petechiæ and larger spots of purpura occasionally occur in various parts, even

on the conjunctiva bulbi, and the scanty urine is rarely free from albumen and nephritic elements.

In a comparatively small number of cases I have observed roseola and erythema, which existed a few days without producing an increase of fever. I place special value on the latter circumstance, because I am convinced that the larger number of exanthemata, which have been described as diphtheritic, are nothing more than scarlatina, the beginning of which is then always associated with a characteristic rise of temperature (page 272). In three children I saw varicella develop during the course of diphtheria.

Among all the symptoms of the severe form, none is so much to be dreaded as the tendency to paralysis of the heart, as shown by collapse. The pulse becomes more rapid (160 or more) and weaker, the hands, feet, and cheeks cold, the skin and visible mucous membranes somewhat cyanotic, though the temperature does not always fall. Some children vomit during this condition, others are delirious; the majority lie in a comatose condition, and can be roused with difficulty, or not at all. The pulse finally disappears under the finger, the impulse of the heart becomes feebler, not infrequently irregular, and the second sound of the heart more indistinct. The number of respirations occasionally sinks to twenty per minute. Occasionally an unusually fœtid, even gangrenous odor from the mouth shows that true gangrene of the pharynx has occurred. Bilateral broncho-pneumonia frequently develops at the end, but can rarely be diagnosed during life.

This form of severe diphtheria, which may be termed septic, always terminates fatally, so far as my experience goes—often in a few days, at the latest after the lapse of a week. If this form develops out of the second (moderately severe), two or three weeks may elapse before death occurs. In this category I include those cases alone in which the fatal collapse occurs at the height of the affection, not those in which paralysis of the heart occurs after entire recovery of the local affection.

The spread of diphtheria from the pharynx to the epiglottis, larynx, and trachea, in the form of croup, is not as fatal as the septic form. I have observed recovery even after the process had undoubtedly spread to the medium-sized bronchi. The mild and moderately severe forms of the disease present a greater tendency to croup than the septic variety. On the average, four to six days, but often one to one and one-half week elapse before laryngeal symptoms become manifest. Occasionally the local affection of the pharynx has entirely healed before the croup-symptoms develop. On the other hand, cases are not wanting in which croup is said to have developed on the second day of the disease, or even appeared as the first symptom of diphtheria; but I believe that the diphtheritic affection of the pharynx has been overlooked in these cases, as the physician is often led astray by the hidden situation of the diphtheria, which is limited to such parts as are not open to inspection during life; this is especially true of the pyriform fossa on both sides of the root of the tongue and the epiglottis, or the posterior surface of the velum palati, the anterior surface being intact.

Pharyngeal diphtheria preceding croup is most often overlooked when the former develops as a terminal affection during severe diseases, especially tuberculosis, chronic pneumonia, typhoid fever, tubercular meningitis, enterophthisis, etc. Autopsies revealed diphtheria of the pharynx not infrequently in these children, although no symptoms had been present during life, or, at the most, fœtor oris or coryza.

I have little to say with regard to the symptoms of diphtheritic croup, as they are similar to those of pseudo-membranous laryngitis. It begins with hoarseness and a harsh cough, interrupted by sawing inspirations; I have rarely found the voice unaffected. The temperature often rises with the development of croup, while in other cases it remains almost normal. In a few cases I have found a temperature of 36.9°–37.4° shortly before tracheotomy, although the other symptoms did not justify the assumption of collapse. The frequency of the pulse is always increased (140–180); it grows rapidly smaller and not infrequently irregular. In one case the pulse, a few days after tracheotomy, assumed the characteristics of the pulsus alternans (bigeminus) described by Traube. After two pulsations rapidly following one another, a pause occurred, and then this alternation was repeated, etc., the second beat being always smaller and weaker than the first. The symptom was uninfluenced by respiration, lasted only three days, and then gradually disappeared, terminating in fatal collapse. The fatty degeneration of the heart found on autopsy cannot be regarded as the cause of this phenomenon, as the former is not infrequently found in diphtheria, with or without croup; although the pulsus bigeminus was not observed during life.

The expectoration of large shreds of membrane or of tubular casts in coughing and straining presents as unfavorable a prognosis in diphtheritic croup as in the primary form (page 141). Almost all these cases terminate fatally, although temporary amelioration occasionally awakens hope.

Bronchitis and broncho-pneumonia develop in diphtheritic croup with almost greater frequency than in the primary variety, and are manifested especially by the greater rapidity of respiration and increased fever, while the physical signs are concealed in great part by the laryngeal sounds. The expectoration of fine or bifurcated coagula points with certainty to the presence of bronchitis crouposa, which has spread to the smaller tubes. I attribute these cases, as well as those of putrid bronchitis and circumscribed gangrene of the lungs, to the aspiration of diphtheritic or gangrenous detritus of tissue from the pharynx, which may not alone give rise to inflammation, but may also directly infect the smaller bronchi and the surrounding parenchyma. A diagnosis of these conditions could not be made in the cases under my observation, as no expectoration was present, and the fœtid breath could be explained by the pharyngeal diphtheria.

The spread of diphtheria to the air-passages makes the prognosis much worse, even in otherwise mild cases. Among 106 cases of pharyngeal diphtheria, 52 recovered, and 54 died in collapse or from broncho-pneumonia and other complications. Among 100 cases which terminated in croup, 83 proved fatal; only 17 recovered—all of them, with the exception of one, on account of the performance of tracheotomy. Croup adds another danger to those already existing, as the tendency to fatal collapse continues after the spread of the disease to the air-passages, and many cases, in which the danger of suffocation is removed by operation, terminate in fatal collapse.

Post-mortem examinations on patients who died at the height of the affection often showed much more extensive and deeper changes than inspection during life would lead us to expect. Dirty gray, brownish or greenish shreds cover the posterior wall of the pharynx, the soft, more rarely the hard palate, tonsils, posterior part of the dorsum of the tongue and nasal mucous membrane, and can occasionally be traced into the lach-

rymal ducts and Eustachian tubes. The exudation is usually infiltrated into the upper layer of the mucous membrane in such a manner that it can be removed only by scraping it with the scalpel, thus giving rise to a loss of substance. This infiltration may also occur in the epiglottis and the upper half of the larynx as far as the vocal cords, while lower down in the air-passages the exudation is loosely adherent to the mucous membrane, and can be readily removed. This change is probably due to the difference in the character of the epithelium in these situations; exceptionally the pharynx and tonsils are covered with a more or less thick membrane, which can be readily detached. On the other hand, I observed several cases in which a yellowish exudation was *infiltrated* into the mucous membrane of the trachea, and even the bronchi, and could only be removed by scraping with the knife, thus giving rise to a loss of tissue. It is thus evident that both forms of exudation may occur in diphtheria, and that its character does not always depend upon the form of epithelium (pavement or cylindrical).

After removal of the exudation the mucous membrane of the pharynx and upper respiratory passages appears either pale, or, more often, reddened, cyanotic, and œdematous; the tonsils are enlarged and occasionally traversed by small, fresh or cheesy deposits of pus. I have rarely observed retropharyngeal abscesses. True gangrene of the pharynx is rare; much more often there are more or less extensive ulcerations, especially of the tonsils, which have been produced by exfoliation of the infiltrated and necrotic parts of the mucous membrane. In many cases the diphtheritic process extended to the first part of the œsophagus, the mucous membrane of which was infiltrated and hemorrhagic, or covered with numerous ulcerations. Very rarely similar changes are found in the stomach, especially in the pyloric region. Hyperplasia of the submaxillary lymphatic glands is almost constant; much more rarely there is a sero-hemorrhagic infiltration, or even a phlegmon of the surrounding connective tissue and exceptionally gangrenous disintegration. In one case I observed purulent parotitis and suppuration of the surrounding connective tissue. When the process has extended into the air-passages, the changes previously mentioned under the head of croup are noticed: infiltration and ulceration of the epiglottis and aryepiglottic ligaments, œdema of these parts, exudation and superficial ulceration of the vocal cords, membranous and tubular exudation in the larynx, trachea, and bronchi. The extent of the exudation, as well as its thickness, vary greatly. In many cases it merely forms a thin coat on the trachea, in others it forms thick white cylinders, which extend from the trachea into the bronchi. In a number of cases only one lung was affected by croupous bronchitis, the other presenting a simple catarrhal process. There may also be a true diphtheritic infiltration of the trachea and bronchial mucous membrane, as previously mentioned. Broncho-pneumonia is found almost constantly, often associated with pulmonary atelectasis, pleurisy, œdema pulmonum, and emphysema of the edges and upper lobes. In a few cases I have observed putrid bronchitis and small gangrenous spots in the parenchyma. The tracheal and bronchial glands are almost always enlarged, and sometimes infiltrated with hemorrhages. The muscular tissue of the heart is often flabby, reddish gray, and shows fatty degeneration, especially the left ventricle and the papillary muscles, while the endocardium never presented any notable changes apart from small ecchymoses, which are also found in various other parts of the body. The liver is usually fatty, the kidneys almost always in a condition of paren-

chymatous nephritis, the mesenteric glands and Peyer's patches moderately swollen. Sprue is often found in the pharynx and œsophagus, and once on the gastric mucous membrane.

The usual interpretation of the diphtheritic product as a fibrinous exudation, with profuse nuclear proliferation into the superficial layers of the mucous membrane, is opposed by another, which considers the chief factor to be a peculiar degeneration of the epithelium. Then comes the parasitic theory, although no positive proofs are advanced that the bacteria are really the specific elements of infection.

In a few cases I have observed otorrhœa and difficulty of hearing from extension of the affection to the middle ear, and once chorea, as sequels of diphtheria. But nephritis and paralysis merit our especial attention. The former is often manifested at the height of the affection by albuminuria and nephritic elements in the scanty urine, but may also continue after recovery from diphtheria. It begins most infrequently during convalescence. In only one case have I seen the diphtheritic renal affection terminate fatally in rapidly increasing collapse; but I believe that, in this case, not the nephritis, but the entirely independent collapse, must be regarded as the cause of death. Unfortunately such a termination is not rare, and may occur even two or three weeks after the recovery of diphtheria:

C. R——, aged nine. November, 1873, suffered from moderately severe diphtheria, which was relieved in ten days. The child recovered her appetite and was looking well. On the twelfth day after recovery I found the child playing in bed, but with an extremely small and rapid pulse (144). Despite the use of stimulants, the frequency of the pulse increased, the child grew weak, the extremities became cool, the pulse imperceptible, respirations irregular, and death occurred on the following day.

I have observed other similar cases, and I therefore consider it the duty of the physician not to give an absolutely favorable prognosis, even in apparently favorable cases, until two or three weeks have elapsed since recovery. This is also true of the cases in which it had been necessary to perform tracheotomy. Fatal collapse may occur even after closure of the tracheal wound has begun. Occasionally the scene is opened by repeated vomiting, and the pulse then becomes slow, feeble, and irregular, but more frequently it gradually grows more rapid and smaller, the extremities become cold, the skin somewhat cyanotic, and the heart-sounds, especially the second, grow gradually weaker, and often show a "galloping" rhythm. The respirations are not always dyspnœal, but are very frequent (60–70), and the children pass into an apathetic, finally comatose condition, which may continue from a few hours to three days. All of my cases proved fatal.

There is no doubt that these cases are essentially due to paralysis of the heart. Although a few autopsies which I have made verified the existence of diffuse fatty degeneration of the heart, it is nevertheless questionable whether this degeneration, which is a result of the infectious disease, should always be regarded as the cause of the fatal collapse, or whether, the structure of the organ remaining normal, this may not also be produced by paralysis of the heart directly, dependent on the nervous system (pneumogastric?). On account of the small number of autopsies at my command, I cannot decide this question, but will refer to the fact that, during the course of diphtheritic paralysis, similar symptoms on the part of the heart may develop and produce almost sudden death by syncope. Thus, I saw a boy, aged eight, become atactic two weeks after an

attack of diphtheria, and die in a few hours with severe dyspnœa, stertor, pallor, and disappearance of the pulse. This termination occurs more rarely after the threatening symptoms have lasted several days:

O. M——, aged nine. Examined October 3, 1876. Diphtheria five weeks previously, followed by paralysis of the velum. This had almost recovered, when ataxia and paresis of the lower extremities suddenly developed a few days ago. The arms are also weaker than in the normal condition. The pulse is extremely frequent (150 and more) and very irregular. Paresis of the left abducens nerve. Mobility of the limbs slowly improved, but the pulse remained unchanged; it then became smaller and more rapid, and, on the seventh day from the beginning of threatening symptoms, collapse occurred, and soon proved fatal.

Concerning the character of one of the most frequent sequelæ, viz., diphtheritic paralysis, we know practically nothing. It is very improbable that the paralysis is due to a merely muscular affection, whether it is a degeneration of the primitive bundles or the migration of bacteria. It is more probably due to an affection of the nervous system. Déjerine[1] found, in the anterior roots of the spinal nerves and in many peripheral nerves, a formation of fatty granules and disappearance of the axis-cylinders, in addition to atrophy of the ganglion-cells of the anterior horns, and increase of the interstitial connective tissue (parenchymatous neuritis and myelitis).

Diphtheritic paralysis, which is most frequent after the milder forms of the disease, begins, as a rule, with paralysis of the velum two or three weeks after the termination of the disease, and this is often the sole paralytic symptom. The children acquire a nasal, more or less indistinct speech, and a portion of the ingested fluids are immediately regurgitated through the nose. On examination the velum palati is found to be flaccid and almost or entirely immovable in inspiration and phonation, so that it is unable to separate the pharynx from the nasal cavity. I have occasionally found the velum insensible to touch, and no reflex movements could be produced in this manner. The paralysis is very frequently associated with disturbances of vision, especially an inability to read distinctly or to recognize objects at a certain distance, and spots of light before the eyes, symptoms which are due to a disorder of accommodation from paralysis of the musc. tensor choroideæ. The movements of the iris are usually retarded or entirely abolished. Near vision is especially interfered with. The majority of these children are somewhat anæmic, and perhaps present traces of albumen in the urine. Recovery often occurs spontaneously in the course of a few weeks.

In another series of cases the paralysis is more extensive, but almost always begins with paralysis of the velum palati and accommodation; much more rarely the latter is entirely absent or has recovered when other parts of the body become affected. I have quite often seen paresis of the muscles of the neck, so that the head dropped forward and could be raised with difficulty. Ataxia and weakness of the lower limbs are often noticeable, interfering more or less with standing and walking. The paresis and ataxia very rarely increase to complete paralysis, which may also affect the upper limbs. I have never observed these complete paralyses, or the hemiplegic and alternate paralysis, or that of cerebral nerves, to which reference is occasionally made. In one boy I observed paralytic aphonia, and, in several cases, paralysis of the respiratory mus-

[1] Jahrb. f K., XIII., S. 132. 1878.

cles, respiration being very superficial, dyspnœal, and frequent. Under such circumstances, severe bronchial catarrh may prove fatal from suffocation, so that paralysis of the respiratory muscles must be regarded as the most dangerous form of diphtheritic paralysis. I have found paralysis of the sphincters in the last stage of the disease alone, but have never noticed sensory disturbances, whether anæsthesia, analgesia, a feeling of coldness, or hyperæsthesia. But, as the recognition of these conditions in childhood is very difficult, and, indeed, usually impossible in very young children, I will not deny that the observations of this kind made in adults may be founded on fact. The electrical irritability and nutrition of the paralyzed muscles in my cases were unchanged, even after the paralysis had lasted for a long time.

This detailed description of the disease unfortunately does not correspond to the remarks which will be made concerning treatment. I must repeat to-day what I said as early as 1874:[1] "In my experience all remedies hitherto recommended (and I believe I have tried almost all with the exception of sulphur preparations) are entirely useless in severe cases of the disease, and these alone should be considered, since the milder ones recover spontaneously." I believe that all experienced physicians will agree with this statement. I will therefore confine myself to a description which seems to me the most rational, though I do not attribute to it any specific action on the diphtheria.[2] Locally I use gargles alone, or, in small children, injections of the pharyngeal and nasal cavities with a solution of chlorate of potash (10 : 500), alumen aceticum (25 : 500), carbolic acid (1–2 per cent.), or aq. calcis and aq. destil. āā an. These remedies are employed most satisfactorily with a spray-apparatus, as it can readily be forced between the teeth if the children resist. The object of this method is to remove decomposed masses as thoroughly as possible, to abolish the fœtor, and to gradually loosen and dissolve firmly adherent exudations. For this reason the method recommended by Rigauer,[3] of pouring four or five teaspoonfuls of cool water into the nose daily, is certainly advisable. I do not approve of pencilling the affected parts with any fluid, as this almost always excites the patient, and, if done with force, may injure the mucous membrane. Such injuries must be avoided at any cost, on account of the almost unavoidable diphtheritic infection of all lesions of the mucous membrane. For the same reason I have long since ceased cauterization of the pharynx. Even the most enthusiastic advocate of the bacteria theory will admit that their local destruction, if at all possible, can prove useful at the time of infection alone, but not at a time when the bacteria have already migrated into the lymphatic and vascular channels.

If the pharynx shows marked inflammatory redness and swelling, I apply an ice-bag to the throat and allow small pieces of ice to be frequently swallowed in order to moderate the inflammation. Internally I would recommend, from the beginning, decoct. chinæ (5 or 10 : 120) with aq. chlori. (10.0–15.0), in addition to nutritious diet (milk, broths, wine),

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[1] Charité-Annalen, Bd. I., S. 589.

[2] Pilocarpin, which has been so much extolled recently, is also useless, according to my experience. I have had no personal experience with large doses of oil of turpentine, which has been recently recommended, but I place as little faith in this remedy as in others (Guttmann: Berl. klin.-Wschr., No. 40; Bosse: ibid., No. 43, 1880).

[3] Die Diphtherie u. ihre Behandlung durch das kalte Nasenbad. Leipzig, 1880.

though this is often rendered impossible by the complete anorexia of many children. If no food will be taken, we must resort to nutritious enemata (meat solution, peptone, bouillon with egg, and wine).

This simple treatment, which endeavors to meet the indications of cleanliness, antiphlogosis, and stimulation, possesses at least the merit of not being dangerous. I must decidedly warn you against all methods which increase the tendency to collapse already present, especially large doses of chlorate of potash (small doses are harmless, but useless), which may also give rise to dangerous intoxication and hæmoglobinuria; also large doses of quinine and salicylate of soda. Subsulphite of soda and inhalations of bromine proved entirely useless in my hands. I have repeatedly used, without any success, carbolic acid internally, as well as subcutaneously along the hyoid bone (0.03–0.05 at a dose).

We are also entirely powerless against diphtheritic collapse, whether it occurs at the onset in the severe form or as an unexpected sequel. Camphor, musk, large doses of wine, finally the application of electricity to the pneumogastric nerve—all were useless.

Nor is there any remedy at our command which will prevent the spread of diphtheria to the air-passages. The antiphlogistic remedies which are employed in primary croup (page 142) are not only useless, but may become dangerous by their debilitating effect, and this is also true of full doses of emetics. On several occasions I thought that recovery had been produced by mercurial inunctions (1.0 ung. ciner. every two hours), and therefore employed this method in a number of cases, but with such bad results that I must regard my previous success as merely accidental. I do not deny that diphtheritic croup occasionally recovers spontaneously, and therefore also during the employment of various remedies; but such recoveries are exceptional, especially if the croupous symptoms have attained great severity. Among 100 cases of croup only one terminated favorably in this manner, 33 died without operation, and tracheotomy was performed upon 66. This operation is the sole measure from which we can expect any results, and I therefore advise its performance in all cases which are not moribund or do not present severe symptoms of infection. Although the chances are much better after the age of three years, examples of recovery are not lacking in the second, and even in the first year. It is of great importance not to perform the operation too late—after cyanosis, coolness of the extremities, and extreme orthopnœa are present. It is advisable to operate on the appearance of the local signs of laryngeal stenosis (continuous stridor in breathing, croupy cough, retraction of the lower part of the neck and the lower ribs). There is nothing to be lost by the operation, and everything to be gained. Even the presence of pneumonia is not a contraindication. We should never forget, however, that tracheotomy merely compensates the laryngeal stenosis, and renders respiration possible, but exercises no influence on the diphtheria. Among my 66 cases upon which the operation was performed, 16 recovered. The success in private practice is somewhat more favorable.

Our autopsies have shown that the chief cause of the mortality after tracheotomy is bronchitis (partially croupous) and bronchopneumonia, which must not be regarded as the result of the operation, as it often occurs in the non-croupous cases of diphtheria (page 291). Great frequency and superficial character of the respirations before the operation is always a bad sign, as it indicates the implication of the bronchi and lungs, and for the same reason the discharge of tubular or branched membranes through the canula after tracheotomy is an unfavorable

symptom. Although the symptoms of laryngeal stenosis may disappear after operation, nevertheless, if bronchitic and pneumonic complications are present, the respirations increase in frequency (60–70 per minute), the temperature remains at 39° and 40°, and death occurs within one or two days.

