Lectures on children's diseases : A handbook for practitioners and students.

Contributors

Henoch, E. H. 1820-1900.
Thomson, John, F.R.C.P., Edinburgh.
Harvey Cushing/John Hay Whitney Medical Library

Publication/Creation

London, 1889.

Persistent URL

https://wellcomecollection.org/works/yb5k5sjk

License and attribution

This material has been provided by This material has been provided by the Harvey Cushing/John Hay Whitney Medical Library at Yale University, through the Medical Heritage Library. The original may be consulted at the Harvey Cushing/John Hay Whitney Medical Library at Yale University. where the originals may be consulted.
This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org



YALE COLLEGE LIBRARY.



1889

TRANSFERRED TO
YALE MEDICAL LIBRARY

THE NEW SYDENHAM SOCIETY.

INSTITUTED MDCCCLVIII.

VOLUME CXXV.

LECTURES

ON

CHILDREN'S DISEASES.

A HANDBOOK

FOR

PRACTITIONERS AND STUDENTS

BY

DR. E. HENOCH,

PROFESSOR IN THE UNIVERSITY OF BERLIN; DIRECTOR OF THE DEPARTMENT FOR CHILDREN'S DISEASES IN THE ROYAL CHARITÉ HOSPITAL, BERLIN, ETC.

VOLUME FIRST.

Translated from the Fourth Edition (1889)

BY

JOHN THOMSON M.B., F.R.C.P., EDIN.,

Extra-Physician to the Royal Hospital for Sick Children, and Physician for Children's Diseases to the New Town Dispensary, Edinburgh.

London:

THE NEW SYDENHAM SOCIETY.

MDCCCLXXXIX.

RJ45
889H
1

PREFACE TO THE FIRST EDITION.

THIS treatise contains almost exclusively the results of experience, gathered during thirty-seven years of practice including almost uninterrupted work at the polyclinic, in the department of children's diseases. When in 1872, I was placed in charge of the Children's Wards in the Royal Charité Hospital, I was enabled to an unusual extent to increase the number (already very large) of my observations on all periods of childhood; and also to place these on a firm anatomical foundation, in a way which could not have been done in a polyclinic and private practice. It is only because my material has been so exceptionally large, so carefully observed, and drawn from so many classes of the city population, that I dare claim for this work, based as it is almost entirely on my own experience, the title "Handbook for Practitioners and Students."

It stands to reason, however, that the observations of a single physician must even in the most favourable circumstances present some gaps, and that as he grows older and sees more he will always be meeting with new facts which modify the results of his former experience. Therefore it must not be expected that every disease which can possibly occur in a child will be found described or even mentioned here. Moreover, I do not think it right that a work on children's diseases should be burdened with the wearisome repetition of matters which are fully treated of in all books on general and special Pathology and Surgery, and which I am entitled to assume are familiar to my clinical class and still more so to my readers. Only those diseases will furnish the subject-matter of this work which either occur far more commonly in childhood or else, when met with at that period, show certain peculiarities as compared with the same affections in adult life. On this ground I have excluded variola, which is very rarely met with now-a-days. My passing over vaccination in silence is excusable only on the ground that I could, from my

personal observation, add nothing material to the innumerable treatises already written on the subject.

It has so long been the custom to write in the form of lectures that I need not say anything on that point. There are drawbacks to this form which I do not overlook; but I consider that these are outweighed by its advantages—freedom from restraint and greater ease of reading. Further, the introduction of cases —which here take the place of illustrations—is thereby very much facilitated. I should ask the reader not to pass over these cases, although they are very numerous; for I believe they will be of use to him. I have always endeavoured to make them as brief as possible, emphasising the points bearing on the matter in hand and avoiding the intolerable diffuseness and tediousness of detailed clinical records.

Any practitioner who has suffered from the undiscriminating and bewildering way in which the different remedies and modes of treatment are mixed up in most text-books, will, I am sure, approve of my having based advice in regard to treatment, like my clinical descriptions, solely on my own experience. The prescriptions at the end of the book (which are referred to in the text as Form. 1, 2 &c.), are not, I consider, out of place in a scientific work. Older physicians may do without them; but younger men—to whose wants I have had especial regard—will be glad to have at hand a help of this sort when beginning their practice among children.

Berlin, *January* 1881. THE AUTHOR.

PREFACE TO THE FOURTH EDITION.

In the preface to the Second Edition of this book, which appeared in April 1883 (two years after the First Edition), I wrote as follows :—"I have also received from far and near so many expressions of satisfaction and appreciation that, even had this work met generally with a less favourable reception, I should still have felt that there was no occasion in any way to change its ground plan. By gathering together a lot of experimental, anatomical and chemical matter, it is very easy indeed to give to a clinical work a dazzling appearance of the most modern science; I refrain, however, in this edition as in the former one from this kind of display, which is always ready with its hypotheses and explanations, and perplexes more than it enlightens readers, especially beginners. The stage of transition in which certain of our auxiliary sciences now are, renders extremely necessary, at any rate for the ends that concern us here, the strictest sifting and criticism. It has been my chief endeavour to be in every sense true to the reader, to criticise my own work severely—especially in matters of treatment, for it is in these that one is very apt to go astray; and out of the large store of observations I have accumulated, I have sought to lay a sure foundation for further study." I can to-day with a clear conscience repeat those words.

The fact that a Third Edition was called for in 1887 and a Fourth in the course of the present year proves that the method which I adopted was the right one. The steady advance of pædiatrics and the continually accumulating results of my own experience, have indeed rendered it necessary to recast certain sections of the book as well as to make various additions to it. By abbreviating as much as possible whatever was of minor importance and omitting some of the older records of cases, I have endeavoured to gain room for these additions without greatly increasing the size of the work.

Berlin, *September* 1888. The Author.

TRANSLATOR'S PREFACE.

CONSIDERING the out-standing position which Prof. Henoch's book holds in Germany, its introduction to English readers requires no apology. It is here neither necessary nor desirable to compare its merits with those of the many excellent works on the same subject written in our own language; but everyone will recognise the extreme and permanent value which must attach to these lectures as the outcome of forty-five years of untiring clinical work by a man of such splendid powers of observation and judgment as Prof. Henoch; and no one who reads them can fail to be struck by the wonderfully wide range of experience and reading which they represent.

In the translation, I have endeavoured to follow the original as closely as possible, except where a somewhat free rendering was necessary to make the meaning clear. As many of the German pharmaceutical preparations differ a good deal in composition and strength from those used in this country, I have, in many instances, altered the forms of the prescriptions a little to bring them into accordance with our own pharmacopœia. I have also, in rendering the various weights, measures, and temperatures, converted the terms of the metric system into those more commonly used among ourselves. I have adopted Prof. Henoch's method of drawing attention to certain emphatic words and phrases, as well as to the names of authorities, by "spacing-out" the letters instead of using italics; this will I believe be found helpful by the reader, although it may at first strike him as unusual and even a little perplexing. A full index will be given at the end of the second volume.

My warmest thanks are due to my friend Dr. Barbour for invaluable help of every kind in connection with the translation, and to Mr. Wm. Macdonald for much literary advice and assistance.

Edinburgh, *2nd March*, 1889. J. T.

CONTENTS.

PAGE

PREFACE TO FIRST EDITION v

PREFACE TO FOURTH EDITION vii

TRANSLATOR'S PREFACE ix

INTRODUCTION AND METHODS OF EXAMINATION ..... 1—21

Children's Diseases—Why worthy of special study, 1; infantile mortality, 2; methods of clinical examination, 4; auscultation, 5; percussion, 6; rate of breathing, 7; the pulse, 8; the heart, 9; laryngoscopic examination, 10; mouth and throat, 10; physiological differences between children and adults, 11; examination of the head, 12; "brain murmur," 12; fontanelles and sutures, 13; buccal cavity, 15; tongue, 16; heart, 16; temperature, 17; urine, 18; fæces, 19; manifestations of pain, 20; attitude during sleep, 21.

SECTION I.

DISEASES OF NEW-BORN INFANTS.

ICTERUS NEONATORUM 23

Symptoms, 23; causation, 24; treatment, 27; complications, 27; obliteration or absence of excretory bile-ducts, 28.

TRISMUS, OR TETANUS NEONATORUM 28

Symptoms, 29; prognosis, 30; post-mortem appearances, 31; causation, 31; treatment, 33; certain other convulsive affections, 34; "encephalitis interstitialis," 34; symptoms and pathology, 35.

CEPHALHÆMATOMA 36

In early infancy, 36; pathology and symptoms, 36; in older children, 38; treatment, 38; diagnosis, 38.

HÆMATOMA OF THE STERNO-MASTOID 39

Symptoms, 39; pathology, 40; treatment, 41.

SWELLING OF THE MAMMARY GLANDS 41

Symptoms and pathology, 41; treatment, 42.

ERYSIPELAS NEONATORUM 43

Causation, 43; symptoms, 45; complications, 47; treatment, 47; erysipelas in older children, 48.

SCLEREMA NEONATORUM 51

Symptoms, 51; œdema of new-born children, 53; pathology of œdema, 55; prognosis in both diseases, 56; treatment, 57.

PEMPHIGUS NEONATORUM 57

Acute form, 57; causation, 60; treatment, 61; cachectic or syphilitic form, 61.

APHTHÆ OF THE PALATE 63

Appearances and pathology, 63; treatment, 64.

MELÆNA NEONATORUM 65

Symptoms, 65; pathology, 66; treatment, 68; ulcer of œsophagus, 69.

SECTION II.

DISEASES OF INFANCY.

PAGE

I. Infantile Atrophy 70
Development of the infant's alimentary canal, 70; breast *versus* handfeeding, 70; symptoms of atrophy, 71; causes, 73; complications, 73; detailed description of symptoms, 73; duration, 75; causes of death, 75; post-mortem appearances, 76; prognosis, 76; hygienic treatment, 77; dietetic, 78; breast-milk, 78; substitutes for breast-milk, 80; cow's milk, 81; condensed milk, 83; Nestlé's food, 83; other "infant's foods," 84; alcoholic stimulants, 84.

II. Thrush 85
Symptoms, 85; pathology, 87; cultivation-experiments, 89; inoculation-experiments, 90; diagnosis, 91; treatment, 91.

III. Hereditary Syphilis 92
Symptoms in well-marked cases in infants, 92; less typical manifestations, 94; affections of the skin, 94; of the mucous membrane, 96; of the intestine, 97; of the lymphatic glands, 97; of the bones, 97; pseudo-paralysis, 101; affections of the joints, 104; of the testicles, 105; of the liver, 106; of the spleen, &c., 108; of the nervous system, 108; of the vascular system, 110; prognosis, 111; recurrence, 111; rickets as a sequela, 112; origin, 113; vaccination-syphilis, 113; question of heredity, 114; treatment, 117; suckling, 118; syphilis in older children, 120; syphilis tarda, 120; condylomata, 121; affections of the tongue, skin, lymphatic glands, 122; of the bones, naso-pharynx, teeth, 123; treatment, 124.

IV. Dyspeptic Conditions of Infants 124
Vomiting of healthy infants, 124; peculiarities of infant's stomach, 124; dyspeptic vomiting, 125; dyspepsia intestinalis, 126; causation of dyspepsia, 126; feeding-bottles, 127; unsuitable food, 127; pathology of dyspepsia, 127; "fat-diarrhœa," 130; acute dyspepsia, 131; "gastro-malacia," 132; dietetic treatment, 133; medicinal treatment, 137.

V. Coryza of Infants 142
Symptoms, 142; suspicion of syphilis, 142; dyspnœa from sucking-in of the tongue, 143; treatment, 145; diphtheritic coryza, 145.

VI. Retro-pharyngeal Abscess 145
An uncommon condition, 145; pathology and symptoms, 146; diagnosis, 147; treatment, 148; rupture into pharynx, 151; into external auditory meatus, 152; causation, 153.

VII. Dentition and its Symptoms 154
Many diseases attributed to dentition, 154; question of reflex irritation, 155; order of eruption of first teeth, 155; presence of teeth at birth, 156; local morbid symptoms produced by symptoms, 158; order of eruption of teeth, 159.

SECTION III.

DISEASES OF THE NERVOUS SYSTEM.

I. Infantile Convulsions 161
Derangements of motor functions commoner in infancy than those of sensory, 161; symptoms, 162; treatment of attacks, 163; causation, 166; organic disease, 166; rickets and teething, 167; digestive disorders, 167; other nervous phenomena brought on by digestive disorders, 170; treatment, 171; influence of internal parasites and foreign bodies in causing convulsions, 172; convulsions ushering in febrile diseases, 172; uræmia, intermittent fever, 174; psychical causes, 175; connection with epilepsy, 175; epilepsy in childhood, 177; further treatment, 178.

PAGE

II. Laryngeal Spasm 179
Slight degree observed in healthy children when screaming, 180; symptoms, 180; alternates with eclampsia, 181; association with rickets, 182; family tendency, 182; frequency of attacks, 183; cause of sudden death, 184; causation, 184; treatment, 186.

III. Idiopathic Contractures 187
Frequently associated with laryngeal spasm and convulsions, 187; symptoms, 187; with cerebral tuberculosis, 188; with reflex irritation, 188; relation to tetany, 190; tremor, 191.

IV. Spasmus Nutans—Nodding Spasm 192
Illustrative cases, 192; clinical description, 194; reflex causes, 194; central causes, 195; with chorea magna, 196; treatment of reflex form, 196; other spasmodic conditions, 196; convulsive laughter, 196.

V. Chorea Minor—St. Vitus' Dance 197
Symptoms, 197; hemichorea, 199; less common symptoms, 201; course, 202; post-mortem appearances, 203; relapses, 204; pathology, 205; causes, 206; connection with rheumatism, 207; heart-disease, 208; other causes, 209; effect of intercurrent diseases, 210; treatment, 211; "chorea electrica," 214; treatment, 216.

VI. The Hysterical Affections of Children 216
Nature, 216; classification, 217; first class—chiefly physical symptoms, 217; second class—chiefly convulsive symptoms, 221; third class—co-ordinated movements, 225; fourth class—with neuralgic or trophic disturbances, 230; hæmatemesis, 231; other symptoms, 232; question of simulation, 232; hysterical paralysis, 233; causation, 234; treatment, 236; prognosis, 237.

VII. Pavor Nocturnus—Night Terrors 239
Symptoms, 239; causation, 239; treatment, 241.

VIII. Peripheral Paralyses 241
Of facial nerve at birth, 241; in older children, 242; causes, 243; of other cranial nerves, 245; of arm at birth, 245; in older children, 246; treatment, 246; paralysis after convulsions, 246.

IX. Spinal Infantile Paralysis 247
Symptoms, 247; onset, 248; further progress, 250; deformities, 252; pathology, 252; affection of facial nerve, 256; diagnosis, 257; causes, 259; treatment, 259; paralysis from vertebral disease, 261; sclerosis and spastic spinal paralysis, 262.

X. Pseudo-hypertrophic Muscular Paralysis 263
Symptoms, 263; pathology, 265; treatment, 266.

XI. Apoplectic Conditions 267
Symptoms, 267; onset, 267; cerebral hæmorrhage, 267; hæmorrhagic encephalitis, 268; causes of hæmorrhage, 269; pachymeningitis, 272; embolism, 272; thrombosis, 273.

XII. Cerebral Tuberculosis 273
Frequency of its occurrence, 273; diagnosis, 274; symptoms, 274; latency, 276; duration, 280; pathology, 280; localisation, 281; chronic hydrocephalus, 284; treatment and natural cure, 286.

XIII. Tumours of the Brain 286
Sarcomata, 286; gummata, 289; other tumours, 289; abscess, 289.

XIV. Atrophic Cerebral Paralysis 290
Symptoms and diagnosis, 291; pathology and illustrations, 292; treatment, 296.

PAGE

XV. Chronic Hydrocephalus 297
Diagnosis from rickety cranium, 297; symptoms, 297; cranium, 297; mental development, 299; power of motion, 300; post-mortem appearances, 301; pathology, 302; hydrocephalus externus, 303; pachymeningitis, 304; treatment, 307; acute hydrocephalus, 309.

XVI. Hyperæmia of the Brain—Thrombosis of the Sinuses 310
Pathology, 310; arterial hyperæmia, 310; cause, 310; treatment, 311; other causes, 312; venous hyperæmia, 313; "hydrocephaloid," 313; treatment, 314; sinus-thrombosis, 315; pathology, 315; diagnosis, 316; treatment, 317.

XVII. Tubercular Meningitis 317
Acute hydrocephalus, 317; classical form of the disease, 317; symptoms, 318; variations from typical course, 326; when combined with cerebral tuberculosis, 329; pathological anatomy, 329; causation, 333; treatment, 334.