But if these complications are absent, the operation produces such a strikingly favorable change that the inexperienced indulge in the brightest hopes. Unfortunately they are often sadly disappointed within a few days. The previously quiet breathing becomes frequent and dyspnœal, the fever increases, and examination reveals the presence of the dreaded complication on the part of the bronchi and lungs. As is generally done, we cover the opening of the canula with a warm, moist sponge, and in many cases fill the room with vapor of water, or allow a spray of a solution of salt to be frequently made through the canula; and nevertheless a large number of the children die from croupous bronchitis and pneumonia. Life may be threatened by other dangers after the operation, such as diphtheritic infiltration of the incised wound, which occasionally gives rise to extensive necrosis of the anterior part of the neck, and erysipelas serpens, which, in several cases I have seen, spread to the chin and epigastrium, and proved fatal. Ingested fluids often escape through the canula, or, after this has been removed, through the wound; solid food can be swallowed more easily, but it also passes readily into the air-passages. This can scarcely be due to the inability of the epiglottis to close the larynx during deglutition, as the disturbance may be absent in individuals who have lost a considerable portion of the epiglottis; it is more probably due to patency of the glottis in consequence of paralysis. The introduction of Trendelenburg's canula has a favorable effect in these cases, but we have also succeeded in preserving the lives of the children, in a few instances, by the exclusive use of solid food (eggs, scraped meat) and nutritive enemata, as the disturbance of deglutition lasts only a few days, at the most a week. There is danger, in such cases, of the development of "ingestion" pneumonia (schluckpneumonie), but the termination is sometimes favorable even with a combination of unfavorable circumstances. In two cases epileptiform convulsions occurred on the second and third days after tracheotomy, and terminated fatally. Whether these should be regarded as an initial symptom of pneumonia, or as symptoms of inanition, I cannot determine; at all events, no lesion was found in the cranial cavity explanatory of the convulsions.

If the progress is favorable the canula may be removed, as a rule, on the sixth day after the operation, and as a matter of course this must first be done tentatively under the observation of the physician. The removal of the canula may be delayed by considerable swelling of the mucous membrane, especially over the arytenoid cartilages, which diminishes the lumen of the larynx. Under such circumstances there is always danger that a canula which has been in the trachea for an unusually long time may produce irritation of the mucous membrane by its pressure, or may even cause ulceration, usually situated 2–2½ ctm. below the wound, and finally give rise to polypoid proliferation. If this has occurred, the canula may not be removed, and the children must retain it for a very long time, until we succeed in removing the proliferation of the mucous membrane, and thus the danger of stenosis.

Finally, I will mention that, like all individuals suffering from wounds, the children in my wards, upon whom tracheotomy was performed, showed

an especial tendency to infection with scarlatina, which often developed within a day or two after the operation. One of these children, upon whom tracheotomy had been performed, suffered from scarlatina, broncho-pneumonia, and nephritis during the next few weeks, and during this entire period continued to discharge shreds of false membrane from the wound. Cases of this kind, in which the croupous process in the trachea and large bronchi continues for weeks after the operation, are comparatively rare, but may finally terminate favorably; examples of recovery have been reported in which croupous shreds and tubes were discharged for a period of thirty-one, even sixty-one days after the operation.[1]

In the treatment of diphtheritic paralysis, all experienced physicians agree in the recommendation of tonic treatment (good diet, ferruginous preparations, fresh air), and many cases recover in this manner after a short period. Since 1874, I have used subcutaneous injections of strychnine in a good many cases. Although formerly recommended (for example, by Trousseau, as strychnine syrup), the use of this remedy, especially in children, appears to have excited fears. But the numerous cases which have been treated by me in this manner, prove that nothing is to be feared in children with proper dosage and caution.

Two cases previously reported by me,[2] of whom one received 0.012, the other 0.02 sulphate of strychnia before recovery, prove by the rapidity of the effect, which was even noticeable after the first injections, that the strychnia really produced recovery, or at least decidedly aided it, and the experience I have had since then confirms this favorable opinion. The following may serve as an illustration:

Elise S——, aged four; admitted December 17, 1878. Diphtheria and croup six weeks previously; successful tracheotomy. For three weeks paralysis of the velum palati, and, at present, of the lower limbs. The former almost recovered; legs flaccid, immovable, the arms also weak; sensibility normal. Strychnine, 0.002, injected daily. December 21st, flexion possible at the knee. December 23d, patient could walk a little with assistance. Complete recovery at end of two weeks.

I do not deny that greater patience is required in severe cases of paralysis, and, under such circumstances, I would also advise the use of electricity in combination with the injections of strychnine.

V. Typhoid Fever.

The following description is based upon a material consisting of 137 cases, 104 of which occurred in my hospital wards, and 33 in private practice. In addition, I have short notes of another series of cases.

These figures show that the former notion, with regard to the rarity of typhoid fever in children, is erroneous.

The pathological anatomy of typhoid fever in childhood does not present the striking appearances found in adults. Rilliet called attention to the infrequency and small size of the intestinal ulcers, which he attributed to the predominance of the so-called "plaques molles," *i.e.*, the enlargement of Peyer's patches from proliferation of the lymphoid cells in the follicles, while the hard plaques, in which the proliferation also

[1] Cadet de Gassicourt: Gaz. Hebdom., No. 29, 1876; Sannée: Traité de la Diphthérie, p. 55.

[2] Berl. klin. Wochenschr., No. 17. 1875.

involves the underlying mucous membrane, were said rarely to occur. The latter are apt to ulcerate, in consequence of necrosis of the tissues, while the former are finally absorbed after fatty degeneration of the new-formed cells. It is difficult to form an opinion with regard to the truth of these statements, as the opportunities for post-mortem examination are comparatively rare. Among my 137 cases, in 10, autopsies were obtained. Of these, intestinal ulcerations were found in three, the disease having lasted from sixteen days to four weeks. In one case, the ulcerations and intervening mucous membrane presented such an appearance that we might be led to think of dysentery, were the clinical history of typhoid fever not placed beyond a doubt. All the other cases showed enlargement of Peyer's patches without ulceration. Even when ulcers were present, they appeared more shallow and smaller than in adults. To this fact must be attributed the infrequency of intestinal perforations and profuse intestinal hemorrhages in typhoid fever of childhood.

Enlargement of Peyer's patches is also present during the first two years of life, but then loses much of its significance, because Peyer's patches and the solitary follicles are often enlarged at this age in intestinal catarrh and various infectious diseases, and occasionally present signs of inflammation and even ulceration, although no typhoid symptoms were observed during life. On the other hand, these symptoms may have been present in a characteristic manner, although post-mortem examination shows either entire absence of lesions or merely inflammation of the mucous membrane of the large or small intestines (Rilliet and Barthez's entérite typhoïde). In my article on typhoid fever of childhood,[1] I reported a couple of these cases occurring in children about six months old. Diarrhœa, enlargement of the spleen, bronchial catarrh, otitis, somnolence, and the characteristic fever-curve were present, but the autopsy (in one case) showed only one Peyer's patch of areolar structure, slight swelling of a few mesenteric glands, normal mucous membrane and spleen; in addition there was broncho-pneumonia of the left lower lobe and a serous effusion between the dura mater and pia mater. How can these cases be explained? The clinical symptoms of typhoid fever are so marked that the absence of the usual anatomical appearances must step into the background. We may therefore assume that the latter may be very slightly developed or entirely absent, without justifying us in denying the typhoid character of the affection.

Children are much more rarely affected during the first two years than later. Only five of my cases occurred during this period, while the larger number varied from the age of five to nine years, 28 from three to five years, 41 from ten to fourteen years of age. The sexes were represented almost equally (73 boys and 64 girls); there appeared to be a predisposition in autumn and early winter. Of 100 cases, 43 occurred in October and November, 12 in December, and 7 in March.

The disease is very slightly, if at all, contagious. I never isolate typhoid fever patients, and have seen those in neighboring beds attacked in but a few cases, when the patients were very young and passed fæces in bed. The frequent occurrence of several cases in one family, either simultaneously or in succession, can be explained by the action of the same cause on all the patients. We are as much in the dark concerning the nature of the infectious matter as we are with reference to the other infectious diseases. Nor do we possess any positive knowledge with regard

[1] Charité-Annalen, II., S. 542. 1876.

to the manner in which the virus enters the organism. After infection has occurred, the outbreak of the disease may be aided by certain influences, prominent among which are mental excitement and severe exposure.

The benignancy of typhoid fever in childhood is recognized by all authors. Among my 137 cases only 16 proved fatal, and in 12 alone could this termination be attributed to the typhoid fever. Death occurred eight times in deep coma and collapse, once from perforation of an intestinal ulcer, once from pneumonia, once from gangrene of the lungs, and once from intestinal hemorrhage. The four other children died from newly developed diseases, or those which had already existed, so that the mortality was about seven and one-half per centum.

In mild cases the physician may be in doubt even more than in adults, whether he has to deal with true typhoid fever or with "gastric fever." Even the decisive feature, viz., the characteristic fever-curve, presents occasional variations.

The fever rarely began suddenly with a chill, followed by heat, and in one case by perspiration, and even then it was doubtful whether fever had not been previously present and had been overlooked. In a boy, aged eleven, who was in the stage of decrease with normal morning temperature, I saw a relapse develop suddenly with a severe chill. The chill was almost always immediately followed by a rapid rise of temperature, so that 40.1°–41.2° was reached in the first nights. In a few cases, the sudden rise on the first day was followed by depression on the second, and the temperature did not reach its former height until the third day. This sudden initial rise of temperature should not be regarded as an unfavorable prognostic sign.

Much more frequently there was a gradual rise of temperature, so that it only reached 40.0° in the last days of the first week. The initial chill is then absent, and at the most there are slight rigors at the period of exacerbation in the afternoon or evening. The fever then assumes a remittent, continuous type, with high evening temperature, that in the morning being about 1° lower, remains at the same height for some time, and then gradually falls into an intermittent stage, with normal morning, but febrile evening temperature. When the latter reaches the normal, the fever ceases. The following was the duration of the fever in eighty cases in which the beginning of the disease could be ascertained with a certain degree of certainty:

7 cases	7– 9 days.	15 cases	20–23 days.
4 "	10 "	6 "	28–30 "
8 "	11 "	1 case	35 "
1 case	12 "	1 "	42 "
17 cases	13–15 "	4 cases	48–49 "
5 "	16–17 "	—	
11 "	18–19 "	80	

Complete apyrexia occurred in the majority of cases at the end of the second week, next at the end of the third week. Twelve cases lasted longer, four until the end of the seventh week. Sixteen terminated during the third week, twenty cases between the seventh and twelfth days. On account of the short duration of the fever, it might have been doubtful whether the latter were not gastric fever, had not the enlargement of the spleen, the roseola and diarrhœa, rendered the diagnosis certain. While the maximum temperature in many did not go beyond 39.3°–39.8°,

and in exceptional cases did not exceed 38.2° in the morning, it varied in the majority from 40.0°–40.6° at night, and occasionally reached 41°–41.3°. On the average, the morning temperature was ½°–1° lower than the evening. More marked differences are rare without the use of antipyretic remedies. An exacerbation usually begins at midday, reaches its acme between two and three o'clock, and then decreases; at five P.M. it again begins, so that there are a midday and evening exacerbation in most cases, the former occasionally presenting a temperature ½°–1° higher than the latter. Cases in which the morning and evening temperature are almost equal are more rare, but are extremely obstinate. In five cases alone have I observed an inverse type of fever, *i.e.*, the morning temperature was higher than the evening.

The duration of the remittent continued fever varied, as a rule, from eight to twenty days; it terminated most frequently on the tenth, thirteenth, and eighteenth days. Its duration was much more rarely shorter (five or seven days), or longer (thirty-five to forty-four days). After the cessation of the acme, the temperature gradually falls to 38.8° in the morning, and 39.5° in the evening. The duration of the decreasing stage was two to four days in more than half the cases, one day in 7 cases, five to nine days in 15 cases; temporary nocturnal exacerbations to 40° may occur during this stage. This is followed by the intermittent stage, during which the morning temperature is normal or subnormal, while fever is present afternoons and evenings. In 61 cases the duration of this period varied in 37 from two to five days, in 10 it lasted but a day; in 6, an entire week; in 2, nine days; in 4, ten days; and in 2, even nineteen days. The nocturnal exacerbation reached 39.5° as a rule; very rarely 40.0°, the latter appearing to me to be connected in several cases with constipation. Overloading of the stomach, premature and prolonged upright position in bed, and emotional disturbances, may have the same effect. The intermittent stage, or even the period of decrease, was rarely absent entirely (nine times in 137 cases), or was so slightly indicated that the continued fever passed immediately into the apyrexial period by a sort of crisis, as is shown by the following illustration:

A boy, aged three. Duration of acme, seven days. Temperature: morning, 39.9°–40°; night, 40°–41.2°. Diarrhœa, somnolence, delirium, etc. On the seventh day, temperature, 39.6°; night, 40.7°; pulse, 160. On the following day, temperature: morning, 36.7°; night, 37.5°; pulse, 88. No fever after this.

With the disappearance of fever, convalescence begins, often with subnormal temperature, until the normal temperature gradually returns in consequence of increased ingestion of food and regular digestion. Ephemeral rise of temperature, and even chills, occasionally occur during this period.

The frequency of the pulse corresponds in general to the height of the temperature, but exceptions to this rule are occasionally observed. Its rapidity varies greatly, and occasionally reaches a very high number (152–180) in favorable cases. Great frequency of the pulse is not infrequently observed during convalescence; much more rare is an abnormal diminution—for example, 60—or irregularity of the pulse; in a small number of cases I observed dicrotism of the pulse. It is always small and readily compressible, especially in children umder five years of age.

The nervous symptoms of typhoid fever during childhood are much less prominent than in adults; in not a small number of cases they are

entirely absent or very slight. I have seen cases in which, of all the typhoid symptoms, the characteristic fever-curve was alone present, so that for three, or even four weeks, there was a suspicion that perhaps we had to deal with miliary tuberculosis or a subacute endocarditis. But usually certain nervous symptoms are present. Headache and apathy, slight somnolence, restlessness, moderate delirium, especially during the evening and night, difficulty of hearing, hyperæsthesia of the skin, especially over the abdomen, sleeplessness, dreams and vertigo occur most frequently. Severe nervous phenomena are rarely observed. In little children, delirium is often replaced by causeless, very violent cries, which disturb the family, particularly during the night. Liebermeister's opinion that the nervous symptoms are due solely to the action of the high temperature, is not tenable in my judgment, because, as I have previously shown,[1] these symptoms do not necessarily correspond to the height of the fever. In addition to the temperature, other causes, among which the action of the typhoid poison on the brain occupies the most prominent part, must, therefore, be taken into consideration. The older the children, the greater the tendency to severe nervous symptoms; after the age of ten I have often observed violent delirium, profound implication of the sensorium, stupor, tremor of the hands and tongue, and attempts to jump out of bed. In a few cases of fatal termination during profound coma, I have noticed that the cornea became covered with shreds of mucus, and finally grew dry and opaque; it was even perforated in one case. In the latter patient, slight twitchings of the limbs occurred during the last few days of life; these I have never observed in any other case, and they could not be explained by any abnormality of the brain found on autopsy. In a girl, aged ten, who suffered from a relapse in the fifth week of the disease, contracture of both legs and the right arm, associated with repeated gnashing of the teeth, occurred toward the last; post-mortem examination revealed a moderate amount of serum in the ventricles, œdema of the pia mater—lesions which are also found in cases of typhoid fever which did not present spastic phenomena during life. Similar appearances were found in a girl, aged four, who, during the last few days, had presented distinct rigidity of the neck, especially on raising the body.

Among the psychical disturbances apathy is the most frequent, and is often associated with mild delirium, especially during the night. I have rarely observed more serious symptoms.

In an extremely emaciated and anæmic girl, aged six, attacks of mania occurred immediately after defervescence, the child being completely apathetic during the intervals and only occasionally manifesting consciousness. Although the general impression created was that of an inanition psychosis fatal collapse could not be prevented by nourishing diet and stimulation. Other symptoms were manifested in a boy, aged twelve, during the decreasing stage (end of the second week), viz.: almost unintelligible speech, childish obstinacy, continued crying and struggling; these symptoms lasted a few days and reappeared on the occurrence of a relapse in the fifth week, associated with délire des grandeurs. The patient treated his family contemptuously, was continually talking unintelligibly, was maniacal, sleepless, and vomited almost everything that was ingested. Pulse very frequent and small, respirations rapid, limbs and tip of nose cool and cyanotic. I then gave hydrate of chloral (2.25 by enema), and in fifteen minutes the hands and feet became warm, the cyanosis disappeared, pulse and respiration became slower, and the former more vigorous. The enemata of chloral were repeated for three nights in succession, and caused rapid disappearance of the threatening symptoms.

[1] L. c., S. 567.

I may mention that this boy had always been extremely spoiled and irritable, and that the previous character of the children appears to me, in general, to exercise an influence upon their psychical condition during typhoid fever.

I have not observed true paralysis as a result of typhoid fever except in a girl, aged eleven, who, during the decreasing stage, became affected with double ptosis, paralysis of the right abducens nerve, and aphasia lasting a week, after the disappearance of which a childish condition, in which the patient often cried, continued for a number of weeks. Complete aphasia occurred in six cases, while in a few others only a single word, for example, "yes," was spoken. The aphasia always occurred during the intermittent stage or convalescence, and usually lasted one to two weeks.

In all these patients the typhoid fever had run a long and severe course, but all the children, with one exception, recovered. In two cases I observed amblyopia lasting a few days during defervescence, and which was due, in one case, to paralysis of accommodation; both children recovered. Whether all these nervous symptoms are due to distinct changes in the brain, is not known; it may be thought that the disease produces degenerative changes in the brain similar to those detected in the muscular fibres, especially of the heart, the cells of the liver, and the kidneys.

Enlargement of the spleen and roseola are among the most important signs of the typhoid fever in children as in adults. Percussion of the spleen may be entirely deceptive on account of tympanitic distention of the colon, morbid conditions of the left lung and pleura, and the restlessness of the children. In seventy-five cases alone could I examine the spleen with sufficient care and persistency, and among these it could be palpated thirty times, either projecting continuously two to three ctm. beyond the border of the ribs, or only distinctly palpable during deep inspiration; it was occasionally tender on pressure. As a rule, the severity of the case had no effect on the degree of enlargement; in all other cases the enlargement of the spleen could be detected by percussion alone, usually to the lower border of the seventh rib, rarely to the sixth rib, and percussion or pressure below the border of the ribs was sometimes painful. In many cases no enlargement could be detected. Examination of thirty cases showed that the more rapidly the temperature-curve rises, the earlier does the splenic tumor appear—sometimes as early as the third or fourth day, usually on the sixth or ninth days, or even later. It usually persisted throughout the intermittent stage. In a few cases, I did not feel it distinctly until during a relapse.

Roseola was entirely absent in eleven cases which I observed from the beginning to the end of the disease. In all the others it appeared in the same manner as in adults—*i.e.*, in the form of very scanty, pale red, slightly prominent papules, from the size of a pin's head to that of a lentil, particularly on the abdomen and lower part of the thorax, more rarely on the back and inner surface of the thighs. The number of spots was usually very small, from five to ten, or even less. In five cases there was an unusually copious eruption, which presented the greatest similarity to that of typhus fever. The roseola appears at the same time as the splenic tumor. When the rise of temperature was rapid, the roseola often appeared on the third or fifth day from the beginning of the fever; while in the large majority of cases it did not appear until the seventh or tenth day, rarely at the close of the second week. As a rule, a few new spots continued to appear during the first few days, rarely at a later period.

Although the individual spots last merely two to three days, the eruption, as a whole, often lasts until the end of the second week, on account of the appearance of fresh spots, and a pale yellowish pigmentation is then visible for a few days at the site of the roseola.

Sudamina, followed by branny desquamation, are often observed when profuse perspiration occurs with the fall of temperature. True petechiæ are rare, and do not necessarily constitute a bad prognostic sign.

Bed-sores rarely occur in children compared with adults, and I have observed but six or seven cases, none of which were severe. Small abscesses and furuncles were repeatedly observed, especially in the sacral region, on the spine of the ilium and on the abdominal walls, the necrosis of the connective tissue in one case being so deep that the abdominal muscles were laid bare.

The symptoms on the part of the digestive organs correspond with those of later life. Anorexia almost always continues into the intermittent stage, and in obstinate and spoiled children occasionally produces attacks of wild violence when the attempt is made to give the necessary nourishment. With the occurrence of convalescence, abnormally increased hunger usually becomes apparent.

The appearance of the tongue varies; exceptionally it is clean and moist, but rarely appears so dry, fissured, and covered with blackish brown sordes, as is so often found in adults. As a rule, a thick, whitish or grayish yellow coating, with marked redness of the edges and the tip, is found during the first period of the disease; or a red, smooth appearance of the anterior half is observed, while the posterior part is thickly coated. At the height of the fever the coating is often exfoliated entirely or partially, and the surface of the tongue is then of a shining red, somewhat dry and brownish in the middle, the papillæ being often quite prominent. The appearance of the tongue often varied from day to day. On the whole, its tendency to dryness, which kept pace with the severity of the nervous symptoms, especially the somnolence, appeared to me to be less marked than in adults; and this is also true of the dryness and fuliginous coating of the lips and gums, which were often entirely absent and rarely reached such a severe grade as in adults. On the other hand, the lips almost always presented shreds of epidermis, which the children gradually tore off by picking at the lips; this phenomenon is almost always constantly observed. This symptom, which belongs to the same category with picking the nose and rubbing the eyelids, is usually noticeable in the first days of typhoid fever, but may continue into the later stages. It possesses no characteristic significance, as it is very often observed during the first stage of tubercular meningitis (page 120).

Small aphthous patches, and superficial ulcers at the borders of the tongue and the angles of the mouth, occasionally lead the children to keep the fingers constantly in the mouth. Occasionally the disease was accompanied by angina tonsillaris; in one case the first attack, as well as the subsequent relapse, began with an angina, while the mucous membrane of the mouth was entirely intact. I observed the formation of sprue (page 39) in nine cases, always associated with extreme general weakness, either at the height of the affection or while the temperature was falling; generally it was found merely on the arch of the palate, but it occasionally affected the entire buccal and pharyngeal cavities; sprue of the pharynx may be mistaken for terminal diphtheria.

Vomiting occurred in sixteen of my cases, usually in the beginning and during the first week of the disease, more rarely at a later period,

and was even repeated from time to time toward the end of the disease. As a rule, it occurred after the ingestion of fluids or solids, much more rarely spontaneously, then ceased for a few days, and then reappeared without any distinct cause. At all events, vomiting occurs more frequently in children than in adults. I do not attribute to it any serious prognostic significance, though I admit that it occurs particularly in moderately severe and severe cases.

Among 112 cases, in 20 the evacuations from the bowels were entirely normal, at the most were a little softer than usual, perhaps absent on some days; in 13 cases I observed constipation throughout the entire disease, the typhoid symptoms being very severe in some of them. Constipation, which was present in the beginning, often gave place to diarrhœa at a later period. The latter was found seventy-nine times in 112 cases, either from the beginning or more frequently from the middle of the first week. The evacuations, whose number varied greatly (usually 1–5 daily, rarely 10–20), generally presented the well-known peasoup-like character, but were often greenish or like milk and coffee; they usually continued into the intermittent stage, and did not become normal until apyrexia occurred. Diarrhœa rarely recurred during convalescence, unless from some recognizable cause. More frequently there is a tendency to obstruction during this period, to which attention must be paid, as it may give rise to ephemeral attacks of fever, which cease at once on the discharge of hard scybala by castor-oil or profuse enemata (page 301).

Intestinal hemorrhages rarely occur in the typhoid fever of childhood when compared with that of adults. All authors agree in this statement, and attribute the fact to the rarity of extensive intestinal ulcerations at this period (page 293). I have observed but 5 cases of intestinal hemorrhage, in 3 of which it was insignificant, while in a boy, aged twelve, the severity of the hemorrhage, which recurred in the relapse, caused an alarming degree of prostration. In the fifth case, an insignificant hemorrhage occurred during a relapse which had developed in the third week, and was followed in the next few days by two very copious hemorrhages and fatal collapse.

I was struck by the extremely small number of cases in which the urine and fæces were passed involuntarily in bed. Almost all the children who had passed beyond the first few years of life informed the nurse of their wants, even if the apathy and somnolence were not inconsiderable. Involuntary evacuations occurred only in very severe cases, attended with profound coma. It appeared to me so much more noteworthy that, during the intermittent stage or convalescence, some of the children began to pass the urine and fæces in bed, the sensorium being perfectly clear. This could be explained by the weakness and the dread of movement connected with it. Retention of the urine is very rarely met with.

Tenderness of the cæcal region is determined with greater difficulty than in adults, and I attach as little importance to this symptom as to the gurgling, which is not infrequent in children suffering from ordinary diarrhœa. The shape of the abdomen is usually normal or moderately distended. Higher grades of meteorism are infrequent, and rarely cause interference with the diaphragm and respiration. Colic-pains, especially before the evacuations, could be determined with certainty in but thirteen cases. In two children these pains first occurred during convalescence, but with such intensity that the development of perforative peritonitis might have been suspected.

I have observed perforation of an intestinal ulcer in but one case—in a boy, aged eleven, after convalescence had begun. The infrequency of these perforations is confirmed by all authors. This is also true of parotitis, which I noticed only three times. In one case, left parotitis developed in the third week of extremely severe typhoid fever; after the pus had discharged spontaneously into the external auditory canal, it became necessary to make a counter-opening below the ear, and recovery occurred in a few weeks. In the first case the parotitis developed on the right side five days before death, and suppuration did not ensue; in the second case perforation occurred into the external auditory canal, and was associated with paralysis of the pes anserinus.

As in the adult, bronchitis is the most constant symptom on the part of the respiratory organs. Catarrhal rhonchi are, however, often absent on account of the superficial character of the respirations. In twelve cases, broncho-pneumonic infiltration could be readily detected on physical examination; lobular broncho-pneumonia, which could not be detected, may have been present in many other cases.

Broncho-pneumonia almost always occurs bilaterally in the posterior and lower portions of the lungs during the height of the disease, more rarely with the fall of the temperature or in the intermittent stage; the symptoms of consolidation may then be increased by hypostatic processes. The development of new spots of broncho-pneumonia may prolong its course and diminish the strength of the children to an alarming extent. The extreme emaciation, pale complexion, anorexia, and remittent fever, which usually continue for weeks in these cases, are apt to excite fears of cheesy degeneration of the infiltration, or of a complication with acute tuberculosis. Fortunately this dread is not always justified, and complete recovery may occur (page 148).

Croupous pneumonia develops much more rarely during the course of typhoid fever than broncho-pneumonia.

H. H——, aged thirteen, admitted November 11, 1878, suffering from epilepsy. February 4, 1879, fever began and typhoid fever developed; roseola on the third day; delirium, stupor, diarrhœa, etc. At first, only diffuse bronchial catarrh was noticed, then extensive dulness over the right side posteriorly, with bronchial breathing. February 20th, death in collapse.

Autopsy.—Brain normal. Small ecchymoses on epicardium and pleura; left lung normal. With the exception of apex, the entire right upper lobe in condition of grayish red hepatization. At the lower border are two small, cherry-red spots, surrounded by a grayish yellow line of demarcation; lower lobe also hepatized. Enlargement of spleen, parenchymatous nephritis; typhoid ulcers in intestines. Deep ulceration at the base of the arytenoid cartilage, superficial ulceration of edge of epiglottis.

The ulcerative process of the vocal cords and epiglottis, which is rare in typhoid fever of childhood, I have found in three cases. In a fourth there was no ulceration, but considerable swelling of the laryngeal mucous membrane, with inflammatory thickening of the perichondrium. All these children suffered until death from hoarseness and a hoarse cough. Hoarseness, or even aphonia, often occurred at the height of the disease, but could be overcome by greater exertion on the part of the vocal organs, and could, therefore, be attributed to atony of the muscles of phonation, or the presence of mucus on the vocal cords.

True gangrene of the lungs was observed in but one case; no gangrenous odor of the breath could be detected during life, and, as it was impossible to obtain any sputa, the diagnosis of gangrene could not be made until the autopsy.

I find no notes in my cases of abnormalities in the urine, but I will admit that the examinations could not be made with the requisite accuracy. In a girl, aged seven, I was struck by the enormous quantity and pale color of the urine; but, as it contained no sugar or other abnormal ingredients, the enormously increased thirst, which was out of proportion to the febrile movement, must be regarded as the cause of the polyuria.

Among other complications and sequelæ, I observed, in one case, facial erysipelas, which extended to the scalp and terminated in five days in a crisis. Otorrhœa, usually unilateral, occurred twelve times, continued from twelve to twenty days, and then gradually disappeared without any bad consequences. A hæmatoma of the right rectus abdominis muscle developed in the eighth week of a case of severe typhoid fever, attended with severe spontaneous pains, which were also produced on pressure and movement; it formed a hard, sharply defined tumor, terminating immediately underneath the umbilicus, and disappeared in a few weeks by absorption. Inflammation of the joints rarely occurs after typhoid fever; in one of my cases synovitis of the left wrist-joint developed three weeks after defervescence. The joint was considerably swollen, and extremely painful on motion. The moderate fever (38.2°) disappeared in two days, and the swelling rapidly subsided under the use of a splint and ice-bag; it reappeared in a few days without fever, and was relieved by tincture of iodine and a plaster-of-Paris bandage. It may be questioned whether this synovitis, which occurred three weeks after the cessation of typhoid fever, should be regarded as a sequela or as an accidental affection. I have an analogous case under observation at the present time.

Dropsy, without any urinary abnormalities, has been mentioned by various authors among the sequelæ of typhoid fever. It has been repeatedly observed in children by Stoeber, and by Rilliet and Barthez, and the cases always terminated favorably. I have observed but one case of this kind in a very marasmic boy, who became affected with œdema of the hands and feet, and moderate ascites in the fifth week of the disease, the temperature at night being constantly elevated (38.4°–38.8°); the urine showed no trace of albumen. The fever ceased at once after the incision of three large abcesses under the scalp, and under the use of Peruvian bark, with wine and nourishing diet; the dropsical symptoms gradually disappeared with the improvement in the general condition. Whether the dropsy in such cases should be regarded as atonic, *i.e.*, due to weakness of the heart and venous stasis, or as the result of parenchymatous changes in the kidneys, is unsettled. The absence of albuminuria does not exclude the latter, since this may exist without albuminuria (page 247).

Finally, a few words with regard to relapses of typhoid fever. While Rilliet and Barthez observed but 3 relapses among 111 cases, 21 instances have come under my observation among 137 cases, and among these were 6 cases in which cool baths or other cold applications had not been used. An error of diet could be proven in one case alone, but whether this should be regarded as the cause of the relapse I am unable to say, because in the 20 remaining cases this could be excluded with certainty.

The severity of the first attack affords no guarantee against the occurrence of a relapse. Although the majority of cases were of a mild type, relapse often developed after severe typhoid fever. As a rule the relapse occurred during the period of convalescence, from about the third or fifth week of the disease, after an interval of complete apyrexia the average duration of which was three to ten days, in one case even eighteen

days. In a smaller series of cases there had been no preceding period of apyrexia, but the relapse began in the form of a gradual or sudden exacerbation during the intermittent stage of the disease. After the morning temperature had, for a considerable period, been normal or subnormal, and only at night reached 38.5°–39°, a rapid exacerbation suddenly occurred and occasionally began with a chill.

O. M——, aged eleven, admitted May 13, 1878, in stage of decrease of typhoid fever. May 18th to 25th, intermittent stage; temperature: morning, 36.5°–37.3°; evening, 39.5°. May 25th, chill followed by increase of temperature; morning, 38.8°; evening, 40.0°. June 7th, apyrexia.

C. S——, aged twelve, admitted November 8, 1878, in intermittent stage of typhoid; great emaciation. Chill on November 9th and 11th; temperature: evening, 40.3° and 39.5°. Complete defervescence during the next two days. November 14th, 15th, and 16th, reappearance of high evening temperature; morning temperature normal. November 17th, the morning temperature begins to rise, the spleen grows larger and sensitive, and a relapse begins, which terminates favorably in nine days.

The beginning of the relapse in the latter case was very unusual. Attacks like intermittent fever occurred on two successive days, followed by two days of complete apyrexia. The next three days showed merely elevation of the evening temperature, and then the relapse pursued its usual course. Its symptoms correspond to those of the first attack; the roseola and the enlargement of the spleen, which has usually subsided, again make their appearance, and in a few cases the disease assumes a more serious aspect than before, so that among twenty-one cases two terminated fatally. The duration of the relapse was as follows:

2	cases	4 days.
10	"	6–9 "
7	"	11–14 "
2	"	16–17 "
21		

I will add, in conclusion, that I have, on several occasions, seen chronic exanthemata (eczema and prurigo) disappear during typhoid fever, and reappear soon after recovery from the latter. In one case apyrexia was followed by varicella, in a few others scarlatina occurred during convalescence.

It now remains to narrate to you my experience with regard to the treatment of typhoid fever in childhood. The chief importance is attached at present to the treatment of the fever. But we should never lose sight of the fact that we are treating a sick individual, not the disease in itself, and that an uncompromising adherence to a certain method will not always lead to the benefit of the patient. This is especially true of the cold-water treatment, which, according to my experience, is by no means tolerated so well by children in general as by more strongly built adults. We possess no means of ascertaining the tolerance of the child in question with regard to this treatment before beginning it, and we should, least of all, rely upon the appearance of the child, as this may lead us to entirely wrong conclusions. I have seen apparently weak, delicate children, tolerate very well the repeated employment of cool baths, while a boy, aged twelve, of very strong frame and previous good health, was so profoundly collapsed after the second bath of 20° R., that

it required an entire bottle of Tokay to give warmth to the cold hands and feet, and restore the pulse to its normal condition. In other cases collapse occurred after the first bath, or even after the child had been lying for a few hours on a water-bed. The first bath should therefore be always regarded as an experiment on which the further treatment depends. The usual method of giving a few spoonfuls of wine before and after the bath is by no means sufficient to prevent an unfavorable effect in unsuitable cases. Furthermore, cool baths during the height of the fever produce but a slight effect, which is limited, at the most, to a few hours. I now restrict the use of baths in the typhoid fever of childhood much more than formerly. I never employ them except when the temperature at night reaches 40.0° or more, and confine myself to two baths daily, their average temperature being 25° R., and never below 22°. These baths act favorably in many children by making them feel better generally, and temporarily alleviating severe nervous symptoms which may be present. The duration of each bath should not exceed five to eight minutes. Symptoms of collapse (tremor, coldness of the hands and feet, small pulse, pinched expression) may develop after the bath, and constitute a decided contraindication against their continuance. In milder cases, in which the temperature is not so high and the morning remissions are greater, I do not employ baths, but merely an ice-bag to the head and abdomen, which are usually well tolerated, but should be removed at once if the patients complain of cold. When there is great restlessness, we may employ lukewarm baths of 26–27° R., which not infrequently produce a sedative action. In milder cases, no active antipyresis, by means of drugs, is requisite. A mild fluid diet (milk, broth) and four to five spoonfuls of wine, according to age, are amply sufficient, and when a prescription must be given, as in private practice, we may administer hydrochloric acid (1:120) every two hours.

In more severe cases I often replaced cold baths by large doses of quinine (0.5–1.0) a few hours before the nocturnal exacerbation, and not infrequently employed it in combination with the baths. According to the antipyretic effect, the quinine was repeated daily, or every other day. Neither the rejection of the remedy by the stomach, nor the frequent tinnitus aurium deterred me from its further employment. During the acme, especially in severe cases, the antipyretic action of this remedy is very slight, or at least very fleeting. Only after the morning temperature begins to fall, does a decided action become manifest, and a large dose of quinine will then often depress the morning temperature of the following day to the normal, or even subnormal, position, and the depression of temperature may even continue twenty-four to thirty-six hours. I have never observed any antipyretic action from the use of quinine in small doses (0.5:120.0).

I cannot recommend the use of salicylate of soda in this affection. Although I recognize the fact that its antipyretic properties equal those of quinine, I have given up its use more and more on account of the repeated vomiting occurring after large doses, but especially on account of the threatening collapse which I have observed in a few instances.

When the diarrhœa was so profuse as to call for special treatment, relief was usually obtained from the administration of subnitrate of bismuth (0.1–0.3 every two hours) (P. 30), or tannic acid (1.0–1.5:120.0 with extr. nuc. vom. 0.1, or tinct. nuc. vom., 1.0) (P. 33). Constipation may be relieved with castor-oil or an enema of water. As a rule, no attention need be paid to the bronchial catarrh. When it was very extensive, or termi-

nated in broncho-pneumonia, decoct. senegæ, with liq. ammon. anis. (P. 20), or benzoic acid, with small doses of camphor (P. 21), was administered as a stimulating expectorant. In a few cases of extensive pneumonic infiltration, dry cups or flying blisters were employed.

As soon as symptoms of collapse become noticeable we should endeavor to counteract them by large doses of Tokay or port wine (a tablespoonful every two hours), musk, camphor (P. 14), subcutaneous injections of sulphuric ether. When great restlessness and insomnia are present, hydrate of chloral (1.0–2.0 internally or by the rectum) was often used to advantage, while morphine, whether given internally or subcutaneously, appeared to act less certainly. In all cases in which the fever continued into the second week, decoct. cort. chinæ (P. 23) was constantly administered until convalescence. Above all, you should be careful that the fluid diet is continued for a week after the cessation of fever. Milk, broth, and wine must suffice as nourishment during this period.

Neither exanthematic typhus nor relapsing fever present any peculiarities in childhood. Nor does intermittent fever in children over two or three years of age differ in any respect from that in adult life. During the first two years it presents the peculiarity that the stage of chill is either replaced by a convulsive attack or is more often entirely absent, or is merely indicated by coldness of the hands and feet and slight cyanosis. As the disease usually assumes the quotidian type, the absence of chills and the sudden occurrence of fever may lead to the erroneous diagnosis of remittent fever, and the true nature of the disease is not shown until the administration of quinine. I give this remedy in large doses (0.3–0.5 in half a wineglass of lemonade) a couple of hours before the expected attack, and, after the disappearance of the latter, continue its use for five or six days in smaller doses (0.03–0.06 every two hours, with 1.0 sugar or chocolate). But even then relapses will occur quite frequently. I do not resort to the hypodermic administration of quinine except in case of necessity, as the injections are painful and irritating.

I will here refer to a few cases which must be attributed to malaria, but obstinately resisted the administration of quinine. The children in question were five to eight years old, with the exception of one, who had not passed the second year. In all of them daily attacks of fever occurred, lasting hours, usually in the afternoon or toward evening, which either began with fever or with a mild rigor. The temperature rose during these attacks to 39.5° and more, and was not always entirely normal during the apyrexial period. Apart from these attacks the children felt well, but became pale and feeble after the disease had lasted for a number of weeks. Despite the most careful and repeated examination of all the organs, not the slightest cause for the fever could be found—not even an enlargement of the spleen—and the suspicion of a subacute endocarditis or beginning miliary tuberculosis became stronger, because the persistent use of quinine in large and small doses proved entirely useless. The blood was examined in one of these cases, but no deviation from the normal could be discovered. In two cases a change of residence produced rapid recovery. In one of these the disease reappeared after returning home, but disappeared permanently after a winter's stay on the Riviera. The source of the malaria in these cases, which lay perhaps in the dwelling, could not be determined.

LEEDS & WEST RIDING
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PART IX.

CONSTITUTIONAL DISEASES.

I. Rheumatism.

Acute articular rheumatism (polyarthritis acuta rheumatica) is now regarded by many as a process belonging to the infectious diseases. Although this is probably the correct view, I have preferred for the present to consider the disease in connection with the other forms of "rheumatism."

Acute articular rheumatism is by no means rare in childhood, and is only distinguished from that occurring in adult life by the milder character of its symptoms. The local affection, as well as the accompanying fever, are usually less severe. With rare exceptions a smaller number of joints are affected, the swelling and tenderness are slighter, and the temperature, on the average, does not rise beyond 39°–39.5°. The copious perspiration and the eruption of sudamina, which are scarcely ever absent in adults, I have rarely seen develop spontaneously in children, but usually after the administration of salicylic acid. The ankle- and knee-joints are most frequently affected, then the joints of the upper limbs, those of the fingers and metacarpal bones, the fingers and dorsal surface of the hands often presenting a slight œdematous swelling. In one case both hip-joints were painful and almost immovable. In a girl, aged five, the ankle- and wrist-joints and the right knee-joint were swollen at the same time, and the integument presented a redness which was otherwise always absent, and was only occasionally observed on the swollen finger-joints. The migration of the affection from one joint to another, and its return to a joint which had already recovered, occur repeatedly, and the course of the disease, which averages eight to ten days, may thus extend to two to four weeks, as in adults. The later exacerbations of the articular affection become shorter and milder, together with the fever, which only presents moderate elevations of temperature (38.2°), with occasional complete intermissions in the morning. A few children complain of pain in the neck on moving the head, or pain in the abdomen, with tenderness on pressure; in others an angina tonsillaris is present, with moderate disturbance of deglutition.

The majority of cases of acute rheumatism in childhood occurred between the ages of nine and thirteen years. It is much rarer between the ages of five to eight years, or even earlier. In one of my cases the child was ten months old, and the symptoms of acute rheumatic polyarthritis

(fever, painful swelling and immobility of the right wrist- and elbow-joints and the left ankle- and knee-joints) were complicated with broncho-pneumonia, and probably with left-pleurisy. After a duration of four to five weeks, a sort of crepitation could still be detected on passive motion of the right elbow, and, while the articular affection was diminishing, hardness and contracture of the adductors of the thigh developed, which slowly disappeared in three weeks, and must probably be regarded as the result of rheumatic myositis.

The complication with pneumonia and pleurisy observed in this case is much less frequent than that with endocarditis, whether occurring alone or in combination with pericarditis. Judging from my own experience, I am inclined to believe that this complication is even more frequent in children than in adults. I have also observed endocarditis in cases in which only one joint was affected. I refer you to my previous remarks on this complication (page 181), and will now merely mention that local symptoms, especially shooting pains in the region of the heart, which increase on pressure and percussion and may prevent sleep; furthermore, dyspnœa, irregularity of the pulse, and increased fever, are present in the smallest proportion of cases. The endocarditis is more often latent, and recognized on local examination alone. I have repeatedly had the opportunity, in children with old valvular disease, of observing a fresh endocarditis of the affected valve (endocarditis recurrens) during another attack of articular rheumatism.