XVIII. Purulent Meningitis 336
Epidemic occurrence, 336; pathological anatomy, 336; sporadic case, 337; symptoms, 337; chronic form, 343; incomplete recovery, 344; causes, 344; treatment, 347.

XIX. Neuralgic Conditions 349
Sensory disturbances comparatively rare in childhood, 349; migraine, 349; cause, 349; symptoms, 351; treatment, 352.

SECTION IV.

DISEASES OF THE RESPIRATORY ORGANS.

I. Inflammation of the Nasal Mucous Membrane—Rhinitis 354
Symptoms, 354; rhinitis pseudo-membranacea, 355; chronic rhinitis, 355; treatment, 356.

II. False Croup 356
Onset, 356; symptoms, 357; recurrence, 357; treatment, 358; occasionally followed by true croup, 359.

III. Atelectasis of the Lungs 359
Pathological anatomy, 359; causation, 360; diagnosis, 361; congenital atelectasis, 361.

IV. Inflammatory Affections of the Larynx and Trachea 363
Idiosyncrasy, 363; symptoms, 363; treatment, 364; laryngeal obstruction, 365; its symptoms, 365; its diagnosis, 366; treatment, 367; œdema glottidis, 369; relation of croup to diphtheria, 370; simple inflammatory croup, 371; "ascending croup," 372; symptoms of croup, 374; permanent injury to the brain, 376; treatment, 377; indications for tracheotomy, 378.

V. Bronchitis and Catarrhal Pneumonia (Broncho-pneumonia) 379
Predisposing causes, 379; in very young children, 380; its treatment, 381; catarrh of small bronchi, 381; symptoms and physical signs, 381; diagnosis between bronchitis and broncho-pneumonia, 383; capillary bronchitis, 383; broncho-pneumonia, 384; physical diagnosis, 384; symptoms, 386; tendency to become chronic, 388; abscess-formation, 389; causation of broncho-pneumonia, 390; predisposition to bronchial catarrh, 391; spastic contraction of bronchial muscles, 395; treatment, 395.

VI. Croupous Pneumonia 399
Common in childhood, 399; pathological anatomy, 399; clinical features, 400; mixed forms of pneumonia, 400; diagnosis, 401; dissecting pneumonia, 402; symptoms, 402; pneumonia migrans, 406; complication with pleurisy, 407; course and termination, 407; prognosis, 411; termination in abscess-formation, 412; treatment, 413.

PAGE

VII. Chronic Pneumonia 414
Follows either broncho-pneumonia or croupous pneumonia, 415; symptoms, 416; termination, 417; bronchiectasis, 417; treatment, 418.

VIII. Pleurisy 419
Varieties, 419; latent pleurisy, 419; cerebral symptoms, 419; physical signs, 423; complications, 424; causation, 425; termination, 429; treatment, 430; indications for puncture, 430.

IX. Tuberculosis of the Lungs 433
Connection between tubercle and caseation, 433; pathological anatomy, 434; symptoms and diagnosis, 435; latency, 437; caseation of tracheal and bronchial glands, 440; diagnosis of disease of bronchial glands, 442; termination and complications, 443; hæmorrhagic diathesis in tuberculosis, 446; separate successive attacks, 447; treatment, 448.

X. Gangrene of the Lungs 449
Pathology and causation, 449; illustrative cases, 450.

XI. Whooping Cough 452
Diagnosis, 452; symptoms, 453; first stage, 453; second stage, 453; third stage, 457; variations from usual course, 458; complications, 460; causation, 462; contagion, 463; nervous element, 464; prognosis, 465; treatment, 466.

SECTION V.

DISEASES OF THE CIRCULATORY ORGANS.

I. Diseases of the Large Blood-vessels 470

II. Congenital Cyanosis 471
Causation, 471; symptoms, 471; diagnosis, 472; malformation of the heart without cyanosis, 473; prognosis, 474; hæmatomata on valves, 476; treatment, 476.

III. Inflammation of the Pericardium, Endocardium, and Myocardium 476
Rheumatic origin, 476; latency of heart disease, 477; acute cases, 477; endocarditis recurrens, 479; recovery from endocarditis, 479; endocarditis preceding rheumatism, 480; other causes of endocarditis, 483; pericarditis, 485; tubercular disease of pericardium, 486; myocarditis, 489; aneurism of the heart, 490; fatty degeneration of heart, 490; hypertrophy and dilatation, 491; treatment of heart-disease, 492.

INTRODUCTION AND METHODS OF EXAMINATION.

GENTLEMEN,—The treatment of Children's Diseases is usually regarded as a special branch of medicine. I hold, however, that this view is scarcely quite correct, because almost all the diseases of children, with very few exceptions, occur in adults also. Still, these diseases are made a special study of, special cliniques are set apart for them, and they have a copious literature of their own. This is chiefly for the following reasons:

(1) Very many of the diseases we are dealing with occur in children with far greater frequency and in a much more striking form than in later years (the acute exanthemata, whooping cough, different forms of dyspepsia, tubercular meningitis, &c.);

(2) The medical examination of a sick child demands a special dexterity which—however much skill one may have in examining adults—can only be acquired by diligent practice amongst children.

Then we must remember that the young physician especially, whose work almost always lies to begin with among the large families of the lower classes, has at the very commencement of his practice a majority of children among his patients. This fact, which was not formerly appreciated as it deserved, is now being more and more realised; at least I think I am justified in gathering this from the ever-growing numbers of my clinical class in which there are many young practitioners. It seems all the more striking that our school alone regards the study of Children's Diseases as not being an essential branch of science, and, sheltering behind long obsolete statutes, grudges a special chair to Pædiatrics.

I must, however, at once admit that even the most earnest study of children's diseases and the richest experience will not

always save you from very painful disappointments in the results of your treatment. Unfortunately the conditions of life in early childhood are such that even the most rational treatment of children's diseases along with the greatest devotion on the part of the physician is in vain in a terribly large number of cases. The attention of the scientific world and the public concern have at all times been turned to the enormous mortality of this period of life, but have not yet been able to cope successfully with this fearful state of things. It has been proved beyond a doubt by statistics that the mortality of children is highest in the first months of life, that during the whole of the first year it is twice as large as that of any later year, and that it begins gradually to diminish after the second, and only reaches the usual rate after the fifth year. Of every 1,000 children born, about 200 die in the first year of life; while the general mortality of the population is about 25 per 1,000.

I can best demonstrate this to you by stating the following figures:—During the years 1874—85, 13,980 children were treated in my department in the Charité, of whom 7,815 were under, and 6,165 over two years of age; there died of the former 5,368 or about 70 per cent., of the latter only 1,420 or about 23 per cent. The first half year of life is quite remarkable in this respect, for out of 4,393 infants under six months, 3,409 died, that is about 78 per cent. These fearful results agree with those drawn from much wider statistics; but with regard to all of them we must certainly take into account as unfavourable factors, the residence in the hospital and the wretched condition of the majority of the very young children on admission.

This enormous mortality of the first two years of life and especially of the first six months is accounted for by two sets of causes, one of which is to be sought for in the natural development of the child, and the other in its surroundings. As you are aware, the development of the child's body by no means comes to an end at birth; but on the contrary, quite apart from growth, the organism subsequently undergoes most important changes. I need only remind you of the closure of certain fœtal blood channels, the differentiation of the grey and white matter in the brain, the development of the intestinal glandular system, the eruption of the teeth and the growth of the bones—processes which of themselves have a tendency to evoke pathological changes in the organs affected. While children of the privi-

leged classes, thoughtfully cared for and appropriately fed by affectionate parents, pass through these threatening evolution-processes more easily, we find that under the unfavourable outward conditions of life which exist among the poor, many pernicious influences tell against the normal development and direct it along pathological lines. The foul air of small overcrowded rooms, the more or less unsuitable ways of feeding which are so injurious to a child's stomach, the influence of cold and hunger, the want of a mother's care (for which that of an unprincipled stranger has only too often been substituted), are all factors which work together to hinder the normal processes of development, and furnish us with those miserable pictures of disease confronting us in the consulting-rooms of parish doctors, in polyclinics, and in the children's departments of hospitals. Many of these unhappy creatures carry with them from the very beginning the germ of death derived from a diseased mother, and fall victims to congenital debility within the first few days after birth. Many others perish from inherited syphilis; most become atrophic, being reduced by constant diarrhœa; or their numbers are decimated by repeated attacks of bronchitis, with secondary enlargement of the bronchial glands, ending in caseation and general tuberculosis. Many of these children are illegitimate, and not a few of the mothers, as I can assure you from my own large experience, send to the hospital the child which has become a burden to them, not because they wish it to be restored to health, but only in the hope—which is too often justified—that they may be freed from it for ever. A large number of children of this class taken into my ward died on the very day of admission. Against such wretched social conditions our efforts as medical men are often powerless, and indeed after we have had some experience of them we are apt from the very first to despair of doing anything. The unsolved and almost insoluble problem in connection with this matter—the real causal indication—is how to remove the unfavourable conditions I have alluded to, for against them medicine in itself has no power whatever.[1]

[1] What a blessing Foundling Institutions with outside nursing, carried out on a large scale, are in such circumstances, may be learned anew from the excellent report of Epstein on the results of the Bohemian Foundling Institution during 1880—84 (*Archiv f. Kinderheilk.*, vii., Heft 2).

We have next to consider the methods of clinical examination, which, during the first years of life at least, differ essentially from those employed in the case of adults. The examination of children is rendered more difficult by the fact that they cannot talk, or at least are unable to give sufficient information to the doctor. In private practice we are helped in this respect by having the mother's account, but in hospital we have usually to content ourselves with a purely objective examination, as in the case of a sick animal, without any help from the relatives or clinical history. The difficulty is further increased by the timidity of the children and their dislike to the physician as a stranger. While in examining adults it is best to explore in order one system of organs after another, regardless of the interruptions of the patient, and to finish up with the history of the case, you will frequently have to abandon this method in childhood. For the refractoriness of the little patients obliges you to seize every favourable moment for the inspection or auscultation of parts which can only be properly examined during a quiet interval, *e.g.*, the fauces or the heart. In this way the examination of patients is apt to lose its continuity and become unmethodical; and this may render the final summing-up of the results obtained more difficult, especially to the inexperienced. On the other hand the anamnesis is naturally much shorter and simpler in children, and this tends to facilitate the gathering-up of the features of the case for a diagnosis. No fixed rules can be laid down for our bearing towards the children. Many physicians, it is true, have more sympathy with children than others, but even those who show most fondness for them will be liable often enough to be very considerably put out by their crying and noisiness while they are being examined. This resistance is to be overcome either by kindness or by firmness, according to the character of the child and the kind of temper it is in at the time. To know how this is to be done will, at the commencement of practice, be very perplexing and difficult, but as our experience grows, the difficulty will be less felt till at last it becomes scarcely appreciable. Many children will allow themselves to be kept tolerably quiet while you are examining them if their attention is aroused by holding before them a watch or toy or lighted candle, or by giving them a stethoscope to play with. For especially important cases we have in chloroform a means by which we are able to

overcome all resistance and to obtain perfect quiet; it is particularly useful in cases where we have to examine the abdomen, bladder, or rectum, and in painful joint affections.

In examining children in the first years of life it is best to have them in their mother's or nurse's arms in front of the physician, but with their faces turned to a window. When it is at all possible I have the children taken out of bed and put in this position even although they are feverish, because one is aided considerably by the co-operation of the nurse, who supports the child and keeps it still, as well as by having better light. Often however the child struggles against the hands trying to hold it, moves itself about, and turns and twists so as to make both percussion and auscultation extremely difficult. On this account an attempt has been made to follow the child's movements by using a stethoscope, the tube of which is made of india-rubber, and this may certainly be done more easily with this than with a solid instrument. After trying these stethoscopes, however, on many occasions, I have quite given them up because they so often give rise to confusing adventitious sounds; and I therefore recommend you to use an ordinary stethoscope.

While auscultating, you must always hold the lower end of the stethoscope between your fingers, both in order to be sure that it is thoroughly in contact with the chest wall and to avoid exerting too strong pressure with your head, which would at once make the child cry. It is a good plan to have a circle of india-rubber on the lower end of the stethoscope to modify the pressure; but it must be often renewed, for it gives rise to creaking sounds when it gets old and worn. Very restless children must be auscultated directly by the ear, and in this way the person examining can easily follow even the most extensive movements of the patient if he has a firm grasp of the chest and keeps his head always in contact with it. Many physicians think that their duty is done when they have examined the back, but let me strongly impress upon you never to neglect the front and sides of the chest. In a good many cases I have found the signs of pneumonia under the clavicle when everything behind was normal; and I have often discovered fine crepitations in the tongue-shaped process of the left lung where it overlaps the pericardium, when they could not be made out at all, or at least not nearly so distinctly, over other parts of the thorax. The

front of the chest may be examined with the patient either sitting or lying (the latter especially in the case of very young children); but the back only while he sits or lies on his side, never when he is lying on his face. The compression of the abdomen caused in this latter position must push up the abdominal organs and diaphragm and so diminish the capacity of the chest; and, when the respiratory organs are already affected by disease, not only will the dyspnœa be increased by this, but sudden death may be caused during the examination.

Percussion is far more irksome to many children than auscultation, and the crying which it causes interferes very much with our obtaining definite results. Moreover, every obliquity of posture and every muscular contraction occasions a slight change in the note; and you will understand from this how careful we have to be in estimating differences in the percussion note when the children are restless. I have very often thought that I made out a difference between the notes on the two sides of the chest the first time I examined a patient, and have afterwards convinced myself of my mistake by repeating my percussion with the thorax at rest and the child sitting straight. In doubtful cases we have in auscultation the best means of controlling our results. You must, moreover, never neglect to percuss both during inspiration and expiration, especially in crying children, because in them the parts percussed are more or less empty of air while the cry lasts, and give a proportionately dull and empty sound which disappears during inspiration. This (as Vogel truly observes) is very specially the case at the extreme base posteriorly, where the liver, forced upwards by crying and straining, may give rise to impairment of the note and consequent fallacy. During such examination our patience is often sorely tried, and it may be very difficult to avail ourselves quickly, for the purposes of percussion, of the inspirations which at long intervals interrupt the crying. Besides this, little children have a habit of holding their breath as long as possible, especially when they are being auscultated. It is with impatience or even annoyance that we wait for them to draw a breath; the sign however is a favourable one, because serious affections of the respiratory organs do not generally allow the patient to hold his breath for any length of time. Crying interferes much less with auscultation than with percussion; in fact, I find that any abnor-

mal sounds that are to be found in the lungs are much more easily heard during the deep inspirations which occur in the intervals of crying than during quiet breathing. On this account I never put myself much about to quiet a crying child before auscultation, and only require absolute silence from those round about.

As regards percussion I should advise you to let your stroke on the pleximeter be as light as possible. The conditions of the child's thorax as regards resonance are so favourable, owing chiefly to the elasticity of its walls, that strong percussion may, by setting up simultaneous vibrations in more distant parts, elicit a loud full sound even over parts which no longer contain air, and which accordingly give only a dull and empty sound with a light stroke. For percussing children I use a small ivory pleximeter with an ordinary plessor; and it is not necessary to use a finger of the left hand instead of the pleximeter except in cases of great emaciation (where the intercostal spaces are sunk in) and in percussing the supraclavicular region.[1]

To judge of the frequency of the respiration you must examine the child in as quiet a state as possible, best of all during sleep, if this can be managed. All excitement, crying &c., tends to render the results unsatisfactory. By laying your one hand very gently on the child's thorax or abdomen while you hold your watch in the other, you may time the rising and falling of the respiration. In weak conditions, even in children who are not crying, this part of the examination is often interfered with by the holding of the breath already alluded to, in which pauses of respiration alternate with short breaths rapidly succeeding one another. For this reason it is very difficult to state the normal number of respirations at a given age, and this accounts for the very diverse statements of different authors. We know that as a general rule it amounts in new-born babies to 32—36 in the minute, that later it sinks to about 30, but that

[1] Any work is to be gratefully welcomed which gives us independent and exact observations on the peculiarities of physical diagnosis in children; and in this connection Sahli's book (*Die topographische Percussion im Kindesalter:* Bern, 1882) deserves especial mention. Its practical results, however, seem to me scarcely proportionate to the work expended upon it, especially in the matter of the percussion of the thorax. And indeed I am of opinion that the control of the results of percussion by auscultation is worth far more for the diagnosis than all set rules, which are only too often liable to exceptions due to chance circumstances.

even in children of 7 or 8 it remains higher than in adults; and indeed, like the frequency of the pulse, it is in inverse ratio to the age of the child.