I need not refer again to the relations of acute rheumatism to chorea, which were previously discussed (page 79). I will merely add that I have not met with cases of so-called cerebral rheumatism, such as have been occasionally observed in adults, and also by certain authors (Picot, Roger) in children, so that I am unable to give an opinion with regard to the correctness of Roger's statement that these cases are always associated with chorea.

The muscles of the throat and neck occupy the chief rank among those affected by rheumatism in children. But not every stiff neck or caput obstipum in a child should be at once regarded as a rheumatic affection; we should bear in mind that a more serious disease, especially spondylitis of the cervical vertebræ or even meningitic conditions, may give rise to this symptom.[1] But cases of caput obstipum in children are not infrequent in which constant contraction of the lateral muscles of the neck may be attributed with certainty to a cold, or at least no other cause can be ascertained, and the use of iodide of potassium, warm poultices and frictions, or the electrical current, soon produces recovery. In two children, aged twelve and fifteen months, this rheumatic contracture of the muscles of the neck and throat was complicated with broncho-pneumonia, and cases are not lacking in literature (Picot) in which a caput obstipum of this kind gave rise to chorea. Other groups of muscles—for example, the adductors of the thigh—are more rarely affected by painful rheumatic contracture. Even in little children who were unable to speak, I have observed, on a few occasions, symptoms which I could alone interpret as muscular rheumatism. These children, who had been perfectly healthy previously, suddenly refused to move an upper or a lower limb; pressure and passive movements were painful, produced violent cries, and slight œdema of the dorsal surfaces of the hands or feet was occasionally

[1] Compare a case of pure spastic contracture of the muscles of the neck, in my Beitr. zur Kinderheilk., S. 24.

noticed. The joints were not involved, but the affection spread rapidly at times from one group of muscles to another, free intervals occurred perhaps occasionally, and the disease then suddenly reappeared. Rapid recovery followed rest in bed and the application of cotton batting to the affected parts; when the lower extremity is involved, a suspicion of beginning coxitis may at first be aroused.

After the termination of acute rheumatism, especially the articular variety, the children have a great tendency to relapses, which may be repeated for a number of years in succession, increase previously existing valvular disease, and not infrequently produce relapses of chorea. After the recovery of the acute affection, I have seen vague articular pain recur from time to time for weeks, associated perhaps with slight œdema around the joints. In one case, a girl, aged ten, hydrarthrosis of the knee-joint developed and required prolonged treatment.

Chronic rheumatism occurs much more rarely in children than the acute variety; severe forms are observed exceptionally. I do not now refer to the not infrequent, previously mentioned cases in which, for months after acute articular rheumatism, fresh though mild exacerbations recur from time to time, with or without fever, and are best treated by iodide of potassium (1 : 100). I refer to permanent changes in the joints and tendinous structures, such as are met with so often in adults, but occur rarely in childhood:

A boy, aged fourteen, presented December 30, 1861. For past six years violent darting pains in hands and feet; almost complete ankylosis and nodular enlargement of the joints between the first and second phalanges of the thumb, the index and middle fingers of the left hand; similar, though not quite such severe changes in index, middle and ring fingers of right hand. Enlargement and swelling of some of the metacarpal bones. Similar changes in the great and fourth toes of left foot. Further course unknown.

In a few cases exostoses, whose number may increase with age, develop in children who have frequently suffered from acute rheumatism. This rare process is referred to by Virchow in his work on tumors. He mentions a case observed by Ebert, and another one in which, after the existence for nine months of muscular rheumatism associated with endocarditis, the right deltoid became hard, immovable, and apparently bony. Much more often no cause can be found for this tendency to the formation of multiple osteomata, occasionally observed in children; in a few cases it appears to be hereditary. The formation of the exostoses is occasionally associated with ossification of the tendons and muscles, and may attain such a high grade that a large part of the muscular and tendinous system is converted into a rigid bony mass, and renders almost every movement of the body impossible. In a few cases (Skinner[1]) it is said that every contusion gave rise to an osseous formation in the muscles, attended with fever and pain.

I have nothing to add with regard to the treatment of rheumatism. In acute cases I employed, with the same success as in adults, either salicylic acid 0.2–0.3 every two hours in wafers, or still more frequently salicylate of soda (5 : 120, a tablespoonful every two hours[2]), in chronic cases especially.

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[1] Bouchut: Maladies des Enfants, p. 903.

[2] In discussing purpura, I shall refer to certain changes of the external skin, which occasionally develop after rheumatic affections.

II. Anæmia.

The diminution of the red blood-globules and hæmoglobulin, which we term anæmia, is extremely frequent in childhood, especially in consequence of all diseases attended with losses of secretion. Children suffering from chronic diarrhœa, extensive tuberculosis, scarlatinous nephritis, are always anæmic. Those who have become atrophic in consequence of deficient nutrition, or who live in crowded rooms or in the damp air of cellars, also present the external appearances of impoverishment of the blood. We will not discuss these cases, in which the anæmia only possesses secondary importance, but rather that form which develops in otherwise healthy children and presents in general the same symptoms as the chlorosis of puberty. This form of anæmia is not infrequently observed in children from eight to ten years old, and almost as frequently in boys as in girls. Every physician is acquainted with such cases, which are presented by the anxious parents with the statement that nothing ails the children but the lack of a healthy complexion. The yellowish, pale color of the skin does not always correspond to a similar lack of color in the visible mucous membranes, which may appear quite red. But the children almost always present an unusual flaccidity, and are readily fatigued, with moodiness or increased nervous excitability, often with loss of appetite—especially for meat, while the well-known pica of chlorotic individuals rarely occurs. Frequent complaint is made of painful sensations in the region of the stomach or in the intercostal spaces, although no material cause for these complaints can be discovered. The anæmic venous murmur in the neck is very often, though not constantly present, and is entirely like that observed in chlorotic subjects, *i.e.*, it is found chiefly or exclusively on the right side of the neck, is markedly increased by turning the head to the left or by pressure with the stethoscope, and is occasionally heard in the upper part of the right border of the sternum, along the course of the common jugular vein, as a dull roaring, sounding from within. But this murmur does not present a diagnostic significance to me unless it is also heard while the head is kept perfectly erect, since an analogous murmur may be produced by muscular pressure in healthy individuals when the head is turned to the left. I could never detect any abnormal cardiac murmurs if I used the precaution of applying the stethoscope lightly; any considerable pressure of the latter upon the costal cartilages may at once make the first sound indistinct, or cause a murmur more readily, as it appears to me, in anæmic than in healthy children.

The implication of the nervous system is often manifested by attacks of headache, more rarely by dizziness or flashes before the eyes, to which reference has been previously made in the description of migraine and its relations to excessive mental work (page 131). It has also been repeatedly mentioned that more serious neuroses (chorea, hysteria, cataleptic conditions) may develop from this cause.

Before coming under my care, almost all these children had already taken iron without any permanent benefit, because the most frequent cause of the affection, in my opinion, viz., the constant presence in the poisoned air of a large city, especially in overcrowded school-rooms, and mental overwork, can rarely be removed. When circumstances permit, it is best to remove the children altogether from the city, and to have them educated in country schools, as very little is accomplished, as a rule, by a vacation of several weeks at the sea-shore or in the mountains. If the

higher grade of the anæmia renders treatment at a watering-place necessary, the most suitable for this purpose are the iron springs of Elster, Franzenbad (especially when a dyspeptic complication is present), Pyrmont, Driburg, Schwalbach, Tarasp, or St. Moritz, in Engadin. On the other hand, I regard a stay at the sea-shore as a doubtful experiment. While it has a decidedly favorable influence in a certain number of cases, it is entirely useless in many others, or acts unfavorably, especially if the timid children are forced to bathe, and I therefore always prefer a sunny, mountainous district. The cold frictions which are so much used are as poorly tolerated by many children as cold sea- or fresh-water baths, and I think that this widespread recommendation is founded more on tradition and the endeavor to do something than upon the observation of favorable effects.

Natural or artificial mineral waters (Spa, Schwalbach, Pyrmont, etc.) are also better adapted for the internal use of iron than artificial ferruginous preparations, because they contain very small doses of iron, and are more readily digested. The black color of the fæces, often observed when iron is administered, always indicates that a portion of the metal is not absorbed, but is removed as sulphide from the intestines, and therefore presents an indication to diminish the dose. It appears to me to be immaterial whether you employ ferrum reductum, lacticum, dialysatum, or the tinctures; the chief factor is the small dose (0.03–0.05 of the solid preparations, 8–12 gtt. of the tinctures two or three times daily), and its continuance for months. In order to prevent the teeth from getting black, the remedy is best given in pills, though these can be used in older children alone. In a series of cases which obstinately resisted the use of iron, and in which the latter was not tolerated, I have had very good results from arsenic (as Fowler's solution, P. 11), and therefore advise you to try this remedy as soon as the condition of the stomach will allow.

III. Purpura.

Under this term are included various morbid conditions, the real nature of which is unknown, which present the common peculiarity of producing hemorrhages into the integument, the mucous membranes, and even the parenchyma of organs. These hemorrhages usually occur spontaneously, without an exciting cause—not, as in the congenital hemorrhagic diathesis, from injuries to the skin or the mucous membranes.

I have previously said that we must be very careful, especially in hospital practice and among the poor, not to mistake the remains of flea-bites for true patches of purpura. Especially in infectious diseases (typhoid fever, scarlatina) I was often in doubt whether the specks of blood were due to flea-bites or were caused by the disease, since, as you will remember, true petechiæ and larger hemorrhages into the skin may result from infectious processes or from endocarditis. You should therefore never fail to examine the heart in febrile purpura. In a case of endocarditis after scarlatina, which did not even present a distinct valvular murmur, but merely an indistinct first sound, I made this diagnosis, especially on account of a quite extensive purpura, and this was verified by the autopsy.

But I shall now simply discuss those hemorrhages which develop independently of a febrile, general disease, or of endocarditis, and are described as purpura simplex when they merely affect the skin, and as

purpura hemorrhagica, or morbus maculosus, when associated with hemorrhages into the mucous membranes. Unfortunately, we know nothing of the nature of these morbid conditions, or even of the anatomical causes of the numerous hemorrhages. The old view that it is due to a vice in the composition of the blood can be proven neither by chemical nor microscopical examination. In several mild cases of morbus maculosus I found the red blood-globules large, full, and in nowise changed with respect to color and number. Small forms were visible only here and there, and the number of white globules was not greater than in the normal condition. Nor has the former theory of diminished coagulability of the fibrin been confirmed, and it was therefore natural that the small blood-vessels should be held responsible. As the hemorrhages could occur from rupture of the vessels, as well as from migration of the red globules through their walls, abnormal friability of the latter was thought of, and, in fact, microscopical changes of the small arteries and capillaries, which are calculated to produce such a result, have been described by various investigators (Hayem, Straganow, and others). Although the occurrence of these changes cannot be denied, I think that they can be taken into consideration in severe and fatal cases alone. If we remember how suddenly morbus maculosus sometimes develops, and how quickly it may disappear, the assumption of any considerable structural changes in the vessels is hardly allowable in such cases, and this very fact proves that we have to deal with various conditions in this disease. The severe form depends, perhaps, upon the changes in the small vessels, while in milder cases we may think of a vaso-motor neurosis, which gives rise to stasis of blood, rupture of the walls of the vessels, or migration of red blood-globules from paralytic dilatation of the smallest vessels. The complication with slight œdema in a series of cases also favors this hypothesis.

Simple purpura, in which hemorrhages into the mucous membranes are absent, occurs occasionally in poorly nourished, anæmic, and rachitic children. It is more frequently associated with leukæmia and enlargement of the spleen (page 237). The specks of blood are then few in number, and, at the most, as large as a lentil. Purpura is observed most frequently, and at the same time more profusely, in children who also complain of pains in the limbs, especially in various joints, or the latter are perhaps swollen, or these symptoms may have been present a few days before. Numerous smaller, and larger, dark red, or bluish, round patches are especially noticed on the legs and feet, while the upper portions of the body are free, or present but few specks. They are not changed by pressure, and here and there present in the centre a papular or more diffuse hardness and prominence caused by coagulation of fibrin. Apart from the previously mentioned spontaneous pains, the tibia, small bones of the feet, and the soles are not infrequently tender on pressure, and movements of the joints are painful, so that walking may be rendered more or less difficult. Occasionally a wheal-like efflorescence (erythema nodosum) is also present, in the middle of which a bluish extravasation of blood can be seen and felt, and I have not infrequently noticed slight œdema of the dorsum of the foot, though the urine did not contain any albumen. In one boy, in whom extravasations were present on the arms and face, the eyelids, cheeks, and wings of the nose were also œdematous. After a few days the specks usually grow pale, but again form as soon as the pains or swellings in the joints reappear, or perhaps without the latter as soon as the little patients begin to walk, so that a number of weeks may elapse before recovery is complete. In the majority of cases observed

by me the affection ran an apyrexial course, was rarely associated with slight irregular elevations of temperature, and with slight or no disturbance of the general condition, and always terminated in complete recovery. In a girl, aged eleven, the pulse was only 68 per minute, and was not entirely regular, though examination of the heart revealed nothing abnormal. In another case the purpura was associated with pemphigoid vesicles as large as a pea, with sero-bloody contents.

There is at present no explanation for the undoubted connection of the purpura with the pains and swelling in the limbs and joints. It is questionable whether the term purpura rheumatica is justified, because the influence of cold and wet cannot always be demonstrated. This etiological factor is especially absent in a more complicated form, in which, in addition to the previously mentioned symptoms, vomiting, intestinal hemorrhage, and colic, are also present. I have observed five cases belonging to this variety.

A boy, aged fifteen; gastro-duodenal catarrh, with slight jaundice in consequence of indigestion. A few days later, pains in the joints of the fingers; a few days afterward, purpura upon the thighs, with colic, vomiting, and black stools. At times the colic was extremely severe; region of transverse colon tender and distended. Moderate fever. Disappearance of the symptoms in five days, but a relapse at the end of three days; convalescence in a week. Three relapses in the next few weeks, always attended with bloody stools. Finally, complete recovery.

A boy, aged four, suffering from colic, tenesmus, and scanty, bloody stools. At the same time large patches of purpura on the elbows and thighs. Improvement in three days, but new patches on scrotum and prepuce. A few days later another attack of diarrhœa, with streaks of blood and severe colic, then constipation and fresh exacerbation of purpura. Entire duration three weeks.

A healthy girl, aged twelve. For a week rheumatic pains in the limbs, followed by tenderness and swelling of the wrist- and ankle-joints, with slight fever; heart intact. A few days later purpura on the abdomen and lower extremities; very severe colic, repeated vomiting, and bloody diarrhœa. Disappearance of the symptoms in five days. Four relapses within a month; finally, complete recovery.

The purpura in these cases was always combined with colic, tenderness of the colon, vomiting, intestinal hemorrhage, and, with one exception, with rheumatic pains, the swelling of the joints being less constant. There was also a characteristic development of the symptoms in exacerbations, with intervals of several days, or even a week, so that the disease was prolonged to three to seven weeks. Fever was not constant, and, when present, was always very moderate. That these symptoms are mutually connected cannot be denied, but how this connection can be explained I am unable to state. I must leave it undecided whether we have to deal with a process similar to that described by Zimmermann[1] in an adult, viz.: stenosis of the small intestinal arteries by cellular and nuclear proliferation of the tunica adventitia and media, and consequent multiple necrosis of the intestinal mucous membrane.[2]

The application of an ice-bag to the abdomen, iced milk as nourishment, and an emulsion of almonds and oil, to which I added extr. opii (0.05 : 120.0) when the pains were severe, appeared to act best in treatment. Absolute quiet in bed is here requisite, as in ordinary purpura rheumatica. In many of the latter cases I think I have obtained good results by the administration of iodide of potassium.

The forms of purpura previously considered are distinguished from

[1] Arch. d. Heilk., Heft 2. 1874.

[2] Compare Scheby-Buch: Deutsches Arch. f. klin. Med., H. 4 u. 5. 1874.

the variety for which I would reserve the term purpura hemorrhagica or morbus maculosus, by the entire absence of pain, swelling of the joints, and the previously mentioned intestinal symptoms. We merely observe purpura and hemorrhages which are confined, in the majority of cases, to the gums and nose. Only in exceptional cases could I detect the presence of blood or albumen in the urine. We often find small extravasations of blood on the mucous membrane of the lips and cheeks; they are not situated loosely upon the mucous membrane, but are infiltrated into its superficial layer, so that a flat loss of substance is visible after their removal. In almost all of my cases the disease began suddenly in the midst of perfect health. Hemorrhagic spots of a dark red, occasionally brownish red or bluish color, from the size of a millet-seed to that of a five-cent piece, spread irregularly over the entire surface of the skin, so that it may be flecked like a leopard's skin within twenty-four to thirty-six hours. Here and there the hemorrhages are in streaks or spread out over the surface. These spots never disappear on pressure, but occasionally a red zone appears around a central spot of coagulation, and its extreme hyperæmic border may pale momentarily on pressure. If hemorrhage from the mouth occurs, the coagula situated between the teeth may impede mastication. Contact with the gums may produce hemorrhages as readily as contusion of the skin, and even scratching it with the finger-nail. Pricks of a pin bled freely, and the introduction of a hypodermic needle almost always produced quite a large infiltration of blood into the skin and subjacent connective tissue. The general condition was usually undisturbed. No enlargement of the spleen could be determined, nor abnormalities of the heart or hemorrhages into the fundus of the eye. Exacerbations of the hemorrhages rarely occurred and prolonged the duration of the affection, which usually lasted ten to fourteen days before the spots paled completely. Fever was never observed, and the temperature was not infrequently below the normal (36.9°–37.2°).

Serious accidents occurred rarely, as, for example, in one boy a profuse epistaxis occurred twice, so that a tampon was required, and in another child the extraction of a tooth gave rise to a hemorrhage lasting thirty-six hours. There is not much danger of exhaustion from profuse hemorrhages; this danger characterizes the severe form of morbus maculosus, which occurs much more rarely, and is perhaps dependent on permanent molecular changes of the small vessels. The gradual development, numerous exacerbations, chronic course, and continually increasing anæmia, distinguish this variety from the ordinary one, which usually runs an acute course; in addition, there are profuse, constantly renewed hemorrhages from various parts—the nose, mouth, stomach, intestinal canal, kidneys, external auditory canal, lungs. These rare cases may terminate fatally after lasting from a month to a year, in consequence of exhaustion complicated by anasarca and dropsy of the cavities, or suddenly by hemorrhage into a vital organ, especially the brain (page 101). During its long course there are long intervals of apparent good health, and may give rise to delusive hopes which are negatived by the sudden reappearance of the hemorrhages.

I could arrive at no definite conclusion with regard to the causes of morbus maculosus in any of my cases. The majority of the children were from eight to thirteen years old, and appeared to be otherwise perfectly healthy. It was sometimes preceded by scarlatina or measles, concerning whose relations to morbus maculosus I have previously spoken (pages 274 and 282).

The treatment of the acute, milder variety may be purely expectant, and I have entirely discontinued the use of ergotin, which I formerly recommended.[1] However, if you wish to use this remedy, it should be given internally alone (P. 44), as subcutaneous injections almost always produced considerable infiltrations of blood, which even terminated in suppuration. In the chronic form I would recommend ferruginous preparations, especially liquor ferri sesquichlorati (P. 45), and country or mountain air, but only at a moderate elevation; cold-water treatment, which gave good results in at least two of my cases, may also be tried. Individual hemorrhages, if they prove serious, must be treated according to their locality; trial may always be made of ergotin in such cases.

IV. Scrofula.

Characteristic as is the symptom-complex known under the name scrofula, we possess no knowledge of its nature. If physicians still adhere to the doctrine of a "dyscrasic" basis of the disease, although examinations of the blood have disclosed no abnormalities, this depends upon the observation of the simultaneous or successive affection of a series of organs, which permits the conclusion that we do not have to deal with simple local processes, but with a morbid cause which impairs the normal conditions of nutrition of various organs in the same manner. Whether this cause is to be sought in an anomaly of the blood or the tissue-elements, or in both together, we do not know. In its clinical relations, scrofula, to my mind, means nothing more than the simultaneous or successive occurrence of chronic inflammation in various tissues, with a striking tendency to hyperplasia of the adjacent or even more remote lymphatic glands, which are apt to terminate in cheesy degeneration and formation of abscesses in the vicinity.

The terminations of scrofulous inflammation, considered in themselves, are the same as in every other inflammation. In both we find suppuration, ulceration, sclerotic changes, etc.; in certain respects, as in the constitution of the pus, in the form of the ulcers and their cicatrices, certain differences are observed in scrofulous inflammation, though by no means sufficiently characteristic to make any pretensions to pathognomonic significance. A so much greater tendency was therefore felt to attach such a significance to a morbid product, which is especially found in various organs of scrofulous patients, and is termed "cheesy degeneration." Since, as you know (page 163), the controversy concerning the relations of cheesy degeneration to tuberculosis is still unsettled, you will not be astonished that the question whether scrofula and tuberculosis should be regarded as entirely distinct, or as closely related, or even identical morbid processes, is not definitively answered at the present day. Statistical investigations are the least satisfactory in this respect. Whoever observes sick children without prejudice will have convinced himself, I believe, that many scrofulous patients finally die from cheesy pneumonia or from general miliary tuberculosis, especially tubercular meningitis. But this undoubted predisposition of scrofulous individuals to tuberculous diseases by no means proves the identity of the two processes, but depends upon the cheesy degeneration which is so often undergone by the products of scrofulous inflammation in the glands, the

[1] Beitr. zur Kinderheilk., N. F., S. 405.

bones, or other parts, and from which a more or less general tubercular infection of the organism may develop at a later period.

The clinical history of scrofula is quite characteristic in its general outlines; the individual symptoms present numerous variations, according to the individual, the number and character of the affected tissues and organs, and according to the surroundings of the patient.

Although there are cases in which individuals with distinct signs of scrofula appear perfectly healthy and well-nourished, they are rare, and almost always occur in children in whom the disease has first developed, or is present in its mildest form. Sooner or later flaccidity of the skin and muscles, and often pallor of the integument, become noticeable, though the subcutaneous adipose tissue may be present in even greater quantities than normal. The pallor of the skin, especially the face, the expression of a diminution of red blood-globules, can be so much less regarded as characteristic, since in a certain number of cases the cheeks may have a fine red color. The older physicians recognized an erethistic and torpid scrofulous constitution: the former being characterized by dark hair and eyes, a blooming color of the delicate skin—on the whole, a pleasant exterior, indicative of mental activity; the latter being manifested by blond hair, pale blue eyes, thick nose and upper lip, sallow complexion of the bloated face, and a dull expression. There is undoubtedly considerable truth in this distinction, though numerous transitions occur from one form to the other; the so-called torpid form is by far the most common, and is most marked in those cases in which the reddened and excoriated upper lip, thickened by inflammatory infiltration and by an acrid discharge from the nose, projects like a snout beyond the lower lip, and the inflamed eyelids contract spasmodically in the light.

In many cases, enlargement of the lymphatic glands in the neck, inguinal folds, and the axillæ, appears as the first sign of scrofula. Movable glands, from the size of a pea to that of a hazel-nut, occasionally collected into masses as large as a hen's egg, or even larger, can be felt or seen under the jaw on the sides of the throat, in the upper part of the neck; they are either painless or more or less tender, especially on pressure from the outside. But we must remember that swelling of the cervical, occipital, and auricular glands not infrequently occurs independently of scrofula—for example, in consequence of dental irritation, from eczematous or impetiginous eruptions on the face, ears, or scalp, even from apparently slight wounds as after perforation of the lobes of the ears. I do not think that we are justified in forthwith assuming a scrofulous constitution in such cases, u less other and more decisive symptoms become evident. In rare cases ukæmic or ps udoleukæmic [1] enlargement of the lymphatic glands occu s in childre this must also be excluded before we can regard the umors as fulous in their nature. The latter may last for many months, even years, usually associated with other scrofulous symptoms, or they may gradually resolve. In many cases they give rise to repeated inflammation of the surrounding connective tissue, especially in the neck, with extensive, hard and painful infiltrations, which finally redden, fluctuate, and either discharge spontaneously, or are opened artificially. This tendency of the hyperplastic glandular elements to break down, to necrobiosis (cheesy degeneration) and suppuration, is,

[1] Both forms of glandular enlargement are entirely similar to those occurring in adults. In the Charité-Annalen, Bd. VII., Jahrg. 1880, I have reported an exquisite case of pseudo-lukæmia with an enormous number of lymphomata.

as Virchow has justly emphasized, an important feature in the symptomatology of scrofula, and distinguishes it from leukæmic and pseudo-leukæmic lymphomata, which usually continue unchanged until the end of life. After the discharge of the pus, rapid cicatrization rarely occurs; but in the majority of cases the opening closes superficially, and fresh, deep accumulations of pus and ichor require repeated incisions. More or less extensive ulcerations, spanned by bridge-like, reddened, and infiltrated bands of skin, with undermined edges, in the bottom of which the diseased glands may be visible, often develop from these abscesses. Such ulcerations heal with difficulty, often only after extirpation of the affected glands, and always leave band-like cicatrices, very similar to those left over after burns.

Next to the lymphatic glands we very often find the external skin and subcutaneous connective tissue affected, the latter in the form of circumscribed enlargements, which may even attain the size of a walnut; they almost always suppurate sooner or later, and are then exactly like the glandular abscesses just described. The affection of the external integument appears in the form of various chronic exanthemata, the symptoms of which correspond entirely to those of non-scrofulous eruptions, so that I may refer to the description of the latter. We most frequently observe eczema impetiginosum of the face, more rarely of other parts of the body, and ecthyma of the back, buttocks, and thighs, the latter not infrequently leaving more or less deep, sharply defined ulcerations, which heal with difficulty. Eczema impetiginodes of the external ear, the scalp, mixed with patches of erythema and red papules on the cheeks, are also often present.

The various forms of lupus occur most rarely, and, as a rule, are usually associated in children with other symptoms of scrofula; they are generally situated on the nose, more rarely on the cheeks and lips. The affected parts appear hard from infiltrated exudation, strewn with larger or smaller red or livid nodules, which either desquamate constantly (lupus exfoliativus), or break down into deep, ichorous ulcers, which grow constantly deeper and do not even spare the cartilages and bones, so that they may last for years, and, in the most favorable event, leave large losses of substance with radiating, deep cicatrices. Lupus serpiginosus is especially obstinate; a part of the ulceration may cicatrize, while the edges become infiltrated with new nodules and ulcerate. I have observed this variety a few times on the back of the hands and on the fingers.

The thickened upper lip, which often appears reddened and excoriated from an acrid nasal secretion, together with the yellowish or greenish brown crusts of eczema adherent to the face, and which are mixed with red papules, vesicles, and pustules, give the countenance a characteristic expression, which is often heightened by inflammatory swelling and redness of the eyelids, which are always firmly closed in direct sunlight. In many cases the eczematous inflammation spreads inward from the concha, and causes a sero-purulent discharge from the external auditory canal.

Among the mucous membranes, the Schneiderian membrane and the conjunctiva are most frequently affected in scrofula. Among the most common symptoms are chronic rhinitis with redness and excoriation of the nose, discharge of a sero-purulent secretion, often associated with swelling of the nose externally, the nostrils being occluded by yellowish green crusts of dried pus; also conjunctivitis with formation of phlyctenulæ at the edges of the cornea, overflow of tears, and severe photophobia. The Meibomian glands are often inflamed (blepharadenitis), the

lids being reddened, swollen, and excoriated, and becoming adherent during the night by the secretion of the glands. Fœtid, sero-purulent otorrhœa, usually bilateral, is one of the most common symptoms; it may be produced by chronic inflammation of the auditory meatus, especially the lining of the membrana tympani, or by caries of the petrous portion of the temporal bone, or rupture of glandular abscesses into the meatus. When these inflammations run a course extending over years, they may finally extend to the tissues immediately adjacent. Thus, the rhinitis not infrequently extends to the perichondrium and nasal cartilages, and to the periosteum. the convoluted and nasal bones, and may give rise to redness, swelling and tenderness of the external nose, and the discharge of stinking bloody pus mixed with necrotic pieces of bone, and gradual ulceration of the cartilaginous septum and the alæ nasi. In some cases the disease is even propagated to the ethmoid bone, and through this to the meninges with development of fatal meningitis; on the other hand, a gradual carious perforation of the hard palate may occur, producing a communication between the nasal and buccal cavities. In the same manner the inflammation of the auditory meatus may cause perforation of the membrane, and then spread to the mucous membrane of the tympanic cavity and its bony walls, and finally even to the petrous portion of the temporal bone and to the spongy substance of the mastoid process. The results of this extension are: fœtid, bloody, ichorous otorrhœa, mixed with necrotic pieces of bone, or with the entire auditory bones; deafness, swelling, and tenderness of the mastoid process and the temporal bone; finally, redness of the overlying integument, dislocation of the concha, and the formation of fistulous openings which lead into the interior of the carious mastoid process. The process may even extend farther to the labyrinth and Fallopian canal, with paralysis of the facial nerve, or it may give rise to inflammation and thrombosis of the petrosal sinus and cause death under meningitic or pyæmic symptoms. Keratitis also often develops, sometimes quite rapidly; under unfavorable circumstances this may lead to ulceration of the cornea, staphyloma and atrophy of the eye. Even in the more favorable cases, more or less extensive opacity usually remains for a long time or permanently.

It appears to me doubtful whether other mucous membranes, in addition to those mentioned, are affected by scrofula. In my own experience, scrofulous children are not subject more often than others to bronchitis, broncho-pneumonia, or diarrhœa; but it cannot be denied that these affections, in the former individuals, are especially obstinate and may become dangerous from the fact that they more readily lead to hyperplasia of the bronchial or mesenteric glands, which become cheesy and may act as the starting-point of miliary tuberculosis. We very often find, in the bodies of scrofulous individuals, enlargement and cheesy degeneration of these glands, together with cheesy spots in the lungs and numerous tubercles. I cannot admit that vaginal catarrh may also occur as a symptom of scrofula; at least, among the large number of children whom I have treated for vulvitis and vaginal catarrh, there were comparatively few scrofulous individuals, and even in these the genital affection could often be attributed with greater justice to other causes (rape, onanism, cold). On the other hand, scrofulous children appear to me to present an undoubted tendency to angina, and to hyperplasia of the tonsils resulting from the frequent recurrence of the former affection.

The osseous system is also frequently affected by scrofulous inflammation, at first in the phalanges of the fingers and toes, and in the meta-

tarsal and metacarpal bones in the form of so-called pædarthrocace. In one phalanx, not infrequently in several, a slowly growing, hard, at first insensible and normally colored swelling of an olive shape, is observed; it may remain in this condition for many months, until finally the skin becomes adherent to the bones, reddens, and is perforated by one or more fistulous openings, from which a thin, purulent secretion escapes. The metatarsal and metacarpal bones may also be affected alone or at the same time as the phalanges. The inflammation is situated primarily in the interior of the bones and in the marrow (osteomyelitis), which finally suppurates together with the adjacent layers of bone, while thin lamellæ of bone are produced by the periosteum, which soon takes part in the inflammation, but are again destroyed by the process of suppuration and absorption going on internally. These symptoms may appear in the long bones of the upper and lower limbs, while in other cases the vertebræ or the joints, especially the elbow, hip, and knee-joints, are affected. These inflammations constitute very serious sequences of scrofula, inasmuch as, on the one hand, they may finally prove fatal by their termination in suppuration, hectic and amyloid degeneration of numerous organs; and, on the other hand, may result, even in the favorable cases, in ankylosis and deformity of the joints and permanent disturbances of mobility, while the spondylitis either produces paralysis of the lower limbs and its sequences, decubitus, etc., by the extension of inflammation to the spinal membranes and the spinal cord, or leads to a fatal termination by sudden luxation of the diseased vertebræ and compression of the medulla spinalis; or finally, by the continued suppuration, the formation of so-called congestive abscesses and hectic fever. I will not enter into a description of this disease, as it is referred to in detail in all works on surgery. But I may mention that spondylitis, as well as the inflammation of the joints referred to above, are usually attributed by the parents to a traumatic cause, a fall, blow, etc. Without denying the harmful effects of these injuries, I believe that the osseous affection is often previously latent and the external cause at the most accelerates its course, while in other cases all traumatic influences may be excluded. Among all these affections of the osseous system, pædarthrocace and scrofulous caries of the long bones offer the most favorable prognosis, inasmuch as complete elimination of the necrotic portions ensues and cicatrization with a funnel-shaped retraction of the overlying skin results, while the rarer analogous affection of the sternum and ribs may become dangerous to life by its extension to the mediastinum and pleura (page 161). Finally, it may be mentioned that the cranial bones, especially the temporal, are occasionally affected by chronic inflammation, independently of the otitis externa described on the preceding page, but with the same grave terminations.

The above-mentioned inflammations of the bones may occur also in children independently of scrofula, at least without any other signs of this disease. This is not only true of pædarthrocace, which may also be of syphilitic origin, but also of other forms of multiple osteitis. Among other cases I observed a perfectly healthy child, one year old, suffer from enlargement of the epiphyses of the bones of the left forearm, the metacarpus of the left thumb, the upper epiphysis of the right radius and the left half of the frontal bone, which soon fluctuated, was incised, and continued to suppurate although the child was otherwise perfectly healthy. I do not believe that we are justified in regarding such cases as scrofulous.

The degree of scrofula and its extension to a smaller or larger number

of organs present great variations in different cases. The disease may consist exclusively of enlargement of the glands of the neck, blepharadenitis and eruptions on the head, or of otorrhœa, rhinitis, pædarthrocace, or other combinations. A number of years may elapse before these affections recover, while in many other cases the implication of the larger long bones, the verterbræ and joints threatens life, or finally the development of general tuberculosis causes death under symptoms of pulmonary phthisis, tubercular peritonitis, or meningitis. So long as the chronic inflammations affect the soft parts alone, there is no danger to life; but the prognosis becomes much more grave as soon as the bones and joints are affected, and is most serious when symptoms of the internal deposit of tubercles or of amyloid degeneration of the organs develop.

Concerning the etiology of scrofula we know as little as of its real nature. That the disease may be hereditary no one will doubt; but this factor is absent in a large number of cases, and resort is then had to various hypotheses, such as very advanced, very young, or unequal age of the parents, or dyscrasic diseases of the latter, such as hydrargyrosis, syphilis, etc. It cannot be denied that such parents will produce weak children, and, as these will become scrofulous more readily than vigorous ones, this theory possesses some justification. This is also true of defective vital conditions by which the child is surrounded. The predominance of this disease in large cities and in the poorer classes of society finds a sufficient explanation in these circumstances.

When the predisposition to scrofulous affections already exists, it may suddenly develop under the influence of an acute disease. These affections include whooping-cough, measles, small-pox, and even vaccinia, after the termination of which we often find a development of glandular enlargements, eruptions, and inflammations of the mucous membranes, from which the children had never suffered previously. This is an undeniable fact, which is known to every physician, but is not yet explained. The best possible vital conditions are necessary for successful treatment. Pure air in light and airy apartments, the removal of cold and moisture, nutritious diet, country air, gymnastic exercise, and careful attention to the functions of the skin, are more important than antiscrofulous remedies. The fulfilment of these conditions is, however, only possible in a minority of cases.

Among the drugs which are usually termed "antiscrofulous," iodine stands first according to my experience; I prefer to give it in combination with iron as syrup ferri iodati (5–12 gtt. three times a day), or with iodide of potassium (according to Lugol's recommendation) (P. 46). If no disturbances of digestion are produced, the remedy must be continued for months, but is contraindicated by the presence or suspicion of pulmonary tuberculosis. I have never observed symptoms of so-called iodism, such as are often described; at the most, there was severe coryza or erythema in the face or other parts of the body. The renowned sool-baths have also earned their reputation on account of their powerful influence upon nutritive disassimilation; but we must not expect any rapid effects from them, and should apprize the parents that only long-continued and repeated baths can prove successful. The very small quantities of iodine or bromine contained in certain sool-baths—for example, Kreuznach, Hall, and others—can scarcely be taken into consideration in the production of this effect, while the quantity of chloride of sodium and the chloride of calcium, which predominates in the mother-lye, are of the greatest importance. In all these cures the fresh air plays a chief part,

and the effect of artificial baths at home must, for this reason, always be far smaller than that of the natural baths. Only when circumstances prevent the use of the latter should we be satisfied with the use of artificial baths, in which we may employ, according to the age of the child, one to five pounds of common salt, together with one to two pounds of Kreuznach or some other mother-lye.

It appears to me to be doubtful whether the traces of iodine, contained in cod-liver oil possess any specific effect. This much-lauded remedy probably acts more as an article of nutrition in the same manner as fatty articles of diet in general, and it is therefore said by some that it may be entirely replaced by olive-oil. In my experience it is far inferior to iodine, and I would warn against too large doses, which readily interfere with digestion. Two to three tablespoonfuls daily are amply sufficient. The inunctions of cod-liver oil on the skin, which are advised by many physicians, I consider objectionable on account of the disagreeable odor and uncleanliness. Nor could I observe any good effects from other lauded remedies, such as Plummer's powders, walnut-leaves, etc.; we only lose time by the use of these articles. The improvement of the vital conditions, the use of iodine or iodide of iron, and the above-mentioned baths, are therefore the only methods from which we can expect any real effects in the treatment of scrofula in general.

In addition to this general plan of treatment, the various local affections, such as inflammations of the eye, diseases of the bones, joints, skin, and mucous membranes, present a number of indications to which I need not refer in detail, as they belong in great part to the domain of surgery and ophthalmology. But I would mention the plan, which has been recently highly recommended in pædarthrocace, of scraping off the diseased bone with a sharp instrument; this shortens its course and is therefore preferable to simple expectant treatment, which may last for many years. I do not possess sufficient personal experience concerning the recently recommended inunction treatment with potash-soap, which is said to cause resolution of scrofulous enlargement of the glands with striking rapidity;[1] at all events, if the local application of tincture of iodine prove unsuccessful, the treatment with soap (one or two spoonfuls daily, rubbed into various parts of the body) should always be tried before proceeding to the radical cure, *i.e.*, to the extirpation of the glands.

V. Rachitis.

This disease comes under observation most frequently from the age of two to three years; I expressely say that it comes under observation at this time, because the disease then usually attains its greatest development, and by many parents the aid of the physician is not sought until the appearance of very marked symptoms. But I have very often noticed that the first symptoms of rachitis develop much earlier, even during the first six months of life, especially on the cranial bones and the ribs. The attention of many parents is alone attracted by the fact that the children, who had begun to walk, lose this power, or, on the other hand, have never been able to walk or to stand alone on the feet. Much more rarely the children are able to walk alone or aided by the hand of the mother, and then have a waddling gait which has been justly compared to that of a

[1] Kappesser, Klingelhoefer, Kormann, and others.

duck. Upon examination we are at once struck by the large size of the head compared with the rest of the body, the prominence of the forehead in profile (frons quadrata), the marked deviation of the bones to the outside. Not infrequently the sagittal suture, occasionally also the coronal, run in a valley which is bounded like a hill by the thickened parietal bones. The anterior fontanelle, which is generally completely ossified by the fifteenth month in the normal condition, is more or less open in rachitic individuals, with few exceptions, until far into the second and third years; the edges of the bones are readily compressed; the sutures, especially the longitudinal, but often the lambdoid and coronal, most rarely a portion of the frontal suture, are still gaping and their edges soft and yielding. In many cases both posterior fontanelles are found to be still membranous. These symptoms, especially the large size of the head, are often regarded by the laity as signs of hydrocephalus, but this is at once negatived by the normal manner in which the head is held and the complete integrity of the psychical functions, which are often even unusually active (page 111). In only a small number of cases have I observed that the children learned to talk at an unusually late period. The development of the teeth is almost always retarded; often the first tooth does not appear until the second year, and the perforation of the various groups occurs irregularly and at unusually long intervals. In some children the teeth become yellow, streaked, blackish, soon after their appearance, on account of a deficient quantity of enamel, and finally crumble down to the edge of the jaw; but in other cases I found the teeth as well preserved as in the most healthy children. The changes in the shape of the jaw, to which Fleischmann called attention in an excellent article,[1] possess great interest. Even before and during first dentition, the lower jaw assumes a polygonal shape instead of its bow-shaped curve, "inasmuch as, from the region of the canines, both sides of the jaw have undergone an appromixation or contraction, while, in consequence of an insufficient deposit of lime salts in the anterior lamella of the middle portion of the jaw, its growth and therefore the curvature remain absent." The incisors are therefore situated alongside of one another in quite a straight line, but from the situation of the canine teeth, the lateral portions of the jaw, instead of being curved, are straight on both sides and diverge somewhat posteriorly. At the same time the lower border of the jaw is turned more to the outside, the alveolar border to the inside, so that the molars and occasionally the incisors are not vertical, but converge internally. The changes in the shape of the upper jaw described by Fleischmann are less striking, the elongation of its vertical axis being most worthy of mention. The examination of the thorax reveals even more characteristic signs. The clavicles are often strongly curved or bent at an acute angle in some part; at the place where the bony part of the ribs joins the cartilaginous portion, they present a more or less prominent nodular enlargement, distinctly visible if the tegumentary coverings are thin, and which, on closer examination, is found to consist of two swellings separated by a groove (one on the osseous, the other on the cartilaginous portion). In this manner a row of nodular enlargements forms on each side of the thorax, running from above and inward, downward and outward, and is usually termed a "rachitic rosary." In many children this is associated with flattening of the lateral portions of the chest, which, in the higher grades of the disease, may lead to convexity of this part; the parts of

[1] Klinik der Pädiatrik, II. Bd., S. 168. Wien, 1877.

the second and eighth ribs situated between the angle of the ribs and the above-mentioned enlargement of the epiphyses are strongly curved inward toward the thoracic cavity, and the lower ribs form a border which is bent outward, an appearance which, in combination with the unusually prominent sternum, makes the flattening and concavity of the lateral surfaces even more striking (pectus carinatum, chicken-breast). The statement of the parents, that the child is short of breath, is always confirmed in severe cases; the respirations are more rapid and superficial than in healthy children, and it is found that the flattened or concave lateral parts of the chest are drawn farther in during inspiration, while the inspiratory action of the diaphragm appears unusually well marked from the simultaneous retraction of the epigastrium. This is often accompanied by bronchial catarrh. The narrow, deformed thorax contrasts with the strikingly prominent abdomen, which is distended by loops of intestines filled with gas; the upper part of the abdomen is filled more than ordinarily by the liver and spleen, which are pressed downward on account of the narrowness of the thorax. The digestive functions are often entirely unaffected, while disturbances are present in a number of cases—especially a tendency to diarrhœa. Among the most important symptoms are those noticeable in the bones of the upper and lower limbs. The epiphyses of the radius and ulna, especially the former, present a more or less striking thickening and broadening, so that in severe cases, especially in emaciated children, the hand appears as if separated from the arm by a groove, while the diaphyses of the bones of the forearms present a convex curve, or even a sharp bend toward the extensor side. Still more distinct is the curve of the tibia, the epiphysis of which, like that of the fibula, is also considerably thickened. The curvature, which is concave toward the inside, is most marked as a rule in the lowest third of the bone, occasionally to such an extent as to present an appearance of pes varus. We more rarely observe curves and bends in the bones of the arm and thigh, and marked enlargement of the epiphyses at the knee- and elbow-joints. The scapulæ and pelvic bones also present, to the careful observer, a more or less marked thickening of their edges in a series of cases. Finally, spinal curvature is very often present, either skoliosis or kyphosis, most frequently of the dorsal spine with compensating lordosis of the lumbar region. This kyphotic curvature is chiefly distinguished from that due to spondylitis by the larger curve which it forms, and by its diminution or entire disappearance in abdominal decubitus, especially when moderate extension of the vertebral column is performed at the same time.