The action of the child's heart is, to begin with, more rapid; it is excitable in a high degree by every psychical impression, and dread of the physician who is more or less a stranger to him is especially apt to increase the pulse-rate—often to such a degree that to count it is absolutely worthless for diagnosis. The best example of this influence is given us by children who suffer from jaundice. The slowing of the pulse which is so characteristic of this disease in adults, I have never observed in childhood till about the seventh year; and I can only account for the fact by supposing that the above-mentioned excitability of the nervous system of the heart fully compensates for the retarding influence of the bile-acids. Accordingly, a correct counting of the pulse, especially in very young children, can only be undertaken during sleep. This is easily done if one keeps perfectly still and lays the point of one's finger softly on the radial artery. We must at the same time remember that the pulse is occasionally somewhat irregular during sleep even in perfectly healthy children and that this need not be in any way alarming. Of just as little real importance is the irregularity or even retardation of the pulse which is observed (sometimes for weeks) during convalescence from highly febrile diseases (pneumonia, typhoid, measles &c.), unless the cause of it is clearly apparent. One can never get reliable results by counting the pulse in states of debility (irrespective of exceptional cases and in older children), and hence it arises that the figures given by various authors differ so materially from one another.

On an average, I think we must regard a pulse-rate of 120—140 as normal for the first months of life, and of 100—120 for the second year, after which a gradual decrease takes place. In children of 3—6 years of age the pulse still keeps above 90, and it is only after the second dentition that it gradually begins to approach the adult rate. Now, the frequency of the respiration alters in a corresponding way; and so we have always to keep in mind its relation to that of the pulse as 1 : 3½ or 4. I repeat, however, that, especially in childhood, such calculations from averages are of little or no practical utility, for the reasons already given. It is only in certain definite circumstances that

the pulse-rate acquires any diagnostic or prognostic value—for instance, the retardation at the beginning of tubercular meningitis and the extreme acceleration towards its close, or again the enormous rapidity in scarlet fever. The rhythm and quality of the pulse have always appeared to me to have much greater significance for the physician. The inequality and irregularity of the beats in the first stage of tubercular meningitis and the diminution and gradual disappearance of the pulse-wave in severe diseases, especially those of an infectious nature, are points of very great importance to which I shall frequently revert in these lectures.[1] The same may be said of the relation of the pulse to the respirations which normally is $3\frac{1}{2}$ or 4 : 1. If this relation is disturbed for any length of time—if for example 40—60 respirations occur to 120—140 beats—you may almost certainly expect to find some affection of the respiratory organs. Even to this rule however the physician must be prepared to find exceptions; rickety children with more or less deformed chests always breathe more quickly than healthy ones, Nervous excitement also may have this effect; in little children during the first dentition I have occasionally observed a rate of breathing of 60—90 in the minute, which lasted many months with otherwise uninterrupted good health, and gradually as teething came to an end this was replaced by the normal rate: one could only regard such cases as due to a reflex irritation of the respiratory centre. Phenomena of this kind also appear transiently in the course of whooping cough and tuberculosis of the bronchial glands. If the breathing appear not only quicker and shallower but at the same time more laboured, certain of the accessory muscles coming into play and expiration becoming noisy, it is still more significant. Where these conditions exist, physical examination will almost always reveal the presence of bronchitis, pneumonia, pleurisy, or some other respiratory disease.

The examination of the heart in the early years of life is attended with almost greater difficulties than that of the lungs. The rapid succession of its beats, and the constant crying which goes on during its examination, often make it impossible to speak positively as to the purity of the sounds and the results of

[1] Special instruments for measuring the tension of the pulse in children (*e.g.* the Sphygmomanometer recommended by von Basch—*Archiv f. Kinderheilk.*, v., S. 272) can hardly be of much use in ordinary practice.

percussion. We must also avoid pressing with the ear on the stethoscope so firmly as to indent the costal cartilage, for this may at once render the heart-sounds impure or even blowing. I may also mention that we can hear cardiac murmurs which are due to valvular lesions more distinctly over the lower part of the back in children than in adults, even although the lower lobes of the lungs are normal.

The laryngoscopic examination is, however, after all the most trying. In very young children it is almost out of the question, and even from older ones, we usually, if not always, meet with an amount of resistance very hard to overcome. We may facilitate the introduction of the mirror by painting the pharynx and entrance to the larynx with a solution of cocaine (5—10 per cent.) to render them non-sensitive; but even when one does succeed in introducing it properly, and getting it fixed in the right position, its surface soon becomes so obscured by the secretion which is forced up from the throat by crying, coughing, and retching, that it is impossible to get a distinct image. Although I do not deny that under favourable circumstances many children may be satisfactorily examined by this method, yet I maintain that in a far greater number of cases it yields either no results or else very unreliable ones. The conclusions which the older authors pretended to draw from the character of the cry are even less to be depended upon. Its hoarseness or its being replaced by a distressed whimper are the only two conditions to which I can attribute any practical importance. New-born children, as you know, shed no tears when they cry; the secretion of the lachrymal glands must therefore be still deficient at this age like that of the salivary glands of which we shall have to speak later on.

After a little practice the examination of the mouth and throat rarely present any difficulties, and I therefore often wonder at the clumsiness with which many physicians perform it. If the child does not open his mouth of his own accord when told to do so, the best thing to do is to push the under lip over the margin of the lower jaw with the forefinger and to press on it. This also prevents the danger of the physician's being bitten, for every attempt at doing so will be very painful to the child when his lip is between the finger and his teeth. A little perseverance will usually soon overcome any difficulty occasioned by the child's obstinately keeping his mouth closed, especially if

you force him to breathe through the mouth by compressing his nostrils. As soon as you get your finger over the lower row of teeth, the child usually opens his mouth sufficiently for the mouth and throat to be satisfactorily inspected. In stubborn cases you can easily accomplish your end by using a tongue spatula. The main point is to obtain a good illumination of the fauces either by bright daylight, or, where this cannot be had, by a small candle with its flame fixed in front of a silver spoon held in the same hand. With this simple contrivance, which acts as a reflector and can be got quickly anywhere, one gets a capital light, and I very often make use of it. Nevertheless, you will from time to time come across children who resist all our efforts to open their mouths with invincible obstinacy, so that at last you have either to give it up, or to attempt to attain your end by forcing the jaws apart.

In order to make the results of your examination available for diagnosis, you must have some knowledge of the points in which certain conditions in childhood, even in a state of health, differ from the same in adults; for otherwise you will be very liable to find yourself speaking of normal conditions as pathological.

In the first place I would draw your attention to the differences in character which the normal breath-sounds present at different ages. During the first weeks and months after birth the breath-sounds are still rather weak, because the short superficial respiration is not sufficient to drive the air strongly in through the bronchi; and, for the same reason, percussion at this age gives a less full note over the whole chest. About the middle of the first year, however, the breath-sounds begin to acquire those peculiarities which we find under certain circumstances in adults, and call puerile breathing. The breath-sounds have a strikingly sharp, almost blowing character; the inspiration is almost the only sound heard, the expiration in a state of perfect rest being scarcely if at all audible, though excitement renders it more distinct.[1] The sharp puerile breathing is further exaggerated in cases where the thorax is narrowed by rachitic deformity, and it is therefore conceivable

[1] I must here further mention that in quite healthy children the peculiar rhythm which is characteristic of respiratory disease in childhood—namely, a prolonged "grunting" expiration predominating over a quite short inspiration which follows it like an echo—may occur transiently from fear alone.

that in healthy children also the relative narrowness of the thoracic cavity may give rise to the rough blowing character by slight compression of the lung when it expands in inspiration.[1]

The morbid sounds originating in the lungs or pleuræ are not, on the whole, different from those in adults. Only one finds medium, and especially fine crepitations far oftener, frequently with this peculiarity that they are very numerous on expiration, while inspiration is almost entirely free from them. The type of respiration in young children up to the third year is mainly abdominal. The diaphragm and abdominal muscles work with remarkable energy, and thus there often occurs, even in health, a slight indrawing of the epigastrium and lower ribs, which suggests a pathological condition such as we find developed to a far more marked degree in serious respiratory affections. In cases of debility one need not be alarmed by irregularity of the breathing or even by short pauses; both occur not unfrequently in little children. The relative narrowness of the thorax is in marked contrast to the large size of the belly which is so often regarded as a sign of disease by anxious mothers, but in reality is caused only by the comparatively small chest, and by a tendency to the formation of gas in the intestinal canal.

Amongst the results which the examination of the head yields, an auscultatory phenomenon deserves the first mention. In children in whom the greater fontanelle is still open (that is to say, roughly speaking, during the first two years of life) when they are at rest, on applying the ear or stethoscope over the large fontanelle we often hear a more or less loud blowing murmur, synchronous with the systole of the heart. Since the respiratory murmur due to the vibration of the air-stream rushing through the pharynx can also be perceived over the fontanelle as well as every sound produced by sobbing, chewing

[1] The explanation given by Sabatier (*Etude sur l'auscultation du poumon chez les enfants:* Paris, 1863) is ingenious, but by no means indubitable. According to the laborious measurements of this author, the capacity of the bronchial branches in adult men and animals increases on the whole from the centre towards the periphery. In children, on the other hand, it diminishes, *i.e.* in them the lumen of the two branches which spring from a bronchus taken together, is smaller than that of the primary bronchus, while in adults the converse obtains. Owing to this circumstance the velocity of the stream of air in the bronchi diminishes towards the periphery in adults, while in children it increases, and the breath-sound consequently appears rougher. This roughness is further increased by the greater sharpness of the angles of cartilage, which project between two diverging branches, increasing the vibration of the air-column as it rushes past.

and swallowing—one must keep one's hand on the pulse while auscultating, to avoid errors, especially in children who are breathing very quickly. With some practice one is soon able, even without these precautions, to hear the systolic murmur easily along with the breath-sounds and to distinguish them from one another. In a very small number of cases I have heard the murmur over a closed fontanelle and at other points on the cranium. Others have perceived it also at the posterior and lateral fontanelles and in the line of the middle meningeal artery (when the fontanelles and sutures were closed), and even over the spinous processes of the cervical vertebræ. The first discoverers of this murmur, the Americans Fisher (1833) and Whitney (1843), regarded it as always indicating disease, especially certain brain-diseases; Hennig and Wirthgen on the other hand directed attention to the fact of its physiological occurrence between the 22nd or 23rd week of life and the time of closure of the fontanelle by ossification. As the result of my own numerous investigations,[1] I agree with these authors in thinking that the murmur is found pretty often even in healthy children when the fontanelle is still open; but especially in anæmic and rachitic subjects,[2] perhaps because in them the fontanelles and sutures remain open much longer than usual. The ultimate causes of this murmur are as yet undetermined. Jurascz holds that it arises in the carotid artery, owing to a relative narrowing of the vessel in the carotid groove, while Epstein, on the contrary, is inclined to compare it with a murmur occurring in the same artery in the neck. At any rate, I hold that the so-called "brain-murmur" is of no interest from a clinical point of view, and cannot be turned to account for diagnosis.

Of far more importance to us is the condition of the fontanelles and sutures of the skull. In the normal new-born child the sutures are closed by a thick intermediate substance which sometimes projects like a border, while all the fontanelles are still membranous, so that one can feel with the finger the pulsation of the brain through the anterior one. This is

[1] *Beiträge zur Kinderheilk:* Berlin, 1861, S. 170.

[2] Roger also, who has examined hundreds of children for this murmur, maintains this view. (*Recherches cliniques sur les maladies de l'enfance*, T. II.: Paris, 1883, p. 261.) *Cf.* also Rohde, "Die grosse Fontanelle in physiologischer und pathologischer Beziehung"—*Inaug. Diss.*: Halle, 1886.

especially distinct when it expands more than usual, so that the membrane projects above the level of the surrounding bones. This distended, elastic and pulsating condition of the anterior fontanelle is therefore valuable in practice as a sign of increased intracranial pressure. On the other hand, the depression of the fontanelle below the level of the surrounding surface indicates an anæmic and collapsed state of the brain such as often occurs in atrophic children and towards the end in exhausting diseases (diarrhœa, cholera). Under these circumstances we also not at all uncommonly find a displacement of the margin of the frontal under that of the parietal bone, only, of course, while the coronary suture is still membranous, so as to permit of such a displacement. While the two lateral and the posterior fontanelles are closed by ossification in the first months after birth, the large anterior fontanelle remains open. That it goes on growing in size during the first six months, as was formerly supposed, is disputed by Kassowitz, who maintains, on the contrary, that he has observed a continuous diminution from birth. The complete closure ought to take place about the 15th—24th month. Still, cases are not very rare in which, well into the third year of life, we may find a membranous spot in the region of the fontanelle that can be covered by the tip of the finger; and one must not set this down off-hand as a morbid appearance. All other variations, however,—especially a greater and more protracted patency of the greater or lesser fontanelles, a gaping of the sutures, or an unusual yielding to pressure of the margins of the bones,—must be reckoned as pathological, and will be taken up later with the consideration of rickets. The same applies to some anomalous forms of the head which are related to certain diseases (rickets, hydrocephalus), while individual differences in the form of the cranium, caused, not by disease, but by abnormal growth of bone (asymmetry, obliquity of the median line, dolichocephaly, &c.), can only lay claim to clinical interest when at the same time there are symptoms of a cerebral affection (hemiplegia, contractures, backwardness of intelligence). The difference between the adult cranium and that of the child is important, and must always be borne in mind. During the first two years of life the circumference of the vault of the skull exceeds that of the face quite disproportionately, so that the relation is stated as about 6 : 1 (in

new-born children even 8 : 1), while in adults it is $2\frac{1}{2}$: 1. We have to remember this in order to quiet the anxiety of many mothers who think that their children are hydrocephalic; especially in cases where the above-mentioned disproportion is aggravated by rachitic thickening of the cranial bones. Under these circumstances many children are unusually late of learning to hold their head up without support, which in a state of perfect health they can often do by the third month. In this particular, however, there are many exceptions, due chiefly to the greater or less strength of the muscles, especially those of the neck. We must not therefore at once assume a congenital disease of the brain merely because a child cannot hold its head erect without support when it is five or six months old, unless there happen to be other symptoms present justifying such an assumption—especially want of intellectual development, a staring look, nystagmus of the eyeballs, awkward catching movements with the hands, or complete apathy.

In examining the cavity of the mouth in new-born children you will be struck with the dark red colour of the mucous membrane. This lasts some weeks and then slowly disappears; it is to be regarded as a normal appearance. A certain amount of dryness is associated with this hyperæmia because the secretion of the saliva does not take place at the same rate as in older children and adults. Recent researches (Ritter, Schiffer and Zweifel) pretty unanimously prove that although the saliva is present from birth its quantity is so small that its sugar-forming power is to be taken into account little or not at all. The salivary secretion first begins to increase perceptibly towards the end of the second month; according to Zweifel secretion usually first begins about this time in the submaxillary gland and pancreas, although at birth the parotid contains ptyalin. This deficient secretion of saliva is also the reason why the buccal mucous membrane of infants in the first months, if it is not very carefully washed, almost always gives a somewhat acid reaction with litmus-paper, and, even after washing out, is neutral and but rarely alkaline. We shall see later how important these conditions may be in connection with the methods of feeding children.

In very many new-born infants one sees in the median line of the hard palate little yellowish white round or oval nodules

from the size of a pinhead to that of a millet-seed projecting only a little from the mucous membrane; they are either single or in a row and are sometimes surrounded by a narrow red border. These nodules are very common in the first four to six weeks of life and have not the slightest pathological significance. Bohn regarded them as occluded mucous follicles analogous to milium on the outer skin, Guyon and Thierry as epidermoid cysts, and Moldenhauer[1] as solid processes of epithelium growing into the mucous membrane and glandular tubes in process of development. Epstein,[2] however, seems recently to have hit upon the right explanation. The investigations of this writer prove that these are spaces filled with epithelium which have been left after the union of the two halves of the palate.

With regard to the tongue you will notice that in infants on the breast it is very often spread over with a thin whitish coating especially after sucking (milk-colouration). Also, that in many older children it presents a peculiar "mapped" appearance; that is to say, the dorsum of the tongue exhibits various greyish-white figures, usually with somewhat raised borders, which are sometimes sinuous, sometimes indented,—and which contrast markedly with the red colour of the normal areas. This state of the tongue (the anatomical cause of which is not yet clear) is due to a superficial irritation of the mucous membrane with copious desquamation of epithelium in places. It occurs very often in perfectly healthy children and has therefore not the least diagnostic value, and particularly nothing to do with congenital syphilis.[3]

The examination of the heart need not detain us long, as the results of it in children agree almost entirely with those in adults. For practical purposes it is sufficient to know that in emaciated children (in the second period of childhood more than in the first two years) the movements of the normal heart are often visible as a pulsation in the fourth and fifth intercostal spaces and that the ribs are more strongly bulged forwards by them than at a later age. One may also very often feel the

[1] *Archiv f. Gynäcologie*, Bd. vii., Heft 2.