All these changes naturally present great variations with regard to degree and extent. A part of them is often found entirely absent, so that the symptomatology of rachitis is incomplete. I have found most constantly, though not always, the enlargement of the epiphyses of the ribs, which is also one of the earliest symptoms of rickets, and may even be noticeable from the age of three to five months. The signs of rachitis in the cranial bones were not infrequently absent. The circumference of the head is then not enlarged, the fontanelles and sutures are closed at the proper period, and even the development of the teeth may proceed in the normal manner. Among others, I observed a girl, aged nine months, who, despite the existence of rickets, had the first teeth at the age of six months, and had six teeth at the time of examination. Occasionally the enlargement of the epiphyses and the curvature of the bones of the extremities are predominant, while the thorax and skull are

but slightly affected. Nevertheless, I have seen a few children of this kind stand and walk at the age of fifteen to sixteen months as well as healthy children. The characteristic deformity of the thorax is most frequently and markedly developed in very young and poorly nourished children, who often suffer from bronchitis, while well-nourished, vigorous individuals present it to a slight extent or not at all. I shall soon return to the cause of all these variations.

Apart from these local differences, the general symptomatology presented by rachitic children also varies greatly. While the general condition is undisturbed and the appearance blooming in a small number of cases in which the characteristic osseous symptoms are slightly developed, the large majority reveal a more serious implication of the organism by the anæmic complexion, emaciation, flaccidity of the skin and muscles, and enlargement of the cervical, axillary, and inguinal glands. In a few cases I even observed purpura patches on the wax-colored skin. The liver occasionally projects more than normal beyond the borders of the ribs, but I could detect an enlargement of the spleen in two cases alone, in which it was associated with leukocythosis and extreme anæmia. On superficial examination the urine presents no deviations from the normal. I shall soon return to the chemical examinations, which gives varying results. The secretion of perspiration is increased in many cases, and is especially profuse on the head, so that the pillow appears wet in the morning. Only a part of the children are ill-natured and restless, and evince pain when the affected bones are compressed or the child is lifted up by the hands placed in the axillæ.

I have never observed the acute or febrile course of rachitis, such as has been described now and then. If it occurs at all, it must therefore be exceptional, since I have seen many thousand cases. When febrile movement was present I could always detect a complication, especially with bronchial catarrh. The rachitis itself always pursued a chronic course of variable duration, the extent of which can be determined with difficulty, as we rarely have the opportunity of watching its course from beginning to end. But there is no doubt that, even in the most favorable cases, many months, and perhaps years, elapse before the morbid process can be regarded as ended. The first favorable indication is the attempt on the part of the children to stand on their feet and walk, *i.e.*, the restoration or acquisition of power and firmness in the muscles and bones of the vertebral column and lower extremities. At the same time rapidly progressing ossification usually occurs in the open fontanelles and sutures, and more rapid perforation of the teeth, while the previously deficient growth in the length of the body increases perceptibly, the general appearance improves more and more, and the deformity of the vertebral column and thorax gradually disappears, unless it has attained too high a grade. Finally, the remains of the disease merely consist of the curvatures and bends of the long bones, especially in the form of "bow legs" (genu valgum), and thickening of the epiphyses, which are often noticeable after the lapse of years, but are more and more compensated by the increasing growth of the bones in length and breadth. The normal position of the permanent teeth is often influenced unfavorably by the inhibition of the growth of the jaw (page 326), and various anomalies are then observed during second dentition, viz., angular or shingle-like position of the teeth, perforation in a double row, etc. Only in very severe cases are the remains of the disease, even the thickening of the cranial bones, recognizable until the age of puberty or later, and it is at this very period of life that one of the

products of rachitis, viz., the deformed pelvis, which plays no part during childhood, may give rise to extreme danger. A description of this deformity would be out of place here, and I therefore refer the reader to the work mentioned below.[1]

While the large majority of cases of rachitis terminate in complete recovery, this favorable termination in a number is interrupted by complications. The previously mentioned predisposition to catarrh of the respiratory passages is especially dangerous in these children, because, on the one hand, their continual recurrence increases the deficiency in hæmatopoësis and the consequent weakness, and, on the other hand, a diffuse bronchitis or catarrhal pneumonia, which would be recovered from in healthy children, may very readily pursue a fatal course, on account of the narrowness of the deformed thorax. The narrowness of the lung-space, the feebleness of the inspiratory muscles and the filling of the bronchi with mucus, often produce extensive atelectasis of the pulmonary tissue, which hastens the fatal course of complications (page 136). A large proportion of rachitic patients perish in this manner. Very frequently also we observe attacks of spasm of the glottis and eclampsia, to which, as you will remember, these children are much more predisposed than healthy ones (page 67). Tuberculosis is the gravest complication, and proves fatal to a large number of rachitic children living in miserable circumstances.

I take this opportunity of referring to certain data which are important in deciding on the results of physical examination, and which, if not taken into consideration, may lead to mistakes in diagnosis. I have previously (page 3) spoken of the effect which muscular contractions may exercise on the resonance of the underlying portion of the thorax; especially in children who resist and move to and fro during the examination, dulness not infrequently appears on one side of the back of the chest, and rapidly disappears when quiet is restored and the tension of the muscles is similar on the two sides. In rachitic individuals, however, in whom there is often a skoliotic curvature of the spine, vigorous muscular contractions on the convex side—especially if the skoliosis is very much developed—may produce permanent, though moderate, dulness, and therefore only acquires significance, under such circumstances, if auscultation also shows pulmonary infiltration in the part in question. In severe grades of rachitic deformity of the thorax, a larger area of cardiac dulness may be found on the anterior surface of the left side of the chest than normally, and the impulse of the heart is also generally found beyond its usual limits—symptoms which cannot be attributed at once to hypertrophy of the organ, but may simply arise from displacement of the heart and insufficient distention of the lungs. Rilliet and Barthez have found the respiratory murmur in rachitis somewhat bronchial in character, and attribute this to the moderate compression of the lungs by the walls of the thorax which have been pressed inward. I have also often been struck with the "indefinite" character of the breathing, but I think that the above-mentioned authors have attached too little weight in its interpretation to the bronchial catarrh and atelektasis, which are so frequent in rachitis.

The anatomical changes which characterize rachitis affect the bones exclusively. They present, in general, a dark redness, which is especially

[1] Ritter v. Rittershain: Die Pathologie und Therapie des Rachitis, S. 181. Berlin, 1863.

marked in the flat cranial bones; their sharp edges and borders are blunted and rounded, the long bones are shortened and rounded like cylinders, thickened at the epiphyses, variously bent and curved longitudinally. Their consistence is considerably diminished, so that sections of the osseous tissue may be made without difficulty. The periosteum is thickened and hyperæmic, and removed from the bones with difficulty, small fragments of the latter not infrequently remaining adherent to it. The diminished consistence is most distinctly shown in the cranial bones, which also present marked congestion and thickening, especially of the anterior parts (frontal bone and a portion of the parietal bones), thus explaining the prominences noticeable during life over the frontal and parietal protuberances. Under the thickened periosteum are found finely porous, pumice-stone-like, spongy layers, which alternate, at the diaphyses of the long bones, with more compact layers situated farther internally, in such a manner that they become firmer near the medullary cavity, and more like the spongy layers of normal bone.[1]

In order to properly understand the rachitic affection of the bones, we must bear in mind the conditions of the growth of normal bone,[2] which occurs in two directions, viz., in length and in thickness—the former from the cartilaginous epiphyses, the latter from the periosteum.

a. *Longitudinal growth.*—The hyaline cartilage of the epiphyses of the long bones passes over into the spongy substance of the newly formed bone by two layers: a bluish one from 1 to 2 mm. in width, and a dull yellow one $\frac{1}{3}$ to $\frac{1}{2}$ mm. wide; the spongy substance is traversed by medullary spaces, and is filled with a very vascular marrow. In the so-called proliferation layer we find numerous large cartilage-cells arranged in longitudinal rows, while in the second layer calcification occurs, inasmuch as calcific granules are deposited in the intercellular substance surrounding the cartilage-cells, and render it opaque. The formation of medullary spaces and true osseous tissue also occurs in the same layers, but the opinions concerning this process vary greatly. In rachitis there is, on the one hand, an abnormal proliferation of the layers of cartilage, rendering them broader, and, on the other hand, the calcification of the second layer is irregular and deficient, and the formation of medullary spaces extends into the proliferating cartilage, the boundary between the layer of cartilage and spongy osseous tissue being irregular instead of straight. These processes also explain the thickening of the epiphyses and the incomplete longitudinal growth of the bones, which cause retarded growth of the children in general, and is even noticeable in many at a later period of life.

b. *Transverse growth.*—In the normal condition, the very thick and vascular periosteum adheres firmly to the bone by means of a meshed, fibrous tissue, with nucleated cells, and growth occurs from the fact that these cells develop into stellated bone-cells, and the basement-substance undergoes a hardening process. Between the lamellæ of the newly formed bone are round or elongated spaces, communicating with one another and filled with a soft, reddish marrow, in which new vessels, anastomosing with those of the periosteum and the inner portions of the bone, finally form from the marrow-cells. This process lasts as long as the long bones continue to grow in thickness, a large cavity filled with marrow forming internally from the fluidification of the osseous tissue and its absorption.

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[1] Virchow, in his Archiv, Bd. V.

[2] Compare Ritter, a.a. O. S., 27, and Rehn: Gerhardt's Handb. d. Kinderkrankh., III., S. 54, v. 1878.

In rachitis the periosteum and its cellular elements are markedly hyperplastic, the new-formed spongy lamellæ of bone imperfectly ossified, with absence or great diminution of calcific deposit. Immediately underneath the thickened periosteum are hyperæmic, marrow-holding meshes of bone, then a layer of compact substance, then another spongy mesh-work, etc., while the normal absorption from the medullary cavity continues, and the cortical layer must therefore grow gradually thinner. Entirely similar conditions are presented by the flat bones of the skull, the scapulæ, and pelvic bones. The recovery of the disease occurs from cessation of the proliferation of cartilage, which is followed by rapid calcification and ossification of the newly formed layers, which become firmer than in the normal condition (so-called eburnation or sclerosis).

These physical alterations of the osseous system are also associated with chemical changes.[1] All examinations show a more or less considerable diminution of lime salts in rachitic bones, with a decrease in their specific gravity, especially in those situated above the diaphragm, with a relative increase in the amount of water of the cartilaginous portions and the organic basis-substance. This goes hand-in-hand with a relative diminution of the entire weight of the body.

Some of the chief symptoms of rachitis can be readily explained by these anatomical and chemical changes:

1. The diminished longitudinal growth of the body, partly from interference with the normal growth of bone from the epiphyses, partly from the yielding character of the long bones of the lower limbs, which carry the weight of the body.

2. The curves, bends, and fractures of the bones, which are usually caused by pressure and other traumatic influences affecting the bones which are deficient in earthy salts. The bends affect the inner, firmer layers of the long bones alone, while the external softer ones only yield with the thickened periosteum, so that it corresponds somewhat to fracture of a willow-twig or a goose-quill. In very few of these cases have I been able to detect crepitation. The pressure of the weight of the body upon the lower limbs produces curvature and incomplete fracture of the latter, usually with the convexity internally (bow-legs), while a compression of the ribs and clavicles, especially in raising the child by grasping him around the thorax, may cause fracture of these bones. Falling on the floor, or even turning in bed, may have similar results, and we thus not infrequently find more or less acute-angled, partial fractures of the clavicles, ribs, forearms, thighs, etc., which have usually healed by callus-formation; they are either isolated or multiple, in which event deplorable deformities are produced. It is probable that similar results may be produced by vigorous muscular contractions. Among other cases I remember that of a very rachitic child, aged seven months, who, after very violent and repeated convulsions, presented a fracture of both radii; but I will acknowledge that we can never, with certainty, exclude other traumatic causes in such cases.

Fleischmann is probably right in attributing the changes in the shape of the jaw to contraction of the mylohyoid and masseter muscles. But the characteristic deformity of the thorax is not the result of any single cause—for example, of paralysis or atrophy of the inspiratory muscles (serrati, intercostals, etc.), but of "the combined action of an entire series of factors," among which we may specially mention the pressure of

[1] Friedleben: Jahrb. für Kinderheilk., Bd. III. Wien, 1860.

the external atmosphere, contraction of the diaphragm, and softness of the bones of the thorax. Even in healthy, vigorous children, we may notice that in very strong and rapidly following contractions of the diaphragm—for example, in hiccoughing—the most yielding parts of the thoracic walls, *i.e.*, the anterior parts of the ribs, are distinctly retracted at the beginning of every inspiration. The yielding character of these parts in the child, combined with the comparatively feeble and incomplete inspiration, is the cause of this symptom, inasmuch as the quantity of air inspired is insufficient to maintain the equilibrium against the external atmospheric pressure, and the latter therefore forces the yielding parts inward. This symptom, which is also observed in tonsillar hypertrophy of very small children (page 193), and may produce a similar deformity of the thorax, must be much more marked in rachitic children, in whom the thorax is abnormally softened and the inspiratory muscles often weaker than normal. In time this will lead to permanent depression of the lateral portions of the chest, with corresponding protrusion of the sternum and its cartilaginous appendages. The girdle-like notch which runs around the anterior surface of the thorax, about three finger-breadths below the nipples, and below which the ribs lying over the liver, stomach, and spleen appear bent outward and pushed forward, must be partially attributed to the downward pull of the diaphragm, partly also to the atmospheric pressure.[1]

I have previously stated that the increase in the size of the head, especially the prominence of the forehead and the parietal protuberances, is due to periostitic deposits, and may occasionally be mistaken for hydrocephalus. Various authors describe cases of "hypertrophy of the brain" in rachitis, but I have never met with such a case, nor could I convince myself of the frequency of hydrocephalic complications, as is stated, for example, by Ritter.

The etiological relations are as obscure as those of scrofula. The spread of the disease in large cities, especially in Northern and Middle Europe, is enormous, and my own experience corroborates that of Ritter, who found thirty-one per cent. rachitic individuals among the children treated by him in dispensary practice in Prague. The predominance of the disease among the children of the poor shows that unfavorable vital conditions, improper nourishment, imperfect care and uncleanliness, breathing bad air in narrow, overcrowded, and often damp rooms, play a decisive part in the production of rachitis. The majority of rachitic children are bottle-fed, and suffer greatly from dyspepsia and diarrhœa, in consequence of over-feeding. Imperfect quality of the mother's milk, usually due to their poor circumstances, may also act as the cause of rachitis. But we often find rachitic children among the higher classes, though they have apparently had the best breast-milk. Although I will admit that Ritter's assumption of a hereditary predisposition, especially from the maternal side, is justified in many cases of this kind, I cannot, according to my own experience, attribute the influence to heredity in general, which is maintained by this author. Nor do we possess any information with regard to the influence of other diseases of the parents, or the children themselves. It has appeared to me, however, that hereditary syphilis, after recovery, has been repeatedly followed by rachitis. As a matter of course, the swelling of the epiphyses and the other osseous

[1] Ritter, l. c., S. 170, and Rehn, l. c., S. 66.

changes (page 43) due to syphilis must not be mistaken for rachitic changes.

Does fœtal rachitis occur? Distinguished authorities (Virchow, Ritter, and others) acknowledge its occurrence, though such cases are exceedingly rare.[1] I have seen two children who were born with multiple curvatures and incomplete fractures of the bones, but they unfortunately passed out of further observation. In both cases the cranium was soft and compressible, and there was a peculiar sclerotic hardness of the muscles of the arms and thighs, such as I had never observed. Much more frequent than the fœtal form is that variety which develops very early, and is therefore called congenital. In these cases the characteristic swelling of the epiphyses of the ribs and the insufficient ossification of the cranial bones are observed, while at a later period—for example, in the beginning of the second year, the head may be entirely free, and only the thorax, limbs, and spinal column present rachitic symptoms. The disease does not appear to develop later than the beginning of the second year; in almost all cases which come under treatment at a later period, it has existed for a much longer time, but was not recognized by the parents until it was found that the children could not stand and walk at the usual period.

I cannot pass over in silence the changes in the cranial bones described by Elsässer[2] under the term "craniotabes." The cranial bones, especially the occipital, may be readily sawed through, or even cut. The bones are soft, succulent, congested, flexible, and rough and porous in many places; the earthy constituents are diminished in amount. The spongy character of the tissue is most marked near the fontanelles and sutures, while the edges are more compact. The periosteum is thick, congested, and can be removed with difficulty. In the occipital bone, but also in the parietal bones, especially along the lambdoid suture, are found impressions and grooves, which are said to correspond to the convolutions of the brain, and are very thin and can be indented like a card, but, occasionally, after the entire disappearance of the osseous substance, constitute irregular, oval, or angular holes, which are even as large as a hazel-nut, and in which the pericranium and dura mater come in contact. These symptoms are occasionally found during the first few months, but usually toward the age of eight months, or, at the most, of thirteen months. Elsässer, who regards craniotabes as rachitis of infancy, assumes that the rachitic softened parietal and occipital bones are gradually absorbed, thinned, and finally perforated by the pressure of the cerebral convolutions on the places of pressure, especially on the occiput, on account of the constant dorsal decubitus. In fact, in many children, during the first year, palpation will reveal yielding, compressible, creaking spots in the occipital bone near the lambdoid suture. But in a part of these cases I found that the thinned spots consolidated with the general progress of the retarded ossification of the skull, without being associated with any other rachitic symptoms. We must therefore agree with Friedleben and Ritter, that these craniotabes may occur within the limits of physiological development. But it cannot be denied that in the large majority of such cases, other rachitic symptoms are also present in the osseous system or develop at a later period.

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[1] Compare Winkler: Arch. f. Gynäkol., Bd. II., S. 101; and Fischer: ibid., Bd. VII., Heft I.

[2] Der weiche Hinterkopf, u. s. w. Stuttgart and Tübingen, 1843.

I will now say a few words with regard to the pathogenesis of rachitis. Examination of the blood showed no noteworthy changes, as a slight diminution of the red globules, or more frequently an increase in the white ones, can only be regarded as an accidental complication. The results of urinary examinations are so divergent that we do not know in what author to put our trust. While earlier observers noticed more or less increase of the earthy phosphates in rachitic urine, almost all recent authors deny the increase in the proportion of lime. Indeed, one of the most recent authors, Seemann,[1] found considerable diminution of lime when compared with the urine of healthy children. The agreement of almost all recent analyses with regard to this point renders it undoubted, and furnishes proof that the insufficient amount of lime in rachitic bones is not, as was formerly supposed, due to the presence of an acid (lactic acid) which dissolves the lime salts, in which event the excretion of lime in the urine would be increased, but to a diminished supply of lime. But, as mother's milk (and the cow's milk to an even greater extent) contains sufficient quantities of lime, the cause of the insufficiency of lime in the bones cannot be caused by a deficiency in the supply of lime contained in the nourishment; we have to deal with an insufficient absorption of lime salts by the digestive organs, and this view is also supported by the fact that the fæces of these children, according to the investigations of Petersen[2] and Baginski,[3] appear to contain more lime than those of healthy children of similar age.

Thus far we stand upon a quite firm foundation, but everything beyond this is hypothetical. The question whether the lime salts are not absorbed and assimilated in sufficient quantities by the digestive organs still needs solution, and this is also true of Seemann's theory, that there is an insufficient formation of hydrochloric acid by the stomach, on account of which the ingested lime salts are not dissolved and absorbed in sufficient quantity. Whether the insufficient supply of lime to the bones is in itself capable of producing the cartilage proliferation characteristic of rachitis (page 330), as Roloff and Seemann maintain, or whether the latter is produced by a constitutional irritant acting upon the osteogenous tissue, as Wegner[4] assumes from his experiments, is also an open question.

Under these circumstances we are unable to furnish a scientific basis for treatment, and must be satisfied that we almost always prove successful with empirical remedies. As a matter of course, it is justifiable that we recommend proper diet and other hygienic measures in prophylaxis as well as in treatment, and it is to be deplored that these can be employed alone in the comparatively rare cases in which the disease occurs in the children of well-to-do people. Nutritious, easily digestible food (milk, bouillon, yolk of egg, wine, later meat), country air, dry, sunny rooms, and careful attention to the skin by means of lukewarm baths—all these important factors often remain merely pious wishes. It appears to me absurd to forbid the use of milk, as this is founded on some unreliable experiments concerning the injurious influence of lactic acid. In the large majority of cases of rachitis recovery ensues despite untoward external circumstances, unless tuberculosis, or some other complication, occurs, or the general cachexia has reached too high a grade. As a rule,

[1] Zur Pathogenese und Aetiologie der Rachitis. Virch. Arch., Bd. 67. 1879.
[2] Rehn, l. c., S. 91.
[3] Ueber den Stoffwechsel in der Rachitis. Veröffentlichungen aus der Gesellschaft für Heilkunde. Pädiatrische Section.
[4] Virchow's Arch., Bd. 55.

I begin treatment with the milder preparations of iron, especially tinct. ferri chlorati, eight to ten drops three times a day; if this, as I have sometimes observed, produces diarrhœa, I substitute ferrum lacticum or reductum (0.03–0.05 twice a day). As a matter of course, the digestive organs must be in good condition in order to tolerate the iron. If anorexia, coated tongue, constipation, or diarrhœa are present, we should first give hydrochloric acid, then mild bitters, especially tinct. rhei laquosa or vinosa (10–12 drops three times a day), and the latter may also be combined with the iron. On alternate days a lukewarm bath should be given, with the addition of salt (page 325), or infusion of malt or aromatic herbs (about a handful of chamomile or calamus), and the lower limbs should be rubbed and kneaded with flannel in the bath, as well as several times during the day. We should persistently wash the occiput in order to relieve the profuse perspiration, and if there are softened spots in the occipital bone, the head should be rested on a hair-pillow, with a hole in it, in order to protect the part in question from pressure. In order to prevent curvature of the spine and incomplete fracture of the lower limbs as much as possible, little rachitic children should not be allowed to sit upright without support, but should lie for the most part on a hard mattress, and make attempts at standing and walking with great caution.

In a very large number of cases this treatment proved successful within a few months; not infrequently I have even seen the children make attempts at standing and walking within a few weeks. In another series of cases, in which recovery made no progress under such treatment, I satisfied myself of the good effects of cod-liver oil. I only give it in cool weather, chiefly in emaciated children, and never more than two tablespoonfuls daily, either alone or in combination with preparations of iron. I have never seen any good results from the administration of lime salts, and have long since discontinued their use.

The rachitic deformities of the limbs which are produced by curvature and fracture of the bones require no special treatment in milder cases, as they gradually disappear after recovery, with the increase in the development of the body. But probably no orthopœdic treatment could relieve deformities due to consolidation of fractures; improvement can only be expected from such treatment if the bones are still soft and flexible. Hauke's "pneumatic chamber," in the rarefied air of which the children are placed for some time every day, has recently been recommended to diminish or entirely remove the rachitic deformity of the chest.[1] The treatment of consolidated curvatures of the limbs belongs to the domain of surgery.

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[1] Kaulich: Prager med. Wochenschr., No. 2. 1880.

LEEDS & WEST RIDING
MEDICO-CHIRURGICAL SOCIETY

PART X.

DISEASES OF THE SKIN.

Almost all diseases which affect the skin of adults also occur during childhood, but as these are almost entirely similar to those of later life, I will confine myself exclusively to the consideration of those affections which occur most frequently in children, or are characterized by certain peculiarities. But I cannot omit touching upon a question which has always interested physicians on account of its practical significance: I refer to metastases of skin diseases. Under this term the older physicians meant the rapid disappearance of a skin affection, followed by the development of some internal or external disease. In recent times the occurrence of such metastases is denied; Hebra especially takes a decided stand against it, and fears no bad results to the general organism from the cure of the skin affection. Indeed many facts bearing on this point were previously falsely interpreted; for example, it was not infrequently overlooked that matters were directly the reverse, that the disease of the skin disappeared because an internal affection was developing. Thus, chronic eruptions on the scalp not infrequently become dry when meningitis develops, in the same manner that, at such a time, the nasal mucous membrane may become dry, an otorrhœa ceases, and enlargement of the glands rapidly resolves. On the other hand, critical observation showed that inflammatory irritation may occasionally spread from suppurating scalp eruptions by means of phlebitis or thrombosis of the small veins of the skin and bones into the interior of the skull, and may here give rise to dangerous symptoms. Nevertheless, I consider the question of metastasis as by no means settled. In the first place, I do not think that hospital observations can claim the same weight that belongs to them in other cases, because the little patients, after recovery of the eruption, are usually discharged at once, and the further course in the majority remains entirely unknown to the physician. I therefore regard private practice as by far more adapted to the solution of this question, and since I have carefully and without bias observed a few cases in which the artificial suppression of a chronic scalp eruption was almost immediately followed by intense exudative pleurisy, bronchitis, or diarrhœa, and striking improvement occurred at once after the spontaneous reappearance of the eruption,[1] I have become somewhat more cautious in absolutely denying the occurrence of metastases. In addition, I afterward saw two young

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[1] Berl. klin. Wochenschr., No. 5. 1864.

children, in whom convulsions, with a fatal termination, occurred eight to ten days after the rapid recovery of eczema of the scalp and face. I am very well aware that these isolated observations are by no means decisive, and that they may be merely the result of a coincidence; nevertheless, they made a deep impression upon me and again aroused the suspicion, which had long disappeared, whether the sudden cessation of a long-continued, extensive purulent or sero-purulent discharge, may not cause hyperæmia and its consequences in other parts. I think that this possibility should not be left unconsidered in the treatment of such exanthems, and I will recur to it in discussing the treatment of eczema.

I. Erythema and Intertrigo.

Erythema is one of the most frequent skin diseases of childhood, especially during the first year. It is characterized by a larger or smaller number of red patches of varying size and form, which appear on various parts of the body, and also on the face: the round, small ones, as large as a lentil or pea, are called roseola by many; the irregularly formed larger ones, erythema. Occasionally the hyperæmia is combined with slight exudation, so that the reddened skin, either in its totality, in parts or at the edges, is infiltrated and appears somewhat elevated. There are thus numerous varieties, known as erythema nodosum, papulosum, marginatum, annulare, which are identical with the forms occurring in adults, and are occasionally associated with small extravasations of blood or urticaria-like wheals. The outbreak of the eruption occurs at times, though not always, with febrile symptoms (general malaise, anorexia, acceleration of the pulse, and elevation of temperature), which cease, as a rule, when the eruption is complete. The latter continues for several days, then gradually grows pale, and finally disappears without a trace or with slight desquamation. These eruptions, which occasionally itch very much, occur frequently in children, especially in the spring, from March to May. I could rarely satisfy myself with regard to the cause; in a few cases—for example, in a child ten months old, which had been weaned for two weeks, erythema papulatum and nodosum developed from cholera infantum due to a dietetic error. As I have previously remarked, the eruption may be mistaken for measles, and especially for scarlatina, if it appears as a diffuse erythema. The moderate degree or entire absence of fever, the absence of characteristic catarrhal or anginal disturbances and its rapid course without subsequent lamellar desquamation, are sufficient to distinguish it from scarlatina, although very mild cases of the latter disease occasionally occur, in which the diagnosis is by no means easy and cannot be made positively until after the occurrence of desquamation. Many cases of repeated development of measles or scarlatina in one and the same child are probably due to such a mistake. Roseola and erythema are not contagious, but they may assume a certain epidemic extent. Treatment is rarely necessary; if there are febrile prodromata, keep the child in bed and give a mild purgative.

Apart from the eruptions of this class which occur in general febrile diseases, such as rheumatism, typhoid fever, pyæmia, and diphtheria in children, as well as in adults, we not infrequently find erythema develop around excoriated or ulcerated portions of skin—for example, around vaccine-pustules, in which the entire arm may become reddened and swollen, or, as I have often observed, around eczematous and impetiginous

portions of skin; the erythema is then distinguished from the erysipelas occurring under similar circumstances, by its more spotted appearance, the absence of fever and of a tendency to spread. Simple fomentations with lead-wash are then almost always sufficient to relieve the erythema.

Entirely distinct from these eruptions are those inflammatory conditions of the skin which are caused by direct irritation of the latter (pressure, chemical irritants), and are described under the term intertrigo. In very many children, during the age of infancy, who are not properly nursed, we notice around the anus, on the genitalia, and the inner surfaces of the thighs, more or less extensive bright or dark red erythemata as a result of contact with the urine and fæces. They are also often found on the heels and the posterior surfaces of the thighs and legs, which are almost always in contact with the wet diapers during dorsal decubitus, or in places where folds of skin are in contact with one another, as in the inguinal regions, the neck, upper part of the chest, the axillæ, neck, and behind the ears. Many children, even those well cared for, present a decided tendency to intertrigo, which may then, especially if proper cleanliness is not preserved, extend over large portions of the skin—for example, over the entire lower half of the body, occasionally over the back, abdomen, or even over the entire body. Here and there we may also find dark red papules on the reddened skin, and the latter often assumes a moist, shining, and sticky appearance, inasmuch as the erythema is intensified into dermatitis, and the epidermis is softened and macerated by serous exudation, so that a large portion of the body is dark red and shining, as if flayed. This also occurs not infrequently in the intertrigo, which is confined to the above-mentioned folds of skin, and, after the epidermis is exfoliated, yellowish gray, irregular, more or less deep ulcerations not infrequently occur in the midst of the reddened skin, and when situated around the anus and genitalia, may lead to the erroneous assumption of a syphilitic origin. The inexperienced observer is liable to the same error in those cases in which the intertrigo around the anus and on the nates is mixed with large papules, whose blunt summits, deprived of epidermis, appear like red or yellowish red excoriations, and, in fact, may present a certain degree of similarity with ulcerated, broad condylomata. All these forms of intertrigo occur occasionally in the children of well-to-do parents, but in a much larger number of cases among the atrophic, neglected children of the poor. Lack of cleanliness, living in overcrowded, damp rooms, insufficient or improper nourishment, cold, etc., combine to produce a cachexia which is very appropriately termed cachexia pauperum. In such cases we most frequently find the intertrigo spread over the greater part of the body. The epidermis may be removed by serous exudation beneath it, and then forms mere shreds upon the red, moist, shining cutis, as in extensive burns, while in other cases the red but dry skin appears covered with numerous desquamated, grayish yellow lamellæ, consisting of epidermis and sebum. Like extensive burns, these cases may prove fatal from complications with pneumonia and diarrhœa.

The treatment of intertrigo, above all, requires the greatest cleanliness, washing the genitalia and neighborhood of the anus after every evacuation of urine and fæces, frequent application to the reddened parts of a powder consisting of equal parts of white oxide of zinc and starch, and separation of the reddened folds of skin, which are in contact with one another, by the interposition of charpie or linen smeared with zinc or lead ointment. Instead of ordinary fat I employ vaseline in these ointments,

and this may also be used without any addition. Warm baths may readily prove too irritating; the bath should therefore, at the most, have a temperature of 26°, and receive an addition of bran or bolus alba (50–100.0). In very extensive forms of intertrigo, baths of corrosive sublimate (1.0) occasionally furnished good results, even when syphilis was not present, while, in obstinate intertrigo confined to the anus and genitalia, daily applications of a solution of nitrate of silver (1 : 50) did good service.

II. Lichen-Strophulus and Prurigo.

A. *Lichen-Strophulus.*—This extremely frequent affection of childhood is characterized by numerous bright or dark red nodules, in part perforated by a hair, which are either discrete or collected in groups upon a red, occasionally infiltrated base; at times they are so small as to be better appreciated by touch than by sight; at times they are larger, and even attain half the diameter of a pea or more. Usually, though not always, it is associated with violent itching, by which the papules are readily rendered bloody. If the eruption is scanty, the general condition is not appreciably disturbed, while, if it is more profuse or perhaps extends over the larger part of the body, slight febrile disturbance, insomnia due to the pruritus, and great restlessness, may develop. A part of the papules gradually grow pale and are absorbed; others show a small vesicle or point of pus upon the summit, which becomes dry and finally leaves small scales on the diminishing papule. Exacerbations frequently occur, and the eruption may then last several weeks or even months, until recovery finally occurs.

These papular eruptions are observed most frequently during the period of first dentition, and are therefore regarded by many as the results of dental irritation, *i.e.*, a reflex angioneurosis, starting from the dental nerves. Among the local irritants which may produce this skin disease, I will mention the direct rays of the sun and heat, which, in addition to a vesicular eruption (eczema solare), not infrequently causes a large number of extremely small red papules on the neck, back, chest, and face. In very many cases the cause of these eruptions remains entirely unknown; no relation to morbid conditions of internal organs can be shown, and the unproven assumption of a dyscrasic basis does not aid us. The fact that these eruptions are more common in general among the poor than in the better classes, indicates that unfavorable hygienic conditions, especially imperfect care of the skin, are not without influence.

The treatment of lichen-strophulus, when there is violent pruritus, is confined to lukewarm baths, with an addition of bran or soap. Washes of a solution (1–2 per cent.) of carbolic acid twice a day may be recommended for the relief of the irritation. Internal remedies are useless, and, in addition, the disease usually disappears spontaneously within a certain period.

B. *Prurigo.*—I have very often observed this disease during the first years of life, and the symptoms do not differ appreciably from those observed in adults. In children also we find prurigo papules, which are partly pale, partly covered with a spot of blood, caused by scratching. They are situated especially on the extensor aspect of the extremities, while the flexor aspect is free or very slightly affected; it also appears on the abdomen, back, and chest. The constant violent itching causes the children to scratch, and to this mechanical injury we must attribute the

further changes in the skin, which gradually develop during the course of prurigo, viz., the eczematous eruptions, the roughness and thickening of the skin, changes which may sensibly modify the originally simple papular appearances. The unusually marked enlargement of the lymphatic glands in the inguinal region and over the adductors of the thighs, referred to by Hebra, I have scarcely ever missed in children. The general condition is undisturbed, but the interference with sleep may finally impair the healthy appearance of the children, and this so much the more as prurigo is a very chronic disorder, even in children, and may last for years with slight intermissions.

In a few children suffering from prurigo, I observed, either at the same time or as prodromata of this affection, an eruption of small pemphigus vesicles which preceded the prurigo, in one case, in quite considerable numbers, in the others developed scantily from time to time between the papules.

The etiology was obscure in all cases. Neither a hereditary predisposition nor a tubercular constitution, to which Hebra attaches importance, could be determined with certainty. I was as unsuccessful as others in the treatment; recovery, lasting several years, was obtained in but one case, while the sister of this patient, who was also affected with prurigo, suffered repeatedly from relapses. Daily frictions of the body with sapo viridis in a lukewarm bath, and later a similar use of Vleminx's solution of sulphide of lime (P. 47), appeared to have been curative in this case, while the same remedies in other cases were as useless as the internal administration of Fowler's solution.[1]

Finally, I must call your attention to the fact that scabies of somewhat long standing may be mistaken for prurigo on superficial examination, if distinct furrows are not forthwith detected. You should always bear this possibility in mind, and not only in practice among the poor. I have repeatedly observed scabies, in children belonging to good families, during the first year of life and under circumstances which appeared to exclude every possibility of infection.

III. Eczema and Impetigo.

Among all affections of the skin in childhood, the vesicular and pustular forms assume the first rank in point of frequency. By a purulent metamorphosis of the contents of the vesicles, eczema is converted into impetigo, the pustules of which grow larger, and, after their rupture, usually form thick, honey-yellow scabs by desiccation. We very often find vesicles, pustules and their remains mingled with one another upon the same part of the integument (eczema impetiginosum).

During the period of infancy, and not infrequently within a few weeks after birth, the eruption occurs very often, especially on the face. In its exquisite forms we find the forehead, cheeks, nose, upper lip, and chin covered, like a mask, with greenish or blackish brown crusts, which are either more or less connected or separated here and there by interspaces of red excoriated integument. In some places the crusts have been scratched off, and the blood trickling from the excoriated skin has coagulated into a dark scurf. On more careful examination we will oc-

[1] I have hitherto had no personal experience with the injections of pilocarpine recommended by O. Simon.

casionally discover small vesicles and pustules, situated either singly or in groups upon a red surface, the dried secretion of which constitutes the crusts; these are found either around the crusts or on intact portions of the skin. Apart from the annoying pruritus, the majority of the children are perfectly well, and even present a blooming appearance; but the neighboring lymphatic glands under the angle of the jaw and the chin are apt to enlarge through the medium of the lymphatics. The duration of the eruption is very variable. As a rule it lasts, with alternate intervals of improvement and relapse, at least four to six weeks, but often months, or even years. During an acute disease—for example, pneumonia or profuse losses of secretion, especially in severe diarrhœa—the eruption not infrequently dries, but reappears after the diseases mentioned have recovered. Recovery finally occurs from disappearance of the eczematous eruption and of the serous secretion from the wounded surface, after which the overlying crusts become dry, fall off, and leave a red skin without any trace of cicatrices.

In a series of cases of this kind the eczema of the face spreads to the scalp, external ear, the interior of the concha, and the entrance to the nose, may also extend to the lower lid and then gives rise to inflammation of the palpebral conjunctiva.

The causation of the affection is obscure. The assumption of a scrofulous constitution is usually arbitrary; but it is undoubted that the disease is hereditary in many families, so that almost all the children suffer during the period of infancy from eczema of the face, even for generations. The affection is also ascribed to the nourishment, especially to the too fat milk of a nurse who is unsuited to the tender age of the child; but the proof, *i.e.*, recovery from change of nurses, can be obtained in exceptional cases alone.

The eczema often occurs after the period of infancy, but then usually affects the external ear, the concha, the region behind the ear, and especially the scalp, even more markedly than the face. Eczema capitis often forms extensive, continuous, quite moist, greenish brown or grayish green crusts, which cover the entire scalp and cause matting of the hair; from the interstices of the crusts trickles a sero-purulent, often fœtid secretion which has accumulated between them. The crusts are very frequently full of pediculi. In other cases the affection is more limited and the scalp is only covered with the crusts in places; it either presents a coin-like or an entirely irregular form and a dry mortar-like consistence. We then often find loosened clumps in the hair, which have been lifted up by the growth of the latter, and look like pearls strung on a thread (tinea granulata). If these are gently removed by fomentations, the scalp appears red, excoriated and covered with secretion. The primary form consists of vesicles and pustules, which are visible around the crusts, especially when the disease, as often occurs, breaks out anew upon portions of the skin which had been previously healed. The children are led to scratch on account of the violent itching, and this maintains the inflammatory irritation. The neighboring glands behind the ears, on the back of the head, under the jaws, and in the neck, become swollen, and the secretion, which stagnates under the crusts and decomposes, gives rise in many cases to a nauseous smell. Not infrequently the inflammation of the skin spreads more deeply. In many cases I saw, in the midst of the eczematous parts, firm infiltrations of the skin, which finally became converted into abscesses; in a few cases a large collection of pus formed under the pericranium of the parietal bones, after the incision of which

the probe penetrated to the bones and the well-known ring of bone was felt at the edges, as in cephalhæmatoma (page 18).

The duration of eczema capitis is usually very protracted. Not infrequently it continues, with longer or shorter intervals, for years, even to the period of puberty, especially in poor children. The hairs are usually changed, become lustreless and thin, and may fall out, but grow again after recovery from the disease. As soon as this occurs, the secretion of the excoriated surfaces ceases, and small yellowish, dry scales form for some time upon the reddened skin.

The occurrence of eczema and impetigo upon the trunk and extremities is observed not infrequently in children who also suffer from eczema of the face and scalp. But the face and scalp may escape entirely, especially in older children at the period of second dentition, who then present the same appearances as in adults. Occasionally the eruption has lasted from earliest childhood—for example, in a girl aged six, who had suffered uninterruptedly since the age of seven months, from eczema extending over the larger part of the body. In these chronic cases I found that the flexor aspect of the elbows and knees, and the inner surfaces of the thighs and the calves, were more especially affected.

As a rule, the course of these eruptions is chronic; but acute affections of this kind, lasting only a few weeks, occur not infrequently in otherwise healthy children. I have observed them repeatedly on the arm and in the corresponding axilla, or on the lower limbs and face, especially the chin, without any other morbid symptoms. A boy, aged fourteen, suffered every spring for the past ten years from an eruption of eczema upon the cheeks and ears, which lasted about four to six weeks, and then disappeared entirely. In a few other cases an acute exacerbation suddenly occurred during the course of a chronic eczema of the face, the eyelids and entire face became considerably swollen, and the newly developed vesicles in the face became converted into large pustules, which occasionally presented central umbilication, like variola pustules, and coalesced with one another. In one of these cases the diagnosis of variola had been made, but this was negatived by the complete integrity of all other parts of the body (except a few pustules which appeared on both wrists), the entire absence of fever, the general euphoria, and the rapid termination of the disease within a few days. These acute exacerbations of chronic eczema are usually produced by violent scratching on the part of the children, giving rise to hemorrhages.