[2] *Ueber die Epithelperlen in der Mundhöhle u. s. w., Zeitschr. für Heilkunde*, Bd. I.: Prag, 1888.

[3] Guinon, "De la desquamation épithéliale &c." *Revue mens. des maladies de l'enfance*, Sept., 1887.

apex-beat somewhat outside the nipple-line without there being any enlargement of the heart. Flattening of the sides of the chest due to rickets favours these appearances, which seem to depend on the higher level of the diaphragm and the consequently more horizontal position of the heart.

I have in the next place to treat briefly of the examination of the temperature, urine, and fæces. On the value of thermometry in childhood I need not waste words; its usefulness cannot be over-estimated at an age when everything depends far more on objective examination than is the case in later life. Unfortunately, however, it is only in hospital and private practice that it can be thoroughly turned to account, for at the polyclinic and in the consultation hours of parish doctors it is scarcely possible to take temperatures in a trustworthy manner, owing to the large number of patients and the want of efficient help. Under these circumstances we must content ourselves, except in specially important cases, with estimating the temperature by applying the hand; and, during the further course of the disease, trust to the accounts of the mothers who generally state correctly the times of exacerbations at least. I usually prefer to take the temperature in the axilla. Although in this position the process takes at least 10—15 minutes (therefore twice as long as in the rectum), still, one must remember that even with every precaution it is possible that the thermometer may be broken in the rectum by a sudden movement—as I have myself seen happen. If you disregard this possibility you certainly save much time by taking the temperature in the rectum; and on this account I have often myself done so in private practice, where each individual child can be thoroughly watched. The variations in temperature in children and adults are the same, except that during the first three to four months of life it has a marked tendency to fall below normal. The heat-production seems at this age to be carried on with less energy, for in very many cases of faulty nutrition, exhausting cachexia or insufficient lung-activity, we observe the temperature gradually falling unusually low—to 86° F. and even lower. We have another instance of this peculiarity in the fact that, at the age referred to, otherwise highly febrile diseases (*e.g.* pneumonia) may run their course with a normal or even subnormal temperature; of this I have had plenty of proofs in the infants' ward of my

department of the hospital. We need not, however, on this account make a special disease under the name of "Algor progressivus" as Hervieux has done, since this enormous fall of temperature may occur under the most diverse conditions possible, these having nothing in common with one another except the final ending in collapse.

The examination of the urine is very difficult in new-born children and other infants, because it is always passed into the diapers and it is very difficult to estimate with certainty its amount and colour from the examination of these. Occasionally there occur cases, even at this age, in which it is necessary to examine the urine for albumen or even for sugar; and for this purpose one must either collect it in special apparatus —in little girls in thoroughly cleansed sponges applied over the genitals, in boys in an indiarubber bladder or some such contrivance fastened round the penis—or endeavour to obtain it by the introduction of a catheter into the bladder, a method which we prefer in hospital practice.[1] In practice one contents oneself as a rule with judging of the urine of new-born children from the diapers. The wetting of these gives us a measure of the nourishment taken, and from a diminished amount of the secretion we assume (and are usually correct in so doing) that the child is either taking too little nourishment or is failing to assimilate what he has taken. It is only very recently that the urine of newly born infants has been made the subject of careful examination by Parrot and Robin[2], Dohrn[3], Martin and Ruge[4], Cruse[5], Camerer[6], and others. The results obtained by these writers do not however altogether agree. It is especially interesting to physicians that Martin and Ruge sometimes found a small amount of albumen in the urine during the first ten days after birth. In some this was transient, but in others it lasted for several days; and they are inclined to connect this condition with the expulsion of the uric-acid infarcts which occur in the renal canaliculi, and of which we shall speak later on. Cruse's researches yielded similar results, but Parrot

[1] *Cf.* Hirschsprung, *Jahrbuch f. Kinderkrankh.*, xix., S. 205.
[2] *Comptes rendus*, Bd. 82, No. 1.
[3] *Monatsschr. f. Geburtsk.*, Bd. 29.
[4] *Ueber das Verhalten von Harn und Nieren der Neugeborenen*: Stuttgart, 1875.
[5] *Jahrb. f. Kinderkrankheiten*, 1877, xi., S. 393.
[6] *Ibid.*, 1880, xv., S. 161.

and Robin state that they have never, and Dohrn that he has only rarely, found albuminuria in healthy new-born children. In children more than ten days old, Cruse[1] never found albumen, although there was a greater amount of mucus than usual in the urine which might be misleading.[2]

The fæces in infants can also only be examined on the diapers, mixed with the urine. In normal conditions they are almost without odour, so long at least as beef-tea and meat are entirely excluded from the diet. They have a feebly acid reaction, are pretty much the colour and consistence of beaten-up eggs, and are passed twice to four times in the day. Exceptions to this rule, especially a seldomer or somewhat more frequent evacuation, are not to be regarded as abnormal, unless the consistence of the motions becomes more liquid or their smell acid or offensive. In many children the colour of the fæces is not like the yellow of an egg even in the normal state, but inclines rather to a brownish shade. If the cloths are left lying for some time, the yellow colour very often becomes greenish, owing to the oxygen in the air changing the brown bile-pigment into biliverdin, and therefore, in order to form a correct opinion, one must always examine the fæces as fresh as possible. Round about the fæces on the diaper we usually see a colourless wet area caused by the urine. I should, however, point out to you here that there are cases of diarrhœa in which fæces of a tolerably normal appearance are first passed, and are followed by a more or less large quantity of a watery fluid from the rectum. The wetting of the cloths which occurs in such cases may occasion error if one thinks that it is caused by the urine and that the fæces are normal. I should not have mentioned this, had I not frequently met with cases in which there was progressing failure of strength along with the above-mentioned appearance of the cloths, the fæces in the middle being pretty well digested but having round about them a pale, apparently urinous, area. From my own observation I have convinced myself that in every one of these cases, after the

[1] *Jahrb. f. Kinderkrankheiten*, 1878, xiii., S. 71.

[2] Hofmeier (*Virch. Arch.*, Bd. 89, H. 3) refers the increase in the quantity of the urine as well as of the urea and uric acid, to the loss of weight during the first days of life and the decomposition of albumen which goes on simultaneously. He also connects the albuminuria of the first day of life with the uric-acid infarcts, while Ribbert (*Ibid.*, Bd. 98, H. 3) sees in the albuminous state of the first urine only a continuation of the transudation through the glomeruli (as yet imperfectly developed) which occurs in all embryo kidneys.

evacuation of more solid fæcal matter, a larger quantity of thin turbid fluid was expelled with force from the anus—that in fact a condition of diarrhœa was present and accounted for the loss of strength.[1]

Finally, I come to the manifestations of pain in little children, which consist almost solely of cries. It is very difficult—and that not only for the beginner—to distinguish the cry of pain from that which expresses hunger or some other undiscoverable source of uneasiness. I consider it quite unnecessary to detain you at this point—as many authors do—with a description of the various modifications of cries. Such descriptions are of no practical use. Any one can distinguish whether a child is crying lustily or only whimpering feebly, and from this we may judge of its strength; likewise whether the voice is clear, or hoarse from an affection of the laryngeal mucous membrane. Continuous loud crying which does not set up a fit of coughing is always a favourable sign in affections of the respiratory organs, because it indicates a relatively small amount of irritability of the respiratory mucous membrane. Violent fits of crying with vigorous movements of the lower limbs, especially drawing them up on the belly, usually indicate colic in infants. But in spite of these and many other hints derived from experience it is often very difficult to decide whether the cry of a child is really due to pain or to some other cause. The presence of the doctor is of itself sufficient to make many children very uneasy and to cause prolonged crying. In doubtful cases, where pressure not only on the apparently painful spot but on every other part of the body excites or increases the crying, the only way to gain your end is to wait until the child is perfectly quiet and then begin the examination over again. If while doing this you can manage to divert the child's attention from the place examined, by toys, by a watch held before it, or by turning its eyes to bright daylight (at the window), you will often—though not always—be able to find the spot which is really tender on pressure. When

[1] The investigation of the bacteria present in milk-fæces, which was first entered into by Uffelmann, has very recently been again taken up by Escherich on an extended scale, and with some success (*Die Darmbacterien des Säuglingsalter u. s. w.*: Stuttgart, 1886). According to his researches the number of bacteria in milk-fæces is relatively small—confined to two kinds only—and real putrefaction does not occur in the colon; the absence of odour in normal milk-fæces is in keeping with this.

children cry violently and will not be quieted, it is always well to have them stripped for examination. By doing so I have frequently found the cause of the violent excitement in midges or flea-bites.

In judging of the condition of new-born infants and children at the breast, I should further recommend you to observe how the hands are held during sleep. Healthy children at this age sleep, as is well-known, with their arms flexed to such an extent that the hands are directed right upwards and are on a level with the neck or lower jaw. This attitude—which is perhaps a reminiscence of intra-uterine life—is changed in the case of serious illness, and its presence may consequently be regarded as a reassuring sign. I may also remark here that healthy children usually have their eyes tight shut during sleep, but that in not a few the eyelids are noticed to remain slightly apart. One must investigate such conditions in each individual case, for, as we shall see later on, they may have a pathological significance.

SECTION I.

DISEASES OF NEW-BORN INFANTS.

The period of suckling extends from birth to about the 9th month, when the eruption of the teeth marks its close. One is justified in treating separately the beginning of this period, *i.e.*, about the first 4—6 weeks of life during which we are in the habit of speaking of the child as "new-born"; for to it belong a number of morbid conditions which later in life do not occur, or are only met with rarely and in an altered form, and which to some extent are connected with what has taken place before birth and with the sudden removal of the child from its mother's womb into the open air.

All new-born children present, in the first days after birth, a more or less intense red colour of the whole skin which is due to hyperæmia. In many children this gradually becomes paler and passes in about a week into the ordinary colour; in many others, however, there is a transition stage, the red at first giving place to a more or less deep yellow, and this we designate icterus neonatorum.

Icterus Neonatorum (Jaundice of the new-born infant).

The yellow colour is usually noticed on the second or third day after birth; it is almost never equally well-marked all over, but is more strongly developed on certain parts, especially on the forehead, round about the mouth, and on the trunk, rather than on the limbs. The more the redness I have just referred to passes off, the more distinct and general is the yellow colour. It usually has a tinge of orange, is not as a rule very intense, and may also be seen on pressure with the finger on the hyperæmic skin. It generally lasts several days, then gradually fades, and in the course of 8—14 days is replaced by the normal colour.

In examining such children, if you recall the symptoms which the jaundice of later years is wont to present, you will find very

striking differences. The urine which wets the diapers is pale; the fæces are yellow or brownish, as in the normal state. The sclerotic, however, which is often very difficult to see owing to the energetic way in which the eyelids are kept shut, shows in all cases a distinct yellow colour; also, the pale spot left for a moment on the red tissue of the gum after pressure with the finger exhibits, sometimes indeed very faintly, the yellow tinge which we are accustomed to see in the jaundice of older people. With the exception of the yellow colour of the skin there are no symptoms whatever; but on the contrary, except of course in those cases which are complicated with more serious diseases, all the functions are in good order, and it is all over within 8—10 days. The harmless character of icterus neonatorum and its very great frequency have caused it to be regarded not as a disease at all, but rather as a physiological condition.

The matter of most importance is to determine what causes the yellow colour of the skin—whether here one has really to do with bile pigment formed in the liver. The opinion originally advanced by French writers, that the yellow discolouration is not truly bilious, but merely proceeds from the red colour of the new-born child, can scarcely nowadays be seriously defended. For in icterus neonatorum, not only is the skin coloured yellow, but also the greater part of the internal organs. I have convinced myself repeatedly of this fact by post-mortem examinations; and Orth[1] describes a case where even the brain, which in jaundice is ordinarily little or not at all coloured, appeared of a deep yellow. There can therefore be no doubt that the staining of the tissues is caused by a pigment the characteristics of which apparently correspond with those of the bile. The researches of Orth give new support to this view. The observations which had formerly been made of the presence, at least after death, of crystalline pigment in the blood and various organs of new-born infants were confirmed by him; and he found that this pigment only occurred when ordinary jaundice was present or in process of disappearing.[2] This colouring matter is very abundant in the blood, kidneys, liver, and many other organs; it occurs in the

[1] "Ueber das Vorkommen von Bilirubincrystallin bei neugeborenen Kindern." *Virchow's Archiv*, Bd. 63.

[2] Out of 37 cases in which Orth found the pigment, 32 were jaundiced, and in the other 5 cases it was impossible to prove that jaundice had not been previously present.

form of red rhombic plates, or cylinders, or bundles of needles, and shows the micro-chemical characteristics of bilirubin; Orth has therefore no hesitation in regarding these crystals as bilirubin formed after death from bile-pigment formerly in solution in the blood-plasma. How this bile-pigment got into the blood remains indeed unsolved, and on this very question there is still great difference of opinion. While some regard the jaundice as hæmatogenous—arising from the formation of yellow pigment in the blood itself—others accept the theory that it is hepatogenous like ordinary obstructive jaundice. Now although (as I have myself repeatedly seen) one can at the post-mortem in many cases squeeze out little plugs of mucus from the ductus choledochus, yet the bile-staining of the intestinal contents and the normal colour of the urine indicate that these plugs are not sufficient to cause any considerable retention of bile, or reabsorption of colouring matter by the liver. Then on the other hand, in many cases one finds the ductus choledochus and hepaticus free from obstructing mucous plugs, and on this account the hæmatogenous theory of icterus neonatorum has secured many supporters. But here also there is no satisfactory proof of the cause which occasions such a considerable separation of yellow pigment in the blood. This theory would at all events presuppose a very considerable destruction of red corpuscles in the blood, and a corresponding liberation of blood-pigment, from which the hæmatoidin and bilirubin are derived. We know that the blood of infants is, at birth, relatively richer in red corpuscles than that of older persons (Thomas, Demme); and Hayem, Helot, and others have also demonstrated, by results obtained from counting the corpuscles in the blood which enter the child's body by the umbilical cord, that these are destroyed in immense numbers. Now, according to Porak[1] and others, when the umbilical cord is tied after some time (after pulsation has ceased) and a larger amount of blood has found its way from the placenta into the circulation of the new-born child, there will result this greater destruction of red blood corpuscles, a more abundant formation of pigment in the blood, and accordingly jaundice to a corresponding degree. Others,[2]

[1] Porak, *Considérations sur l'ictère des nouveaux-nés:* Paris, 1878. Schücking, *Berl. Klin. Wochenschr.*, 1879, No. 39. Violet, *Virch. Archiv*, Bd. 80, S. 353.

[2] Hofmeier, "Die Gelbsucht der Neugeborenen": *Zeitschr. f. Geburtsh. u. s. w.*, Bd. viii., Ht. 2.

again, ascribe this action to the large consumption of albumen which takes place during the first days of life and occasions a greater destruction of red blood corpuscles; the insufficiency of the liver cells and biliary passages to meet the increased demands upon them is also to be taken into consideration.[1]

The reabsorption theory as opposed to the hæmatogenous one was put on a better footing by the work of Cruse.[2] This author found the colour of the urine—when carefully collected—yellower than normal; and he further discovered by micro-chemical examination that the little yellow bodies (*masses jaunes*—described first by Virchow, and afterwards by Robin and Parrot, Violet, and others), always found in the urine in icterus neonatorum, either imbedded in epithelial cells, or floating free, or enclosed in hyaline casts, are real bile-pigment. He also states that in all cases of intense jaundice he has discovered bile-pigment in solution in the urine by shaking it up with chloroform—which former observers had not succeeded in doing. The theory of the origin of icterus neonatorum which he puts forward is, however, without anatomical basis. According to Birch-Hirschfeld[3] an interstitial œdema of the connective tissue of the liver occurs (owing to venous engorgement) which brings about compression of the bile-ducts, obstruction to the outflow of bile, and reabsorption. The circumstance that this author was always able to discover bile-pigment, and in one case bile-acids, in the pericardial fluid although not in the urine, must certainly be regarded as strongly in support of the hepatogenous theory. He considers that the bile-colouring of the fæces is due to the continuance for days of the discharge of meconium. The researches of Silbermann[4] also are in favour of the hepatogenous nature of the jaundice, and according to him it is caused by compression of the bile-capillaries and interlobular bile-ducts by the dilated blood capillaries and branches of the portal vein. However, we must always be prepared to meet with new views on the nature of this disease.[5]

The development of jaundice is favoured by prematurity of

[1] Hartmann, "Ueber den Icterus Neonatorum," *Inaug. Diss.:* Berlin, 1883.
[2] *Archiv f. Kinderheilkunde*, Bd. 1., 1880, S. 353.
[3] "Die Entstehung der Gelbsucht neugeborener Kinder," *Virch. Archiv*, Bd. 87, Heft 3, and Schulze, *Ibid.* Bd. 81, Heft 1.
[4] *Arch. f. Kinderheilk.*, viii., Heft 6.
[5] *Vide e.g.*, Quincke, *Archiv f. experim. Pathologie u. Pharmacie*, Bd. 19.

birth, weakness of the new-born child, unfavourable conditions at or after birth, the operation of cold, atelectasis of the lung tissue, defective respiration, and bad air; these explain the especially frequent occurrence of jaundice in lying-in hospitals and foundling institutions, and in children who are under the average weight.

One need scarcely speak of treatment since the affection disappears spontaneously. All that is required is good nursing and attention to the bowels when necessary.

In a considerable number of cases the jaundice is complicated by other much graver morbid conditions which are of themselves sufficient to bring about a fatal issue. Many of these children come into the world in the last degree of sickliness, emaciation, and debility; they exhibit an extensive growth of aphthæ on the mouth and gums; and suffer from the very beginning from vomiting and diarrhœa. In such conditions also I have frequently been able to discover a yellow colour and even the presence of bile in the vomited matters. The most unfavourable complication is sclerema neonatorum, fortunately a tolerably rare one. A case which occurred in my private practice in July, 1875, seems to me worthy of being noted here on account of the obscure etiology and the unexpectedly favourable result.

> A child, 14 days old, had suffered for about 10 days from jaundice, which, during the last few days, had suddenly increased in a marked degree. The motions were dark, blackish brown, soft, and scanty; the urine stains on the diapers greenish yellow. There was also an extensive growth of aphthæ reaching back into the pharynx with livid colour of the mucous membrane, and the child was steadily sinking, in spite of having a capital nurse and drinking abundantly. One was struck with the great number of miliary red spots which were scattered over the greenish-yellow skin of the neck, back, and extremities; these did not disappear on pressure with the finger, here and there projected somewhat, and later on passed off with a slight desquamation. The child recovered, contrary to all expectation, under the administration of a mixture containing quinine and hydrochloric acid, a mouth-wash of chlorate of potash, and aromatic baths; and he has since grown up a strong boy.

I have not hitherto had an opportunity of observing a second case of this kind, *i.e.* of jaundice combined with the hæmorrhagic eruption just described. There could be no question that it was not a case of malignant jaundice following puerperal infection

of the umbilical wound; still, neither did the clinical picture entirely correspond with that of ordinary icterus neonatorum. From the latter we must also distinguish that form of jaundice which occurs in rare cases in new-born infants in consequence of an obliteration or congenital want of the excretory bile-ducts, and which, in every respect, is to be placed along with the obstructive jaundice of older people. In the whole course of my practice I have come across at most three cases of this kind, and of these only two came to a post-mortem.

A child of 4 months old, brought in summer 1850 to the University Clinique, had suffered from jaundice since birth, with perfectly dry, almost milk-white, evacuations and dark bilious urine. One could feel the left lobe of the liver distinctly in the epigastrium. In spite of all the means used, not only did the jaundice persist, but the colour of the skin became steadily greener, and the child died in a state of extreme emaciation five weeks after it was first seen. After death we found the liver smaller by at least a third than it usually is at this age; the lobes were of equal size, the left flattened, and reaching right into the left hypochondrium, of a moderately firm consistence, and through and through of an olive-green colour. The gall-bladder was present in a rudimentary condition, but there was no trace of the bile-ducts to be found, and the opening of the ductus choledochus into the duodenum could not be discovered.

You find in this case therefore, not only during life, but also after death, all the appearances of a jaundice caused by obstruction within the liver to the outflow of bile, and especially the familiar diminution in size of the formerly enlarged organ, due to the retrogressive metamorphosis and atrophy of the liver cells. In such cases any treatment is of course out of the question. I shall return afterwards to a case which was apparently the result of a syphilitic periphlebitis.[1]

In contrast to the mild character of icterus neonatorum, which we can scarcely regard as a disease, the first period after birth presents one of the most violent and fatal of all known maladies—

Trismus, or Tetanus neonatorum.

Although the phenomena of this disease are essentially the same as those of tetanus in adults, still they are more or less

[1] Compare E. Gessner, "Ueber congenitalen Verschluss der grossen Gallengänge," *Inaug. Diss.:* Halle, 1886.

modified by the child's tender age. Most frequently it begins between the 5th and 9th days after birth, but I have once or twice seen the earliest symptoms appear on the 20th day. Usually the first symptom which strikes those round about the child is the difficulty or impossibility of sucking; every attempt to seize the nipple or bottle with its mouth calls forth a rigid contraction of the muscles of mastication and of the orbicularis oris, which renders sucking impossible. The other facial muscles also take part in the contraction, and the countenance is then disfigured to an extreme degree. At first these spasms occur only paroxysmally, whenever an attempt at sucking is made, and it is still possible to give the child milk with a teaspoon, but after a few hours the symptoms usually become rapidly worse; the fits I have described now occur spontaneously also, without evident cause; in them the forehead gets puckered into furrows, the eyebrows are wrinkled up, the lids fast shut, the lips drawn into a point like a proboscis, and surrounded by radiating folds. Soon the pharyngeal muscles participate, and their contraction interferes with the swallowing of milk poured into the mouth; the attempt to swallow is often accompanied by symptoms of choking with cyanosed visage and arrest of the respiration, which in the intervals between the paroxysms is usually extremely rapid and shallow. If one endeavours to pass a finger into the mouth, the jaws are found to be firmly clenched together owing to rigid contraction of the masseter and temporal muscles; any attempt to overcome this resistance is invariably followed by the accession or aggravation of the convulsive seizures. It is only in the rarest cases, however, that you find this limited to the groups of muscles already mentioned; usually there is rigidity of the muscles of the neck and back also, with backward retraction of the head and complete immobility of the spinal column, which last one sees most strikingly on grasping the child's body about the middle and supporting it horizontally. The muscles of the upper and lower extremities also often participate more or less. The arms and legs are extended, their muscles hard and unyielding like those of the abdomen, and it is scarcely possible to flex them by force. All these spastic symptoms show, it is true, intermissions or at least remissions, but they become more lasting as the disease progresses, and often, though by no means always, are occasioned or considerably intensified by touching the

patient, or by attempts to administer nourishment or enemata. Short convulsive seizures which shoot through the trunk and limbs like electric shocks are also not uncommon.

Under these circumstances nourishment by the breast or bottle becomes an impossibility. I have only in one case seen a child taking the bottle during the height of the disease and certainly not sufficiently.

The complete interference with the nourishment, combined with the contraction of the muscles above described, which is unquestionably painful, must bring about a rapid sinking. The temperature (the examination of which is important) either remains normal or shows only a moderate rise to 101° or 102° F., and in many cases this will be little if at all exceeded in the whole course of the disease. Sometimes, however, the temperature rises pretty quickly and finally reaches from 104° to 106° F. or higher, as in many cases of tetanus in adults. The disease generally exhibits a steadily progressive character, but a deceptive appearance of improvement in the symptoms occurs occasionally, either spontaneously or as the result of treatment, but it is wont to be followed, mostly after a very short time, by fresh exacerbations of the muscular contractions. Finally the child sinks into a state of stupor, the extremely rapid pulse becomes imperceptible, and death follows either from exhaustion or from asphyxia due to tetanic contractions of the inspiratory muscles. The disease lasts from 24 or 36 hours to 9 days according to the severity of the case.

By far the largest proportion of new-born infants attacked by trismus perish; you must therefore from the beginning give a bad prognosis. Complete recovery however is by no means impossible, and I have myself met with one or two cases of it. Just as in adults, so here, the cases in which the temperature is high apparently justify from the first a specially bad prognosis; and even where the temperature is low (99° or 100° F. during the whole course) a fatal termination is common enough. In cases which end favourably the improvement is always quite gradual, never sudden; the rigidity of the muscles and the convulsive exacerbations disappear slowly; and in two cases which I myself observed one could after three weeks still make out a rigidity of the muscles of the extremities, which offered both to extension and flexion an almost springlike resistance. In a third

child there was still in the beginning of the fourth week a slight stiffness of the back and closure of the jaws on the introduction of a finger into the mouth ; at the same time the child took the bottle well. None of these cases, however, were to be accounted very bad ones, even during their acmè the temperature was only a few points above the normal, and one of the children who was treated as an out-patient was able after the first two days to have milk administered to it by means of a tea-spoon forced between its jaws.

In new-born infants, as in adults, post-mortem examination yields nothing characteristic. The old statements about blood being found in the spinal canal have long ago been disproved; and where this was really found it must be regarded as having been the result of venous obstruction brought about by the arrest of the respiration, and not as the cause of the disease. You will not rarely meet with little hæmorrhages due to the same cause between the meninges of the brain and on the serous membranes. The central organs themselves appear normal apart from a more or less marked venous hyperæmia and its results (œdema, miliary hæmorrhages). That in tetanus we have to do with a heightened reflex activity of the spinal cord is beyond doubt, although the production and aggravation of the spastic symptoms by every stimulation of the sensory nerves (feeling the pulse, touching, &c.) is not equally well marked in all cases. Further, in trismus neonatorum this symptom is sometimes more pronounced than at others, and is the more easily understood because at this age even in health the reflex impulses predominate. According to Soltmann's experiments, performed upon new-born animals during the first period of life, all their movements as a rule take place reflexly without the influence of the will, and all the centres in the brain and spinal cord controlling reflex action are still wanting. In this way then we can explain the extreme frequency of reflex spasms in new-born children, in comparison with those of a later age, but not the cause which gives to this uncontrolled reflex action the peculiar and dangerous form of trismus. The frequency of this form is inconsistent with Soltmann's idea that the excitability of the peripheral nerves in these very first weeks of life is less than in adults, for it is very probable that the exciting cause of tetanus comes along these nerves. I regard tetanus in new-born children, as in adults, as the result

of various influences which cause irritation either over the area of distribution of a single nerve or the whole sum of sensory fibres, and, a predisposition being present, produce the disease by rapidly transmitting this to the spinal cord. As such I should name—

1. Injuries (T. traumaticus)—at this age by far most commonly affecting the navel, separation of the umbilical cord, omphalitis; rarely other injuries, *e.g.* the rite of circumcision. In two cases which occurred in my own practice what remained of the umbilical cord was forcibly torn off on the morning after birth, and there resulted an umbilical sore surrounded by an inflamed area. I should add that in this connection I only attach importance to real injuries and not to the "inflammation of the umbilical arteries" which Schöller laid stress upon some years ago; this is nothing but thrombosis in them which has partially broken down into detritus, and has nothing whatever to do with trismus.

2. The action of changes of temperature on the skin of the new-born infant—on the one hand taking it out into the cold air too soon (*e.g.* to be christened); on the other, too hot baths. Thus we have the cases which now and then have been occasioned by midwives who could not appreciate differences in temperature and prepared baths for the infant without the aid of a thermometer. This happened for example in Elbing where trismus was for years endemic in the practice of the busiest midwife, and hundreds of new-born children died of it. At last it was discovered that the midwife was unable to distinguish between a bath at a temperature of 106° F. and one at 95° F.; a bath thermometer was used, and this "epidemic" of trismus was brought to an end.[1] We can easily understand that many other sources of irritation may still remain undiscovered and that the disease may thus originate apparently without cause. Perhaps its origin in vitiated air (*e.g.* in Iceland, where it was caused by exhalations from whale-blubber, and in the Maternity Hospital in Dublin, from which good ventilation has now banished it) as well as its occurrence as an epidemic in some of the West Indian Islands, is to be explained by one of the causes named. The presence of albumen in the urine of new-born infants has been alluded to above (p. 18); and I should add

[1] Bohn, *Jahrb. f. Kinderheilk.*, 1876, ix., S. 307.

that after death one not very rarely finds in them the appearance of parenchymatous nephritis. Although in one case symptoms were observed (Ingerslev[1]) which corresponded on the whole with those of trismus neonatorum, and the urine collected contained a large quantity of albumen and numerous casts, partly hyaline, partly granular, and partly studded with fatty epithelium, yet at the post-mortem there was more the appearance of engorged kidneys with capillary hæmorrhages than of parenchymatous nephritis; this is readily intelligible in the state of venous engorgement, which in tetanus may affect all the organs. We cannot therefore at present maintain that uræmic processes manifest themselves at this age under the form of trismus.

In my opinion therefore trismus neonatorum is, just as one might say of epilepsy, a form of convulsion which is a unity only so far as its manifestations are concerned, and which may be caused by a number of different sources of irritation. To discover these causes in each individual case may certainly be difficult and only possible under favourable circumstances, *e.g.* when due to wounds, umbilical sores, changes of temperature. The etiology of trismus would gain considerably in certainty should the view expressed by Beumer[2] be fully confirmed—namely, that here, as in the traumatic tetanus of adults, we have to do with an infection by "tetanus-bacilli" which gain entrance to the body by the umbilical wound. Dirty hands or dressings are supposed to carry these bacteria "which are apparently so widely distributed" to the umbilical wound. As a matter of fact the results of Beumer's inoculation experiments have since been confirmed by Peiper.[3] Should this view be correct, the causes which I have alleged (traumatic and thermic) will only come into operation if the specific bacilli and their products (ptomaines) happen to be present. Even then the treatment will always have to contend with the greatest difficulties; for we know that this same disease, whether of traumatic, rheumatic, or toxic origin, even when it attacks older people who are better able to contend against it, is one of the most dangerous that we know of.

The only remedy, under which I have seen two cases of trismus

[1] *Oesterr. Jahrb. f. Pädiatrik*, viii., S. 173.
[2] *Berl. klin. Wochenschr.*, 1887, No. 31.
[3] *Centralbl. f. klin. Med.*, 1887, No. 42.

neonatorum recover, is chloral, which I gave in doses of gr. $\frac{3}{4}$—i every hour. If this medicine cannot be swallowed, one must give it in enemata—gr. iss. every hour. In other cases the same treatment gave no result, nor did the inhalation of chloroform, which caused at most only a momentary relaxation of the clenched jaws. From opium (tinct. opii., gtt. $\frac{2}{3}$ every two hours) I have observed only a passing effect, lasting as long as the narcosis caused by it. Whenever that ceased, the tetanus recommenced. From extract of physostigma, which I have used hypodermically, a $\frac{1}{2}$ per cent. solution in doses of gr. $\frac{1}{15}$, three or four times a day, I have seen just as little result; while others (Monti) say that they have seen some good from this very drug. Considering the extremely unfavourable results of every method of treatment in this disease, we must insist all the more strongly on careful prophylaxis; that is, on avoiding as completely as possible all injuries, and everything that can have an irritating influence upon the cutaneous nervous system (cold air, too hot baths).

Besides trismus, other convulsive seizures localised and general occur in new-born infants, corresponding in every respect to attacks of eclampsia in older children. I mention this because some, on the strength of certain of Virchow's observations, are inclined to make the conditions which he described answerable for those cerebral symptoms. Under the title "Encephalitis and Myelitis interstitialis" he described[1] a morbid condition of the brain and spinal cord which he had observed in children who were still-born or had died soon after birth from the influence of infectious diseases or syphilis, or even without evident cause. This consisted essentially in a proliferation and fatty infiltration of the neuralgia cells, which could sometimes be recognised by the naked eye as little soft spots of a yellow or pinkish colour. Hayem and Parrot confirmed the occurrence of these conditions, though not their directly inflammatory significance; and Jastrowitz,[2] in a work based on 65 cases, explained them as due to a physiological fatty degeneration found in every fœtus, especially in certain parts of the centre of the brain, and in the posterior columns of

[1] *Archiv*, 1867, Bd. 38, S. 129; 1868, Bd. 44, S. 472. *Klin. Wochenschr.*, 1883, Oct., Nov.