I must dwell a little longer on the hemorrhages occurring in eczema of the face, because they are not always due to traumatic causes, and are therefore of a mild character; I saw exhausting fatal hemorrhages occur in three cases in children three to four months old. They appeared to be due to a hemorrhagic diathesis, which was manifested in one of the children by slight hemorrhages from the stomach and intestinal canal, while no other hemorrhage occurred in the two remaining cases. One of the latter children appeared to be perfectly healthy, while the other suffered from rachitis and spasm of the glottis, and presented a very miserable and anæmic appearance. In all these cases the blood flowed almost constantly from the eczematous surface and fissures, although these had not been scratched, and coagulated into soft, black crusts, which were soon swept away by the subsequent flow of blood. All styptics, including ergotin, proved useless, and the children died in a few weeks from increasing weakness, with symptoms of collapse. From this experience, I would always regard spontaneous hemorrhages from eczema of the

face, which are repeated without any definite cause, as a not unsuspicious symptom.

Concerning the etiology of eczema in general, we know very little, and in comparatively rare cases can we ascertain the cause with certainty. Among these are included eczema solare (sudorale, æstivum), which occurs during summer in very many children, even in nurslings, upon the back, chest, neck, but especially upon the forehead and temples, in the form of closely grouped, extremely small vesicles and nodules on a reddened surface. Larger papules and even pustules are occasionally found, and in a girl, aged three, I also observed marked erythema and œdema of the left eyelid and half of the forehead. Traumatic influences are etiologically important in other instances.

A child, aged two months. Contusion and abrasion of the forehead followed extraction with the forceps, and lukewarm fomentations were applied. Two weeks later eczema developed on the injured parts and extended to the scalp, attended with marked œdema of the eyelids. In a number of small children piercing the ears gave rise to eczema, which was either confined to the external ear or spread to the neck and back. Other eruptions, especially psoriasis, may also occur in a similar manner.

You will very often find that vaccination is regarded as the cause of eczema. According to the statement of the mother, the eruption appears in the face or other parts of the body immediately or soon after vaccination. Although I believe that many of these cases are due to coincidences, I will so much less deny the possibility of such a connection, because other acute exanthemata, especially measles, but also scarlatina, varicella, and small-pox, are often followed by eczema and impetigo. Its relations to dentition are also uncertain, and the frequent assumption of a scrofulous constitution is devoid of all foundation in many cases; it is only justified when other scrofulous symptoms are present. The accompanying secondary enlargement of the neighboring glands is not, in itself, decisive.

It has been recently held that eczema impetiginosum may become contagious. I have observed a few cases occurring in the children of one family, one of which is especially noteworthy: a little child, suffering from eczema of the face and scalp, conveyed the disease, after a few weeks, to an elder sister, who was accustomed to carry the child around, holding her head against the cheek of the latter. I have been unable to discover the spores, described by Hebra and others, which are said to cause the infection.

The question arises whether the disease should be rapidly cured. In view of my previously mentioned experience (page 336), I have made it a rule for years not to cure at once chronic eczema which has lasted for many months or even years, especially on the face and scalp. I apply local treatment to one part of the diseased skin after another—a plan to which we are forced, in many cases, by the great extent of the affection. The crusts should be first removed by inunctions with vaseline or fresh oil, or fomentations of lukewarm water (the latter being preferable on the scalp), which are covered with a cap of oiled silk or gutta percha paper. After the removal of the crusts, the red and moist skin is washed once a day with soap-water (sapo viridis), and then bandaged with unguent. Hebræ, which should be applied for twelve hours. When the inflammation is severe I first order fomentations of lead-wash. The most difficult part of the procedure is to fasten the bandage to the face in little children, and prevent scratching. The former is best effected by the ap-

plication of a linen mask, upon the inner surface of which the ointment is smeared, and the latter by enveloping the hands and fingers with cotton and linen. Instead of Hebra's lead-ointment I also used successfully ointments of salicylic acid (P. 48), tannin (P. 49), or zinc; more rarely, and only in eczema of small extent, an ointment of hydrarg. precip. alb. or rubr. (0.5 : 15.0 vaseline). It is not advisable to use tar ointments from the beginning, as they may prove too irritating and increase the inflammation; on the other hand, they should be recommended after previous treatment with the ointments mentioned above, in order to make recovery more lasting. I generally employed oil of cade, which is applied daily as a liniment (1 part to 2 or 3 parts olive-oil), after previously soaping the diseased parts. But, in applying tar ointments to extensive surfaces, you should always bear in mind the possibility of an irritant action on the kidneys (page 251), and therefore carefully examine the urine, the blackish color of which or the presence of albumen at once requires a discontinuance of the treatment.

The duration of the treatment naturally varies greatly. While many eczemas, even those which have lasted for a long time, recover in a few weeks, others require treatment for months, and even then we often find that relapses occur without any ascertainable cause. In these very obstinate cases, I obtained good results occasionally from the use of arsenic in the form of Fowler's solution (P. 11). Even children two or three years old tolerated the remedy excellently in small doses (3 to 5 gtt. of the mixture t. i. d., given on a full stomach). In the scrofulous diathesis I also had good effects from the administration of syrup ferri iodati, or a mixture of iodine and iodide of potash (P. 46). I cannot recommend salt-baths, as they not infrequently render the eruption worse by irritating the skin too severely. I would rather advise lukewarm (26° R.), soap, or sulphur baths; the latter may be prepared by the addition of 50–100.0 kali sulphurat. to the bath.

IV. Ecthyma.

Large pustules, surrounded by a red border, are often observed in children, either singly or in groups, and may be associated with eczema or separate. They are located usually on the nates, the thighs and calves, may attain the size of a pea, and dry into a blackish brown crust, after the exfoliation of which a red spot remains without a cicatrix. Ecthyma is often found in scrofulous children, but also in perfectly healthy ones who are not kept clean; it not infrequently appears to be produced by the irritation of vermin (especially body-lice), so that we should direct our attention to this cause in all cases.

Ecthyma also occurs often as the expression of a cachexia in miserable, poorly nourished children, who are exhausted by distress or disease (especially general tuberculosis, measles, scarlatina), and then often acquires a grave significance. As a rule, the foundation is constituted by the so-called cachexia pauperum, which is produced by the miserable conditions of life, and is manifested by profound anæmia, emaciation, weakness, and a tendency to chronic inflammations of various tissues. The skin not infrequently takes part in the shape of ecthyma or rupia cachectica, which may also be combined with one another. The difference between both varieties consists more in the size of the epidermic elevations than in their contents, as the flaccid rupia vesicles, which may

attain the size of half a mark or more, and appear especially on the trunk and back, may also be filled with opaque, pus-like contents, very similar to those of ecthyma pustules. Under these conditions deep ulcerations readily develop from the pustules and vesicles, which I have especially observed on the scrotum and its vicinity—but also on the back, as more or less numerous, round, sharply bordered losses of substance as large as a pea or groschen. With the improvement in the general condition these ulcers may gradually heal, leaving a corresponding cicatrix, but in the opposite event they become larger, and grow deeper and more numerous. The most serious event is the transition of ecthyma and rupia into gangrene, which occasionally occurs under these conditions.

J. B——, aged fifteen months; admitted March 11, 1879, very emaciated and anæmic; presented a number of ulcers of the skin which, according to the statement of the parents, had developed from pustules. They are situated almost exclusively around the genitalia, the scrotum, mons pubis, inguinal region, and thighs, a few on the nates. The ulcers are sharply defined, as large as a lentil or groschen, extending to the papillary bodies of the cutis, with a yellowish gray base and somewhat undermined edges. In a few days similar ulcers appeared on the right ear and coalesced, and the external ear was almost severed from the skull by a deep ulcerated fissure. March 21st, death in collapse. Autopsy.—Double broncho-pneumonia, cheesy degeneration of the bronchial glands, chronic intestinal catarrh.

C. P——, aged two and one-half years; admitted April 2, 1879, tolerably well nourished. A number of round ulcers on the right leg, with yellowish base, and red, sharply defined edges. These were said to have developed from pustules two weeks previously; similar recent pustules are observed here and there on the body. Severe coryza, bilateral otorrhœa, eczema of the ear, enlargement of the glands. April 6th, fresh eruption of pustules on left leg, the back and nates, which burst and soon ulcerated. From April 16th, the latter coalesce in part on the back, and form large ulcers, which have a blackish brown crust and a decidedly gangrenous smell. The entire back, abdomen, and the extremities in part are gradually destroyed by these gangrenous, deep ulcerations. Increasing emaciation and debility, irregular fever, which reached 40.8° on April 27th and 28th, cough and diarrhœa. May 5th, death. Autopsy.—Chronic fibrinous pleurisy, bilateral broncho-pneumonia, circumscribed pulmonary gangrene, tuberculosis of the right lung, peritoneum and internal genitalia (tubercular salpingitis, perisalpingitis, and perioöphoritis).

In this case the widespread tuberculosis gave rise to the cachexia, which was followed by ecthyma cachecticum and the consequent multiple gangrene of the skin. The pulmonary gangrene found on autopsy must undoubtedly be regarded as embolic (page 169). The treatment of ordinary non-cachectic ecthyma is in general the same as that of eczema. We should never forget to look for vermin, and to remove these whenever found.

V. Abscesses of the Subcutaneous Tissue.

The tendency to abscesses of the connective tissue is especially marked during the first few years of childhood. I do not now refer to the isolated phlegmons, confined to one spot, which are either due to traumatic influences, or to the irritation of inflammations of the adjacent integument (eczema impetiginodes), or to hyperplasia of the lymphatic glands, especially under the angle of the jaw; but to the multiple abscesses, which develop simultaneously or successively in various parts of the body without any known cause, and must therefore be regarded as the expression of a diathesis. We do not know upon what this diathesis depends. There is no doubt, however, that although the infiltrations and abscesses in

question occur now and then in healthy children, they usually affect those individuals who are markedly atrophic or tuberculous. The younger the children, the more frequently do we meet with the abscesses. Even in the first few months of life we find multiple infiltrations in various parts of the body, from the size of a pea to that of a walnut or a hen's egg; in a few days they become red, fluctuate, burst, and leave bluish pigmented cicatrices after recovery. The continually repeated suppuration tends to increase the atrophy and weakness; the abscesses are occasionally converted into deep ulcerations, which, as I have several times observed, lay the muscles bare, and may result in extensive necrosis of the skin and connective tissue.

Another form of abscess is often found in scrofulous children or those suffering from affections of the osseous system. Around the ankles, on the dorsal surfaces of the hands and feet, over the ribs, on the head, etc., are often found smaller or larger abscesses covered with integument of normal color, which may continue many weeks before they become red, and after opening them the sound comes in contact with carious bone. In a few instances I have observed colossal abscesses on the head, in which the pus had accumulated between the bones and pericranium, and finally perforated the latter and the external skin. In these cases, as in cephalhæmatoma (page 18), we could feel the wall-like ring of bone surrounding the abscess; this was due to periosteal deposit at the border of the abscess, where the bone and pericranium are in contact. We should not mistake a prominent suture for such a ring of bone, as happened to me in one case. The region behind the ear is also often the site of extensive abscesses, which separate the concha from the head, so that the former is directed forward. If the opening of the abscess is delayed, it readily opens into the external auditory meatus, and, after a deep incision has been made, the probe often shows that we have to deal with caries of the petrous portion of the temporal bone, or the mastoid process.

You should examine with special care all abscesses forming on any part of the back, nates, in the inguinal region and the inner surface of the thighs, because these are often cold abscesses, due to vertebral caries.

FORMULARY.

The numbers of the prescriptions (P. 1, etc.) correspond to the similar ones occurring in the text.

P. 1.
Hydrargyr. oxydul. nigr. 0.01
Sacch. alb. 0.5
M.—Give this powder twice a day.

P. 2.
Calomel ..... 0.005—0.01
Sacch. alb. 0.5
M.—Give this powder twice a day.

P. 3.
Acid. hydrochlorat. ..... 0.5—1.0
Aq. destil. 100.0
Gm. arab. 1.0
Syrup. alth. 20.0
(Tinct. opii., gtt. 2—4.)
M.—Give one teaspoonful every two hours.

P. 4.
Creosoti ..... gtt. 2—4
Aq. destil. 35.0
Syrup. alth. 15.0
M.—Give one teaspoonful every two hours.

P. 5.
Pepsini 1.0
Acid. hydrochlor. 0.5
Aq. destil. 120.0
Sacch. alb. 10.0
M.—Give one dessertspoonful four times a day.

P. 6.
Pulv. rad. ipecac. ..... 1.0 —2.0
Tartar. stibiat. ..... 0.03—0.05
Aq. destil. 30.0
Oxymel scillit. 15.0
M.—Give one dessertspoonful every ten minutes until the effect is produced.

Pulv. rad. ipecac. ..... 0.5 —1.0
Tartar. emetic. 0.01
M.—Give this powder every ten minutes until the effect is produced.

P. 7. Calomel ..... 0.03—0.05
Sacch. alb 0.5
M.—Give this powder every two hours.

Inf. sennæ comp.,
Syrup. spinæ cervin ..... āā 25.0
M.—Give a dessertspoonful every two hours.

P. 8. Kali bromati 3.0
Aq. destil 100.0
Syrup. simpl 20.0
M.—Give one dessertspoonful every two hours.

P. 9. Hydrat. chlorali ..... 1.0—2.0
Aq. destil 100.0
Syrup. cort. aur 20.0
M.—Give one dessertspoonful every two hours.

Hydrat. chlorali ..... 0.3—0.5
Aq. destil 50.0
M.—To be given by enema.

P. 10. Morphii acet. s. muriat ..... 0.01—0.03
Aq. destil 35.0
Syrup. alth 15.0
M.—Give one teaspoonful two or three times a day.

P. 11. Solut. arsen. Fowl 2.0
Aq. destil 8.0
M.—Give ten to fifteen drops three times a day.

P. 12. Ferri lactici s. reducti ..... 0.03—0.05
Sacch. alb 0.5
M.—Give one powder two or three times a day.

Tinct. ferri chlorati s. pomati 7.5
Tinct. rhei vinosa 2.5
M.—Give twelve to twenty drops three times a day.

Tinct. ferri chlorati 10.0
Give eight to twelve drops three times a day.

P. 13. Kali hydroiodici ..... 1.0—2.0
Aq. destil 100.0
Aq. menth. piper 20.0
M.—Give one dessertspoonful three to four times a day.

P. 14. Camphoræ tritæ ..... 0.05—0.1
Sacch. alb 0.5
M.—Give a powder every two hours.

Camphoræ 0.6
Spirit. vini,
Aq. destil..... āā 5.0
M.—Inject a hypodermic syringeful.

P. 15. Ammon. muriat..... 1.0—2.0
Aq. destil..... 100.0
Tart. emet..... 0.05
Syrup. liquirit..... 20.0
M.—Give one dessertspoonful every two hours.

P. 16. Inf. rad. ipecac (0.2—0.5)..... 100.0
Natr. nitrici..... 2.0
Aq. laurocer..... 1.5
Syrup. alth..... 20.0
M.—Give a dessertspoonful every two hours.

P. 17. Calomel ..... 0.01—0.03
Pulv. rad. ipecac..... 0.01
Sacch. alb..... 0.5
M.—Give this powder every two hours.

P. 18. Tartar. stibiat..... 0.05—0.1
Aq. destil..... 100.0
Syrup. simpl..... 20.0
M.—Give a dessertspoonful every two hours.

P. 19. Vini stibiati,
Oxymel scillit.....āā 10.0
M.—Give a dessertspoonful every ten minutes until the effect is produced.

P. 20. Decoct. rad. senegæ s. polygalæ amaræ (5.0)..... 100.0
Liq. ammon. anisat..... 1.5
Syrup. alth..... 20.0
M.—Give a dessertspoonful every two hours.

P. 21. Camphoræ tritæ ..... 0.03—0.05
Acid. benzoic..... 0.05
Sacch. alb..... 0.5
M.—Give this powder every two hours.

P. 22. Infus. hb. digital. (0.3—0.5)..... 100.0
Natr. nitr. s. kali nitr. s. kali acetici..... 2.0—3.0
Syrup. simpl..... 20.0
M.—Give a dessertspoonful every two hours.

P. 23. Decoct. cort. chinæ (5.0—10.0) 100.0
Syrup. cort. aur..... 20.0
M.—Give a dessertspoonful every two hours.

P. 24. Extr. chinæ frigide par ..... 2.0—3.0
Aq. flor. aurant..... 100.0
Syr. flor. aurant..... 20 0
M.—Give a dessertspoonful four times a day.

P. 25. Calomel 0.015
Pulv. hb. digital..... 0.01
Sacch. alb..... 0.5
M.—Give this powder every two hours.

P. 26. Kali chlorici..... 3.0
Aq. destil..... 100.0
Syrup. simp..... 20.0
M.—Give a dessertspoonful every two hours.

P. 27. Decoct. cort. chinæ (5.0—10.0) 100.0
Kali chlorici 3.0 s. aq. chlori..... 15.0
Syrup. simpl..... 20.0
M.—Give a dessertspoonful every two hours.

P. 28. Electuar. e senna..... 25.0
Aq. destil..... 100.0
Acid. tartar..... 1.2
Sacch. alb..... 10.0
M.—One dessertspoonful, well shaken, to be given every two hours.

P. 29. Inf. rad. ipecac. (0.2)..... 100.0
Mucil. gm. arab.,
Syrup. simp.....āā 10.0
Tinct. opii, gtt. 2—4 s. extr. opii..... 0.02—0.03

P. 30. Magister. bismuthi..... 0.1—0.2
Pulv. gummosi..... 0.5
M.—Give this powder every two hours.

P. 31. Dec. rad. colombo (5.0—8.0)..... 100.0
Syrup. alth..... 20.0
Tinct. opii..... gtt.4
M.—Give a dessertspoonful every two hours.

P. 32. Dec. cort. cascarillæ (5.0—8.0)..... 100.0
Syrup. alth..... 20.0
Tinct. opii..... gtt.4
M.—Give a dessertspoonful every two hours.

P. 33. Acid. tannici,
Tinct. nuc. vomicæ.....āā 1.0
Aq. destil..... 100.0
Syrup. alth..... 20.0
M.—Give a dessertspoonful every two hours.

P. 34. Argent. nitrici ..... 0.05—0.1
Aq. destil. 100.0
Mucil. gm. arab 20.0
M. S.—To be kept in a black bottle. Give a dessertspoonful every two or three hours.

P. 35. Plumb acetici 0.015
Pulv. gummosi 0.5
M.—Give this powder three times a day.

P. 36. Ol. ricini 30.0
Gum arab. 1.0
Ft. emulsio.
Aq. destil. 75.0
Syr. emuls. 15.0
M.—Give a dessertspoonful every two hours.

P. 37. Ext. nuc. vomic. spirit. 0.06
Aq. destil. 30.0
Syr. alth. 15.0
M.—Give a teaspoonful three times a day.

P. 38. Ext. secal. cornut. aquos. 1.0
Glycerini,
Aq. destil. .....āā 5.0
M.—Inject a hypodermic syringeful.

P. 39. Inf. rad. rhei. (5.0—8.0) 100.0
Kali tartar. 5.0
Syrup. simpl. 20.0
M.—Give a dessertspoonful every two hours.

P. 40. Quiniæ sulph. s. muriat.,
Ferri reducti .....āā 0.05
Sacch. alb. 0.5
M.—Give this powder two to three times a day

P. 41. Kali acet. ..... 2.0—3.0
s. Liq. kali acet. ..... (5.0—8.0)
Aq. destil. 100.0
Syrup. simpl. 20.0
M.—Give a dessertspoonful every two hours.

P. 42. Decoct. cort. chinæ (5.0—8.0). 100.0
Kali acetici 3.0
Syrup. cort. aurant. 20.0
M.—Give a dessertspoonful every two hours.

P. 43. Acid. tannici. 0.05
Sacch. alb 0.5
M.—Give this powder every two hours.

P. 44. Extr. secal. cornut. aq. 1.0
Aq. destil. 100.0
Syrup. simpl. 20.0
M.—Give a dessertspoonful every two hours.

P. 45. Liq. ferri sesquichlorati 1.0
Aq. destil. 100.0
Syrup. simpl. 20.0
M.—Give a dessertspoonful four times a day.

P. 46. Iodi puri ..... 0.03—0.05
Kali hydroiodici 1.0
Aq. destil. 100.0
Syrup. simpl. 20.0
M.—Give a dessertspoonful three to four times a day.

P. 47. Sulphur. citrin,
Calc. vivæ.
Boil with water 800.0 until 500.0 remains, then filter.
To be used as an inunction.

P. 48. Acid. salicyl. ..... 2.5—5.0
Spir. vini,
Glycerini puri, q.s.
Vaselini 30.0
M.—To be used as an ointment.

P. 49. Acid. tannic. 2.0
Vaselini 30.0
M.—To be used as an ointment.

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