Arch. f. Psych. u. Nervenk., 1872, ii., and iii.

the medulla, which reaches its maximum about the 7th month of intra-uterine life, then diminishes, and soon after birth disappears. He regards this fatty degeneration as morbid, only when it persists beyond the normal time or implicates other portions of the brain than the white substance of the centrum ovale, *e.g.* the great ganglia, the grey substance of the convolutions, or the nuclei of the cranial or spinal nerves. Concerning the etiological conditions of this imperfect reabsorption of fat we are still in the dark. The whole question in spite of repeated investigations[1] remains as yet unsolved. These conditions have at present only an anatomical interest, since their relation to definite clinical symptoms in new-born children is not yet decided. Further, a form of keratitis ulcerosa[2] which occurs between the 2nd and 5th months, and is described as the result of an "Encephalitis" of this kind, is by no means established as such.

The same may be said of certain naked-eye changes which one finds sometimes within the cranial cavity in new-born children—œdema and hyperæmia of the pia mater and little ecchymoses in it. When we compare clinically the cases in which these post-mortem appearances are observed, we find no characteristic symptoms at all, but often a general clinical picture which we may describe as that of "congenital debility." A more or less extreme degree of atrophy, a greyish yellow tint of the skin, extreme weakness and apathy, piteous whining instead of the normal cry, quick shallow breathing, a cyanotic tinge of the extremities and a subnormal temperature:—such are the symptoms which these unhappy beings are wont to exhibit soon after birth, and under which the majority of them succumb in the first days or weeks of life, unless they have the good fortune to be placed in particularly favourable circumstances. The lot of most of them, alas! is to be badly nursed or to be sent to a children's hospital where what they need most, human milk and fresh air, cannot be got. My department in the Charité can show, all the year round, a number of such children who in spite of all our efforts die of collapse from steadily increasing heart-failure, with or without convulsions. The frequent occurrence of œdema, hyperæmia and little blood extravasations in the pia

[1] Kramer, "Ueber das Vorkommen von Körnchenzellen im Gehirn Neugeborener," *Dissert.:* Berlin, 1885.

[2] Graefe und Hirschberg, *Arch. f. Ophth.*, xii., S. 250, and *Berl. klin. Wochenschr.*, 1868, S. 324.

mater in these cases, is in my opinion only to be regarded as the result of venous obstruction. It is due to the failure of the heart and collapse of the lungs almost always present, and is certainly not an active process; it is not therefore the cause of the final convulsive phenomena. I shall revert to this again when speaking of the so-called "hydrocephaloid" of older children.

Cephalhæmatoma.

Your advice will often be asked by anxious mothers about a swelling on the head of the new-born child which is known by the name of cephalhæmatoma, and consists of an effusion of blood between the bone and pericranium. It appears to be due to the pressure which the skull of the fœtus suffers in passing through the pelvic outlet; and to produce it, the birth does not need to be a specially difficult one. The occurrence of a cephalhæmatoma has also been observed in breech-presentations. In many cases the pressure affects only the scalp and its subcutaneous and subaponeurotic connective tissue, and then all that results is a sero-sanguinolent effusion in them forming a moderate-sized doughy tumour, which is known to you from obstetrics as the caput succedaneum. If, however, the pressure is exerted more deeply or for a longer time, the pericranium itself is implicated and the bleeding now takes place between it and the corresponding cranial bone. As a rule this is one of the parietal bones, especially the right, which in the usual presentation of the child is the one most frequently exposed to pressure during birth. The blood which flows from the torn vessels gradually raises the pericranium from the bone and forms a fluctuating swelling on it which does not reach its maximum all at once, but increases in size gradually (as the bleeding goes on slowly) and usually does not come to a standstill till the third day. Not infrequently the swelling by that time covers the whole parietal bone; it does not reach beyond, because the sutures of the cranial bones to which the pericranium is especially firmly attached set a limit to its further extension. I have never myself seen a cephalhæmatoma on both sides, but examples are not wanting in medical literature.

On examination you find a more or less tense, distinctly

fluctuating tumour usually over the right, more rarely over the left parietal bone, or over other cranial bones. The skin covering it is of a normal colour, less commonly it has a bluish tinge shining through, or it may even be itself infiltrated with blood. Even when it is very tense you will generally be able by sharp pressure with the point of the finger to feel the subjacent bones through it; although in the first few days a hard, somewhat projecting border forms round about the tumour which is apt to be mistaken for the edge of an aperture in the cranium, especially when the swelling is small in size. The cephalhæmatoma hardly seems to cause even discomfort to the infant. Only when one presses on it does the child begin to cry, and that is easily explained by the tenderness of the tightly-stretched soft parts. Moreover the general health remains undisturbed, and the reabsorption of the effused blood proceeds rapidly as a rule. Absorption is all the more rapid because the blood in these swellings may remain at least partly fluid for a very long time (more than four weeks). After one week the swelling considerably diminishes and the bone can be distinctly felt through it, and in the course of two to four weeks, according to the size of the tumour, it is completely absorbed. During this period of recovery the above-mentioned hard ring round the tumour continues to be perceptible, only it gets smaller in size simultaneously with the diminution of the latter. In many cases where the process of reabsorption occupies a longer time, you experience when you press on the soft parts, which are approaching nearer and nearer to the subjacent bone and becoming applied to it, a feeling of crackling, as if you were pressing on parchment; at last the reabsorption is at an end and the pericranium is once more firmly adherent to the bone. The cause of this hard ring at the base of the cephalhæmatoma is the process of bone formation which still keeps going on on the inner surface of the separated periosteum, at first taking place most freely where the periosteum and bone border on one another, that is round the base of the tumour. At a later stage little plates of bone are also formed on the inner surface of the raised periosteum which cause the above-mentioned sensation of crackling to the person examining, and form a sort of shell over the remainder of the effused blood.[1]

[1] Virchow, *Geschwülste*, 1, S. 140.

Cephalhæmatomata of a quite similar description to those in new-born children may also occur in later life from traumatic causes. I have observed such in children of 2, 4 and 8 years of age as the result of a violent blow against a lamp-post, or of a fall on the back of the head, occasionally also without any evident cause. The swelling was situated either on the parietal or occipital bone, or covered the entire surface of the latter. Here also the tumour was observed gradually to increase in size; and in the case of a boy 8 years old a week after the fall, when the cephalhæmatoma was fully developed, an additional hæmorrhage accompanied by great swelling took place into the subcutaneous connective tissue of the forehead and eyelids. A week later nothing remained of this but a greenish yellow discolouration, while the immense cephalhæmatoma on the occipital bone after lasting 14 days had been reabsorbed, and only a flat swelling scarcely as big as a shilling was left, surrounded by a hard ring of bone.

According to my experience the treatment should be purely expectant. Formerly I used frequently to make incisions, evacuate the blood, and then at once apply pressure with strips of plaster. The result of this was usually good, still I was not always able to prevent suppuration; and I have repeatedly met with cases which had been incised by other practitioners and which presented gaping suppurating sores. Although now this danger is materially lessened by antiseptic dressing, yet I see no reason for opening a swelling which I have always seen disappear completely by absorption in a few weeks. I should therefore advise you only to incise if the tumour suppurates spontaneously and threatens to burst; an event which is very rare and which I have never myself observed. Under all circumstances, however, it is well to protect the tumour as much as possible against external injuries by a soft covering (cotton wool).

Only by the utterly inexperienced could a cephalhæmatoma be mistaken for a congenital encephalocele—the protrusion of the brain or cerebral membranes distended with fluid (meningocele) through a congenital aperture in the cranial bones. This mistake is rendered possible by the apparent or real fluctuation in such a tumour and the hard border of the bony aperture which can be felt round about it. The diagnosis is based on

the fact, that the encephalocele generally occurs at a place which is almost never affected by cephalhæmatoma in new-born infants, namely on the occipital bone, much seldomer on the glabella or parietal bone. The encephalocele is as a rule smaller,[1] and when the hand is laid on it one can make out a pulsation proceeding from the cranial contents, as well as a rising and falling with the respiration, which never occurs in cephalhæmatoma. In these also by a sharp pressure with the finger we can almost always make out the bones lying under the fluid, while we can never do so in encephalocele and meningocele. The same holds good of the so-called spurious meningocele in which penetrating fissures of the cranial bones, usually fractures, have arisen (either before or after birth), and cerebro-spinal fluid has passed out through them under the pericranium. In doubtful cases—and these must indeed be extremely rare—we may make certain by an exploratory puncture.

Hæmatoma of the Sterno-mastoid.

You will not very rarely have children brought to you in the first weeks of life who have a hard roundish or elongated uneven swelling on one or other side of the neck, very rarely on both sides, corresponding to the anterior division of the sterno-mastoid muscle. The size of it varies, being sometimes that of a pigeon's egg; often however it is larger and of an elongated form—so that I have occasionally found a great part of the anterior border of the muscle hard and knotted, with band-like processes spreading into neighbouring muscles. Sometimes there occur two or three separate indurations in the border of the muscle. As a general rule the upper half of the muscle is much more frequently affected than the lower. Occasionally I have found almost the whole anterior half of it of a really cartilaginous hardness throughout its entire extent. The right sterno-mastoid is by far more frequently affected than the other, for out of 30 cases recorded in my journals I find 23 of the right side and only 7 of the left.

The youngest child I have seen with an affection of this kind

[1] Very large meningoceles (*e.g.* the size of a child's head) are generally pedunculated and somewhat transparent when held against the light (*cf.* a case of this kind which I observed—*Charité-Annalen*, Bd. i., S. 569).

was three weeks old, the majority were 4—6 weeks, but 4 had reached the ages of 3, 5, and 12 months respectively. In no case did it cause any pain; in most it was discovered quite accidentally while the child was being washed. Less frequently the mother's attention was first attracted by the fact that the child's head was not held straight when it was lying, but had always an inclination to one side, usually the right. This position of the head, however, was by no means always present, and it has seemed to me to be less common the younger the child was.

The nature of this swelling of the sterno-mastoid muscle becomes clear to us when we find that almost all the children affected by it have had an abnormal presentation at birth, which either delayed the labour or rendered artificial assistance necessary. Out of 30 cases which I have observed, there had been a breech presentation in 20, and some force had been used in bringing the labour to a conclusion. Of the remaining 10 cases, 7 were born with the normal presentation, but in all it was expressly stated that the labour was unusually prolonged because the child's shoulders would not engage, and that strong traction was required. In one case the child was born asphyxiated and had been violently swung about in the attempt to resuscitate it. No one, therefore, can doubt that the cause is to be sought in a forcible stretching and partial laceration of the muscle occurring during or after birth, and that the disease consists in an effusion of blood into the muscular tissue (hæmatoma), followed by myositis, which forms a capsule round it and leads to the formation of a fibrous induration; this is confirmed by post-mortem examinations (Skrzeczka, Taylor). The use of force in such circumstances occasionally has other effects. Thus in one of my cases there was simultaneously a fracture of the upper arm, and in another, in which the presenting part (nates) had exhibited an extensive ecchymosis immediately after birth, an apparently paralytic weakness of the right lower extremity.[1]

As far as my observation goes, the swelling always takes a

[1] In one new-born child I found a gangrenous cavity, about the size of a walnut, on the left side of the neck, just under the mastoid process, which was caused by the separation of a black slough. This had evidently been caused by pressure within the pelvis during a prolonged labour, occasioning a hæmatoma and ending in necrosis. In this case the muscle was not implicated, and only the superjacent tissues (skin, connective tissue and fascia) were affected.

favourable course, gradually diminishing, and at last leaving an induration of a varying size in the muscle which scarcely if at all interferes with its functions. I have never myself seen suppuration, but it cannot be denied that a serious disturbance of function may arise from it and I have every reason to assume this as the original cause in a case of torticollis in a girl six years of age, which dated from the first weeks of life. Also the wry-neck of a boy of seven, which had already been operated on with partial success, three years before, was due to a hæmatoma of this kind, resulting from a breech presentation; and the retracted scar could still be distinctly recognised in the anterior belly of the muscle. Unfortunately, almost all my cases of hæmatoma were subsequently lost sight of, and I saw few of them a second time. In the case of a child of six weeks old, first examined on 31st March, 1873, the swelling could be felt distinctly—although it was considerably smaller—on 25th October. The natural cure by the formation of a fibrous induration makes any treatment superfluous. If you like to order the inunction of iodide of potash ointment over the tumour, you may thereby perhaps gratify the anxious mother and—especially among poor patients—ensure to yourself further observation of the case. But no one will anticipate any benefit from this treatment.

Swelling of the Mammary Glands.

In very many new-born infants you observe swelling of the mammary glands during the first weeks of life. In the position of one or both mammæ you find a tolerably hard swelling, globular or bluntly-conical in shape, about the size of a pigeon's egg or small walnut, and of the natural colour of the skin. Pressure on this seems to be painful, as it usually makes the child cry. Now, if you take hold of the base of the swelling with two fingers and compress it laterally with moderate firmness, you see a whitish, opalescent drop rising out of the shallow funnel-shaped hollow which exists at its summit; and this shows under the microscope fat-globules and larger conglomerations made up of them.

To understand how these swellings are formed, one must remember that all new-born children, boys as well as girls, have a secretion from their breasts resembling milk, which begins

about four days after birth. This is usually accompanied by a slight swelling of the mamma, goes on increasing till the ninth day, then gradually decreases till, about twenty days after birth, it is no longer perceptible. I have, however, in one child found both breasts, four weeks after birth, still much swollen, nodular and containing milk. Natalis Guillot[1], by squeezing the mammary glands, obtained from a child about fifteen minims of whitish fluid which, under the microscope, presented all the characters of colostrum. According to Sinéty's[2] investigations on making a section of the breasts of new-born children, one finds milk-canals near the surface which are filled with epithelium. These become wider as they pass inwards, divide, and form cavities containing a fluid resembling colostrum. This process is said to begin during fœtal life, to reach its acmé between the fourth and tenth day after birth (in virtue of a stronger development of the above-mentioned milk canals and cavities), to be aggravated by squeezing the breasts, and, in rare cases, to last possibly as long as six to eight weeks. Epstein[3] connects this with the active cell-formation and desquamation of the epithelium which take place during fœtal life in other parts regarded as invaginations of the skin, especially in the sebaceous glands, and which appear in the form of vernix caseosa, seborrhæa, or milium. Moreover, according to Guillot, this secretion of milk is observed only in strong healthy children, and not in those that are weak and sickly from birth.

Now in new-born infants, as in women, the secreting breast may become the seat of morbid processes. One need not, like Bouchut, assume in such a case a "puerperal" condition of the child, when there is absolutely nothing else indicating it to be observed. On the contrary, the purely local process may become aggravated to such a degree of inflammation as to bring about firstly a greater swelling of the glands, and then the formation of abscesses in them. In this case the little swelling becomes red, very tender, and fluctuating; and a quantity of pus is evacuated either spontaneously or by incision. Since I have seen this happen two or three times from the swelling having been squeezed too hard or very often—which midwives especially

[1] *Arch. de méd.*, 1853.
[2] *Gaz. méd.*, No. 17, 1875.
[3] *Centralzeitung f. Kinderkrank.*, Bd. ii., No. 4, S. 53.

are apt to do—I always guard against any maltreatment, and prefer to have it simply covered with wadding soaked in oil. Under this treatment very considerable tumours disappear with surprising rapidity. Should redness or suppuration follow notwithstanding, you may favour the evacuation of the abscess by warm poultices and incisions. Guillot observed three cases ending fatally from complications; and Bouchut[1] saw one case with a considerable undermining of the pectoral muscle, which ended fatally. I have myself only once met with an unfavourable termination—burrowing of matter and gangrene of the skin over the pectoral muscle in a sickly, wasted child. Strictly circumscribed suppuration in the gland may also occur, as was shown by the case of a child from the upper part of whose mamma (which was only slightly swollen) a few drops of yellow pus oozed on compression by the fingers, while from the lower part there trickled white milk. In some cases also I have seen the two mammæ affected in succession.

Erysipelas Neonatorum.

There used to be many who were inclined to deny that the erysipelas of new-born infants was in any sense a distinct disease, and preferred to regard it always as merely a symptom of the condition described under the name of "puerperal infection" of new-born children.[2] I have not myself any very extensive experience of this condition, which for obvious reasons occurs most frequently in lying-in hospitals and foundling institutions. This much, however, I think I am justified in giving as my conclusion:—that erysipelas in new-born infants is by no means always to be regarded as a symptom of puerperal infection. In adults, erysipelas occurs sometimes as a symptom of serious general diseases—pyæmia, septicæmia, typhoid, &c.,—sometimes begins as a local affection proceeding from a wound and of parasitic nature as proved by recent research (Fehleisen). In the same way we must, I think, distinguish two forms in new-born children. The first and most serious of these is connected, without doubt, with the already mentioned puerperal infection of infants, the various phenomena of which come to be joined to those of erysipelas—rapid collapse, very high temperature

[1] *Traité prat. des. maladies des nouveaux-nés*, &c., 5 éd., 1867, p. 719.
[2] v. Hecker, *Archiv f. Gynæcol.*, Bd. x., H. 3, S. 533, 1876.

(to 106·° F.), jaundice, vomiting and purging, inflammations of various serous membranes (pleura, peritoneum, joints), convulsions and coma. This is the form of erysipelas which occurs in the children of women who are suffering from sporadic puerperal fever or have died of it (of which I have myself seen several examples). It attacks also on a more extended scale the new-born infants during epidemics of puerperal fever, and in the lying-in hospitals where this disease prevails. The second form has nothing to do with puerperal infection; at least no connection can be traced with disease of this kind in the mother. At some place or other on the body there is an abrasion, it may be very trifling, which becomes the starting-point of the disease and a true erysipelas traumaticum is developed with the well-known tendency to spread.

As at certain times sores of the most diverse kinds are apt to give rise to erysipelas—especially in hospitals—while at others this seldom or never happens; so, the wounds one finds on the bodies of new-born children, when exposed to foul air, uncleanliness and infectious influences—which are certainly not of a puerperal nature—are very apt to give rise to an attack of the same disease. Hence, also, one meets with the second form of it far seldomer in private practice where the surroundings are favourable than among the poor. But even with the best nursing and the most favourable conditions of life, erysipelas neonatorum may develope. As an example of this I shall only mention the case of a Jewish boy in a very well-to-do family, in whom I saw erysipelas starting from the penis after circumcision. It gradually spread over the whole body, produced, after a fortnight, a circumscribed patch of gangrene on the scrotum, then an immense abscess on the back; and finally brought about a fatal result with general collapse, jaundice, and symptoms of peritonitis. In this case a puerperal source of infection was out of the question.

The traumatic form also of erysipelas neonatorum may begin during the first few days after birth. Sometimes it occurs much later. Thus I have seen it begin on the fifteenth day after birth in a child who had had a fall, whose mother was not quite sixteen years old. Very often a raw surface at the umbilicus first gives rise to its development; almost as often, however, the genitals form the starting-point; the anus less frequently. In these cases we have to do less with actual wounds (except in

the case of circumcision) than with those red excoriations which form in this region on the parts of the skin which have become the seat of intertrigo, from the contact of the urine and fæces, and want of cleanliness. Erysipelas may also originate in other regions of the skin, if only abrasions of it are present; but this is far less common. You will therefore most frequently find erysipelas commencing at the umbilicus, or lower down in the pubic region at the root of the penis, as a more or less bright-red flush spreading over the skin, and a tolerably resistant swelling which is bounded by sharply defined borders, is raised a little above the surrounding healthy skin, and feels hot to the touch. Pressure, which momentarily lessens the redness but does not make it quite disappear, evidently pains the child. It is rare to have the process limiting itself to the areas of skin originally affected. Almost always the raised margins are pushed gradually onward in different directions; sometimes simultaneously on all sides, oftener more towards one side, in which case the spread of the disease may be quite irregular. Thus, for example, it often happens that it spreads mainly in a downward direction, the erysipelatous rash becoming diffused over the thighs, then over the legs down to the feet; while at first it does not pass upwards beyond the level of the umbilicus. But also in these cases we not uncommonly see the erysipelas beginning suddenly to spread upwards from the anus, and thence over the nates and back till it reaches the upper half of the body. In this way the process may be arrested in all directions and come to an end; but often it spreads over the whole surface of the skin, even over the face and scalp. Wherever the erysipelas makes its appearance, the skin is bright or dark-red, often glazed, œdematous and firm, sometimes of a board-like hardness, so that it scarcely pits at all on pressure with the finger. On the upper and lower extremities, the hard infiltration of the skin sometimes increases to such an extent that in a few cases I have found it scarcely possible to move them at the joints. In general, however, the redness and tension of the skin do not occur to such a high degree on the parts attacked at a later stage as on those first affected; and at the same time the raised border becomes gradually less marked. In many places it may be accompanied by an eruption of vesicles, or of larger bullæ, filled with yellowish serum, as in the erysipelas bullosum of older individuals.

The œdematous swelling of the skin and underlying tissue is most marked on the laxer parts, so that the penis, scrotum, vulva, eyelids, hands and feet, appear not only reddened but considerably swollen. Lines drawn on the red skin with the finger-nail or any blunt object, remain visible for a long time as white streaks; in one of my cases they were visible for more than a quarter of an hour. As in every case of erysipelas migrans, while the redness gradually spreads, the parts first affected become pale; and hence it sometimes happens that the chest and neck as well as the legs are still of a bright red, while the intermediate parts have resumed their normal colour; but this does not protect the latter from being again affected by a retrograde process as it were. Thus, in a child of five weeks I have seen erysipelas, which had affected the whole body almost up to the neck during three weeks, suddenly attack the scrotum a second time. One finds therefore, not unfrequently, in the stage of decline, when the disease has ceased to spread, patches of redness irregularly distributed and no longer continuous but isolated in the form of numerous islands,—partly on the chest, partly on the back or limbs. Between these the skin is of a normal colour, but generally appears more or less œdematous and is covered with fragments of desquamated epithelium or the remains of bullæ. Sometimes after the colour has quite faded there remains behind an œdema spreading over the whole skin, and in cases which are in this stage when they are first brought to the physician doubts may arise concerning the nature of this œdema, which are only solved by the history of the disease and the traces of desquamation of the epidermis which still remain.

During the course of the disease which I have just depicted, a remittent fever is present in all cases, the evening temperature rising to from 102° to 106° F., the morning temperature being about 2° F. lower. The pulse is exceedingly quick (up to 170 and more) and small, the breathing correspondingly rapid and superficial. Many children at an early stage refuse nourishment, especially the breast, even while they will still take milk from a teaspoon. I have seen others take the breast almost as well as when in health. With the arrest of the erysipelas the temperature generally falls rapidly, and the child recovers more or less quickly. On the other hand, should the erysipelas go on spreading further and further over the surface of the skin, the fever

continues, and we are very apt to have complications added, with morbid conditions of the internal organs (especially profuse diarrhœa, pneumonia, and peritonitis), which may put an end to life. The last-named affection I observed in two non-puerperal cases, with very considerable enlargement, tension and tenderness of the abdomen, and frequent vomiting. Probably the inflammatory process spreads from the skin of the abdomen directly to the peritoneum through the umbilicus, which in such cases is generally swollen and sore. Apart from these complications, the high fever may so exhaust the strength of the feeble infant that a fatal termination may ensue with symptoms of collapse. One should never, however, lose heart, since even in cases of extensively spreading erysipelas the children, after weeks of suffering, may get off with their lives and completely recover; others, however, after having made a good recovery from the erysipelas, fall victims to abscesses and gangrene of the skin arising from it. I have observed this result frequently on the scrotum; also on the malleoli, on the back (almost a third of it was in one child covered with an immense accumulation of pus), on the arm, and on the external ear. In the case of smaller patches of gangrene of this nature recovery may take place on their separation.

In a child aged three weeks, erysipelas had spread twelve days before from the umbilicus over the greater part of the body, upwards and downwards. An abscess on the left side of the scrotum remained after this; and when it burst, a deep cavity the size of a florin was left, containing fragments of gangrenous connective tissue. The penis and lower limbs were œdematous, and on the left cheek there was another extensive red infiltration. Under the use of hot poultices, the gangrenous tissue of the scrotum separated in four days; while the erysipelas, of which nothing could any longer be seen on the upper parts of the body, except on the cheek as mentioned above, suddenly spread a second time over the left upper extremity from the elbow to the fingers, and caused a large abscess on the elbow, which I opened a week later. In the end the child recovered completely.

The fact which I have already mentioned above was seen in this case, viz., that after the disease had apparently ceased spreading, certain areas of the skin—in this case the left forearm—was suddenly attacked again, although no continuity could be discovered with an already existing patch and no wound existed on the part newly affected.

Treatment in this dangerous disease is practically powerless.

At its commencement, when the erysipelas is usually limited to the umbilical or pubic region, one may attempt to mitigate the inflammatory process by large fomentations of lead lotion. Internal remedies—except mild purgatives when the bowels are confined—are quite useless. Should the erysipelas begin to spread, no medicine of any kind is capable of limiting its extension any more than in later life. The only thing that can be done is to administer tonic remedies, wine and decoction of bark; but from this I have not seen any really successful result. The matter of chief importance, however, is whether the erysipelas is arrested or continues to spread; in the latter case I have no confidence in any medicine. Injections of carbolic acid (1 to 2 p.c.) into the neighbouring healthy tissues have not in my hands done any good; and on account of the danger of poisoning in the case of such small children, their use seems to me more than questionable. Complications must be treated according to their nature; but when the erysipelas is extensive, they almost always prove fatal at this tender age. Abscesses are to be poulticed, opened as soon as distinct fluctuation is present, and dressed antiseptically.

That I may not have to return again to this condition, I shall take the liberty of adding here a few words on erysipelas in later infancy and in older children. In them also one almost always finds, on careful examination, an excoriation, which may be regarded as the door of entrance for the infecting bacteria and the starting-point of the disease. The sores which I have found most frequently are, that of vaccination, eczema of the scalp, excoriations on the genital organs or arms, such as often occur as the result of erythema intertrigo occurring in these situations, diphtheria of the vulva, large ecthyma pustules; lastly, in older children—especially those who are scrofulous—chronic rhinitis, with excoriations of the nasal mucous membrane. Nothing is more common under the last-mentioned circumstances than a recurrent erysipelas—*i.e.*, one which returns once or even oftener every year. In these cases the erysipelas spreads from the excoriated and scabbed nostrils towards both sides over the cheeks, presenting the appearance of red butterflies' wings; but it does not usually extend further. It is not always possible, however, to discover, even by the most careful search, an excoriation as a starting-point.

Thus in a child of fifteen months I have seen erysipelas starting from the right labium majus, on which there was not the slightest abrasion of the skin. It spread (with smart fever for ten days) with a raised margin over the right lower extremity and descended in paler patches as by leaps,—*i.e.*, with unaffected skin between them—down to the inner ankles, while red islands were also noticed here and there on the skin of the abdomen. The attempt to limit it by painting on collodion failed entirely, and in spite of this it continued to spread for about twenty-two days; after which recovery took place.—In a child of two and a-half years, the erysipelas spread for the third time within seven months from the anus over both nates, with the formation of numerous bullæ, although there was not the slightest abrasion to be seen about the anus.—In a child five months old, the disease seemed to originate from the vagina, which at this tender age was already the seat of fluor albus; extension took place upwards and downwards over the whole body, diarrhœa and pneumonia came on, and death ensued.—I have also observed it in an infant three months old, the result of an incision situated on the right side of the neck. The erysipelas was accompanied by fever (102° to 104° F.) and extended from the wound with a thickened raised border over the right ear, the cheeks and both eyelids, then over the forehead and scalp to the neck, where it came to an end after a week. The treatment consisted of compresses of ice-cold lead lotion, later an ice-bag on the head; internally, quinine (gr. ss. every two hours).

When the erysipelas arises from eczema capitis, it is apt to remain hidden under the hair and the crusts on the scalp; and it reveals itself only by the accompanying fever, the cause of which is not recognised until the erysipelas passes beyond the border of the hair and becomes visible on the forehead or neck, or in the neighbourhood of the ears. In such cases we sometimes have relapses, or rather an extension of the disease on different sides of the eczematous area, *e.g.* first over the forehead and then again towards the temples, each extension being ushered in by a fresh accession of fever.

A boy of four years, with eczema capitis, especially on the left side, admitted into my ward in September, 1873. In the night between the 26th and 27th September, fever with restlessness and headache. On the 27th, continuance of these symptoms without evident local cause. Temp. 103·5° F., ev. 103·8° F. On the following day redness and swelling of the left side of the head passing beyond the border of the hair and extending to the temple; anorexia, thickly-coated tongue. Emetic. Temperature in the evening 105·1° F. During the next few days the erysipelas gradually

diminished in intensity, the redness became less continuous, the tenderness less, the fever diminished, and on 1st October the temperature was 99·5° F. Of the erysipelas there remained visible only a number of vesicles on the margin of the forehead. On the evening of 11th October the fever recommenced, reached on the morning and evening of the following day 104·9° F.; and erysipelas again appeared, starting from the eczema and extending about an inch and a quarter beyond the border of the hair. An ice bag was applied over the reddened area, and the erysipelas ceased spreading and faded on the next day; on the 14th the boy was free from fever, so that we were able, after a few days, to take in hand the treatment of the eczema.

I have repeatedly seen tracheotomy wounds in cases of diphtheria and even other incisions covered with diphtheritic membrane, become the starting-point of erysipelas migrans, which sometimes crept onward till it reached the thorax, and even the epigastrium. In one infant it took its origin from little pricks which I had made in the scrotum with an entomological needle for hydrocele. The scrotum and pubic region soon after became deep-red, hard and swollen, up to the level of the umbilicus. Gangrene and separation of part of the scrotum followed, and the child died in a state of collapse. In many cases erysipelas is developed as the result of vaccination, rarely within the first few days, usually commencing towards the end of the first or second week, or even later, when the vaccination sores are already covered by scabs. Only one arm as a rule is affected, and in that case a spreading of the erysipelas over the body is less to be dreaded than when both arms are attacked. In one case I have seen it spread upwards as far as the auricle, which became swollen, dark-red, and covered with bullæ. In other cases it is impossible to decide whether one has to do with the ordinary areola of the vaccine vesicle extending further than usual, or with erysipelas limited to the upper arms. At certain times, and especially in certain localities, *e.g.* foundling institutions, vaccination-erysipelas may appear as an epidemic; and this is equally likely to occur whether animal or human lymph has been made use of. The treatment of these forms of the disease is the same in all particulars as that already mentioned (p. 48).

Sclerema Neonatorum.

The chief characteristic of this dangerous disease, which occurs almost exclusively in lying-in hospitals and foundling institutions and is rare even in them, is an induration and rigidity which the skin of the infant offers to pressure with the finger over the greater part of the body. In the most severe cases one finds a tense induration as if the body had been frozen; but this is not equally well marked in all places. A more or less considerable fall of temperature accompanies this. The children thus affected are feeble, prematurely born and atrophic, and they invariably die.

Such is a brief and very general statement of the features of a complaint concerning which, till the most recent times, there prevailed a greater confusion of opinion than concerning almost any other disease. Owing to the rarity of the affection and the vague descriptions of it given by most medical writers, there have been widely differing views among practitioners concerning the nature of the disease, and many have no clear conception at all of what is meant by the name sclerema.

The credit of having cleared up this confusion is, in my opinion, pre-eminently due to Parrot, who in his capacity as physician to the Paris Foundling Institution had abundant opportunity for studying the diseases of new-born children. In his work on *Athrepsie*[1] he points out that two morbid conditions entirely distinct from one another—the real induration and the œdema of new-born children—have been hitherto almost universally confounded with one another, and have been included in one vague description. He explains this confusion thus: the real cellular-tissue-induration (sclerema) was first described by Underwood, and this designation soon after, in the year 1781, was transferred by Andry to the œdema of new-born children frequently observed in the Paris Foundling Institution.

(1) The true induration (sclerema) occurs, according to Parrot, exclusively in extremely atrophic (or as he expresses it athreptic) new-born infants, especially where the atrophy affects children of medium bulk immediately after birth. While ordinarily the skin in atrophic children forms broad folds around

[1] *Clinique des nouveaux-nés:* Paris, 1877, p. 116.

their limbs, in these cases it is very tense and smooth; it loses its softness; and finally can no longer be raised up from the subjacent parts, to which it appears to be firmly attached. This alteration in the integuments usually starts from the lower extremities, and spreads upwards over the loins and back; it may, however, in time affect the whole body, even the face. The tension and hardness increase from day to day, and the skin soon acquires the consistence of thick leather. All soft parts then appear as rigid as wood or stone, there is no pitting on pressure; the colour of the skin being usually a dirty yellow, slightly cyanotic on the extremities. Under these circumstances the limbs become immobile, are persistently extended, and only the slight movements of the thorax—perhaps also those of the facial muscles—distinguish the condition from that of cadaveric rigidity. When such a child is grasped by the neck and lifted, it may be held out horizontally like a rigid body, just as in cases of trismus neonatorum; for this disease sclerema may be mistaken, especially in cases where the mouth is shut by the lips and cheeks becoming affected, and sucking is prevented. Even where this is not the case, one is apt to suspect, if not trismus, at least tetanic contractions of all the muscles. I remember especially two such children, who lay for weeks in my ward in a rigid condition and in the highest degree of emaciation, but were still able to suck a little, or to take milk from a spoon. They finally died, the temperature steadily falling to 86° F., in one case even to 83·3° F. At the post-mortem the brain and spinal cord, to which we specially directed our attention, were found absolutely normal; while the integument presented the appearance of sclerema. In some other cases I have found this condition not so generally diffused, but confined to the regions of the calves, the adductors of the thighs, the nates, the cheeks, or even the forearms and upper arms; and in these cases the fall of temperature could be at once verified, not only by applying the hand to the surface, but also by introducing the finger into the mouth. Almost all of my cases were at the same time more or less jaundiced.

The result of Parrot's post-mortems are as follows:—Extreme atrophy with consolidation of the skin, including the rete Malpighi, the cells of which are scarcely visible and form a compact mass with ill-defined contours. In the subcutaneous fat, the

fibres of connective tissue are more numerous than usual and thicker, and the fat itself is considerably diminished; the fat-cells are smaller, and their nuclei can be distinctly seen. Most of the fat-cells are, as in every form of atrophy, almost or entirely deprived of their fat; they are shrivelled into an oval shape, and have a great resemblance to the epidermic-cells of the rete Malpighi. The blood vessels—especially those of the papillæ of the skin—are narrowed to such an extent that one cannot disguish their lumen.

We have, therefore, according to Parrot, a drying-up of the skin with consolidation of its layers, and atrophy of its adipose tissue; and in certain cases observed in my wards a dissection of the skin yielded quite similar results.

(2) The second form, the œdema of newly-born children, presents an entirely different picture. While in sclerema the rigid atrophic skin is firmly attached to the subjacent parts, in œdema exactly the opposite condition obtains; the skin being raised up and distended by œdematous infiltration of the subcutaneous connective tissue. Thus we find all the clinical symptoms of œdema as they appear at any age, especially swelling of the affected part occurring either at one part of the surface only or over the whole body, according to the extent of the œdema. Most frequently the swelling extends from the legs over the lower half of the body, the penis, the scrotum, or the labia majora; and the calves sometimes become affected—earlier than the feet. Not uncommonly the trunk, the upper extremities, and the cheeks are also affected in the same manner; or the swelling may be confined to the dorsum of the hands or feet. All the œdematous parts are swollen, and feel doughy or hard according to the amount of infiltration and consequent tension of the skin. In extreme degrees the affected parts may thus appear very hard and yield little, if at all, to pressure with the finger, just as in extreme degrees of œdema at a later age. The skin is then usually glossy, while in lesser degrees of œdema it appears dull, and for the most part reddish or yellowish, but sometimes mottled and bluish in places. When the skin is very greatly distended, a certain amount of rigidity of the limbs and of the features may occur, interfering with their mobility; this disease, however, never presents the same degree of tetanic rigidity and board-like hardness as sclerema, any more than the consolidation

of the skin equals that of the latter. The body temperature in œdema is usually very low, and in cases which end unfavourably may reach 86° F. or even lower. At the post-mortem one finds an infiltration of the subcutaneous connective tissue with yellowish serous fluid; while the fat seems consolidated to a reddish-yellow or brownish granular mass. Thus, therefore, the anatomical condition also differs fundamentally from that in sclerema; in which, on incising the integuments, not a drop of fluid exudes and only the merest remnant of adipose tissue remains.

In spite of these differences there still exist certain similarities between the two forms; which, however, concern not the skin-affection itself, but the symptoms which accompany it. For example, we have common to both the steadily increasing debility, the smallness and impercebtibility of the pulse, the disappearance of the second sound of the heart, but very specially the fall of temperature of which we have already spoken. I have myself found the temperature in the axilla 88·3° F; others have found it, towards the end, only 71·6° F. External heat produces under these circumstances either a very temporary warmth or none at all. The voice becomes weak and whining; the breathing slow and interrupted, or frequent, superficial and noisy —owing to the presence of pneumonia, which in these circumstances is not as a rule sufficient to raise again the sunken temperature. The children usually lie in a completely apathetic somnolent condition, and may exhibit towards the end local or general convulsions. Many also have more or less serious attacks of diarrhœa which considerably increase the debility. We find after death various complications according to the predominance of this or that symptom; especially bronchitis, pneumonia, more or less extensive pulmonary collapse, pleurisy, various degrees of enteritis, hyperæmia, and small hæmorrhages of the cerebral membranes and other parts. In one of my cases gastritis hæmorrhagica was found. When we consider the age of the little patients we easily understand that a number of other complications may also occur; *e.g.*, jaundice, diseases of the umbilicus, pyæmic and "puerperal" affections, &c. We must now consider the first-mentioned of these conditions, the true sclerema (which was described a hundred years ago by Denman and Underwood, and lately again by Parrot under its proper

heading) as a drying-up of the skin and adipose tissue (occurring as the result of extreme general atrophy?).

Œdema neonatorum may be due to just as many pathological conditions as œdema in later life. In one set of cases, as above mentioned (p. 46), a preceding erysipelas is the cause of the œdema; and it is only thus we can account for the dark-red flushes about the pubes and other regions of the skin, which have been described by some authors, and the purulent infiltrations into the connective tissue and patches of gangrene which have been occasionally found. In another class of cases the œdema is to be regarded as the result of extreme debility of the heart, of fœtal myocarditis[1] or extensive collapse of the lung; following upon which an engorgement of the venous system of the body and transudation of serum take place. Sometimes, also, a nephritic process lies at the root of the œdema, and of this Elsässer[2] has already given examples. The following case came under my own observation :—

> A child of four weeks admitted on 24th March, 1874. Intertrigo in all folds of the skin; well-marked, tense œdema of the face, and all the extremities. Pulse 136. Temperature 97·7° F. The urine, obtained with difficulty, was turbid, albuminous, and extremely scanty. On the 27th, severe dyspnœa and cyanosis. Pulse 144—160. Temp. 101·1° F. The respiratory organs apparently unaffected. Death on the 29th. At the post-mortem there were found parenchymatous nephritis, serous fluid in the pleura, pericardium and peritoneum, little hæmorrhages on the serous membrane covering the heart, consolidation of the left lower lobe.

We see that sclerema and œdema of new-born children have at least one pathogenetic point in common—extreme debility, either congenital, or acquired through causes acting immediately after birth. The extremely low temperature is also connected with the diminished energy of the heart-muscle (which has sometimes been found fattily degenerated) with the disturbed circulation, the weak respiration and atelectasis, and the interference with the necessary tissue-change. And it is this perhaps that occasions that peculiar alteration in the subcutaneous adipose tissue which makes it like solid mutton suet, and which is occasionally met with in children who are not very emaciated. It

[1] To this class belongs, *e.g.*, the case described by Demme as "Sclerom" (19 *Jahresbericht*, S. 75).

[2] *Archiv f. physiol. Heilk.*, xi., 3, 1852.

seems to me therefore by no means necessary to make "induration of the adipose tissue" a special form of disease, as some writers do.

It follows from the pathogenesis just discussed that you will observe sclerema exclusively, and œdema most frequently, in children who were prematurely born, or who from the beginning have been placed under the most unfavourable circumstances (cold, bad air, and wretched nourishment). Hence illegitimate foundlings, particularly during the cold time of the year, are especially liable to this condition; while in private practice, and even in that of a polyclinic, we have far less frequent opportunities of observing it. All other causes mentioned are hypothetical. Owing to the frequent occurrence of certain etiological conditions which may occasion on the one hand sclerema and on the other œdema from engorgement, it is conceivable that cases may occur in which both forms come on simultaneously or at least successively in one and the same individual. This fact has aggravated still more the confusion existing in the minds of most authors. Parrot describes an instructive example of this sort: a new-born child which at first presented a partial œdema continued to emaciate under the influence of atrophy and from the re-absorption of the œdema; and, while the œdematous swelling was still visible on the upper half of the body, true sclerema began on the lower extremities and on the back.

After what has been already said about sclerema, you will be able to judge of its incurability. The children die with symptoms of extreme exhaustion; not always quickly, for I myself had two such cases under observation in my ward for 2—3 weeks. The prognosis of œdema is somewhat more favourable should the cause of it be curable. Thus, for œdema to follow erysipelas appears on the whole to be most favourable; although here also fatal cases are not uncommon. The prognosis in all passive œdemas (which are to be regarded as the expression of extreme cardiac debility, pulmonary collapse, or nephritis) appears to be altogether bad. In all these cases recovery is exceptional, and the treatment must be confined to dietetic and hygienic measures. It is of the very greatest importance to procure a good nurse and to nourish the child, when it is no longer able to suck, with the nurse's milk drawn-off or with good cow's milk. At the same time care must be taken to apply

artificial warmth to its cold body by enveloping it in cotton-wool, rubbing it with warm flannel, by hot bottles, and by warm aromatic baths (camomile and calamus). In the foundling institution at Moscow they use for this purpose a metal cradle with double walls, containing warm water.[1] We may endeavour to maintain the sinking energy of the heart by giving small doses of wine (10 to 15 drops of tokay every hour), but we can scarcely expect very much result from this.

The confusion which till quite recently prevailed in the views concerning "induration and œdema of the cellular tissue" was considerably increased by Bouchut, who connected scleroderma—a disease sometimes observed in adults and older children—with the sclerema of new-born infants. Scleroderma, however, has nothing in common with our sclerema; and is in its whole phenomena and course so different from it that one does not understand how Bouchut's error could have had any supporters. For further information on this disease, I must refer you to works on Dermatology. It has been repeatedly observed in children, and sometimes it has ended favourably.[2]

Pemphigus Neonatorum.

This skin-affection of new-born children presents very many varieties in regard to the number, form, situation, and fulness of the bullæ; still, from a clinical standpoint, I consider it sufficient to distinguish two principal forms: the simple (acute) and the cachectic pemphigus.

The former (pemphigus simplex *sive* acutus) I shall first illustrate to you by a few examples from my own practice.

> The child of a physician, healthy at birth, under my observation in March, 1873, suffered from the ninth day of life from an eruption of pemphigus, which developed successively, but without definite order, on the throat, neck, trunk, and extremities. Only the hands and feet remained free. The bullæ attained the size of a florin, but varied in some parts from only that of a pea to a hazel-nut. They were semi-globular, rather tensely filled with yellowish serum; in some places they were close together, in others separated

[1] Clementowsky, *Oesterr. Jahrb. f. Päd.*, 1873, i., S. 7.

[2] Cruse, *Oesterr. Zeitschr. f. Päd.*, 1876, ii., S. 189.—*Jahrb. f. Kinderheilkunde*, xi., 1877, S. 318.—*Ibid.*, xiii., 1876, S. 36.—Silbermann, *Jahrb. f. Kinderheilk.*, Bd. xv., 1880.

by considerable intervals. The whole number was at least 30 or 40. The intervening skin was of a bright red colour. In the course, of the next few days the contents of the bullæ became turbid; not, however, in all of them. The period of their formation lasted altogether 12 days, during which the child—apart from a moderate tracheal catarrh—was perfectly well; all functions were normal. The temperature of the skin was not taken, but it appeared to be scarcely elevated. After many of the bullæ had either burst or dried up, the formation of new ones ceased on the 12th day; the red skin became pale, and after a week there was nothing left of the whole disease but red patches, skinned over and surrounded by a whitish ring of epidermis. The child has since remained free from any return of the disease.

A child of 14 days, to which I was called on the 8th January, 1874 (birth normal), whose father, twelve years before, had had a chancre but had since remained quite healthy, suddenly, on the 9th day after birth, in the midst of perfect health became affected by pemphigus. The temp. rose slightly and bullæ broke out in succession over all parts of the body, varying from the size of a sixpence to that of a half-crown or larger. They were semi-globular, transparent, yellowish, and more or less tensely filled. The face also did not remain free, and especially on the forehead, the neighbouring bullæ ran together and formed enormous elevations of the epidermis. The skin of the body was extremely red. The soles and palms were unaffected in this case also, except that one bulla formed on the left palm. Along with this the general health was unimpaired. The mucous membrane of the mouth was unaffected, and sucking not interfered with. The formation of bullæ which followed one another in successive crops, lasted about ten days; and recovery followed, as in the first case. So that after several days the situation of the bullæ was indicated by thin dry crusts surrounded by a ring of epidermis, and after these had separated the skin remained reddened for a considerable time. No syphilitic symptoms were ever observed in this child subsequently.

In a child three weeks old seen at the polyclinic in July, 1875, there were numerous bullæ which only reached the size of a sixpence; many remained considerably smaller, scarcely the size of a pea, and on the reddened skin smaller vesicles also appeared here and there. In this case the child felt perfectly well and recovered within a fortnight.

In a boy 14 days old, brought to the polyclinic 18th March, 1873, the whole body was likewise covered with numerous pemphigus bullæ, a number of which had opaque, puriform contents. Particularly large bullæ on the hairy scalp. The inguinal glands somewhat enlarged; health otherwise perfect. Recovery.

I think that these examples will be enough to bring before you clearly the clinical picture and course of the disease in new-

born children; since it is not here my business to enter into a description and explanation of pemphigus in general. You find a rapid development of the eruption in quite healthy children during the second week of life, sometimes as early as the second day, an acute course lasting about fourteen days, and a favourable ending. Only rarely have I observed the mucous membrane of the mouth to be also affected; *e.g.*, in a child two days old there was extensive formation of bullæ on the mucous membrane of the lips and hard palate, the epithelium of which was separated from the bleeding corium in the form of large fragments.

> Unique of its kind was the case of a child of deaf and dumb parents, who, though otherwise well formed, was born with large hæmorrhagic pemphigus-bullæ on the lips and tongue, and a few scattered over the rest of the body. This eruption lasted as long as I had the child under observation (about a year and a half) especially on the tongue and gums, but the intervals between the bullæ became greater, and the child throve very well. This case, then, was one of congenital pemphigus, and it gained further interest from the fact that the father's brother suffered from chronic pemphigus.

More than once I have had occasion to fear from the large number of the bullæ and the redness of the skin—especially at such a tender age—that complications might occur similar to those in extensive burns of the skin; my fears, however, were but seldom justified. Almost all those children recovered. Apart from extreme restlessness and severe itching during the stage of recovery (which one could distinctly recognise from their movements) the children did not seem at all ill. A favourable result is, however, by no means invariable. Chance complications with inflammatory states of the internal organs, sudden collapse as in severe burns (especially in those where the vesicle formation is very extensive, implicating more than a third part of the skin), or a furunculosis following this disease, have been frequently known to cause death. I would specially emphasise as important the fact that in this form the palms and soles either remain quite free or (as I have seen once or twice) present bullæ of an enormous size, which implicate the half of the sole, and are quite different from the flaccid purulent bullæ of pemphigus syphiliticus. In certain cases the skin of the face and head remained free from eruption.

The causal conditions are obscure. The disease is sometimes observed in lying-in hospitals in an endemic form. Thus, we have the endemic occurrence of it observed by Ahlfeld[1] in Leipzig, where within two months it attacked 25 children between the second and fourteenth day after birth of totally different constitutions, who were almost all born of healthy mothers. In these cases also, the palms and soles always remained free, while the fingers were sometimes severely affected. Ahlfeld considers that the disease is of a contagious or at least miasmatic nature; though he is unable to bring forward definite proofs of this. Koch[2] thinks that the contagion is carried by the nurse, because within three months he observed eight cases of pemphigus which all occurred in the practice of the same midwife; and he supplements these observations in a later report[3] in which 23 cases of pemphigus are mentioned from the practice of the same nurse; while among 200 new-born children attended to by other midwives, not a single case occurred. Palmer[4] has had a similar experience. Both authors have also observed the transmission of the eruption to adults, and Koch states that once, "after many negative results," he succeeded, by inoculating with the contents of a bulla, in producing a bulla on his own arm after about 60 hours. Vidal and Blomberg[5] also report a few successful attempts at inoculation. The epidemic in Leipzig and the surrounding districts, described by Moldenhauer[6] (the same which Ahlfeld observed) ceased when those affected were strictly isolated. Nobody, however, has any explanation to offer as to the nature of this contagion. Especially it has not been possible hitherto to demonstrate with certainty the presence of fungi or spores in the contents of the bullæ.[7]

I have not yet in my own practice met with pemphigus neonatorum spreading epidemically, or endemically, in the way described by the authors I have mentioned; and previously also by Hervieux, Abegg, Olshausen, Klemm[8], and others. I have always had to do only with sporadic cases, and most

[1] *Arch. f. Gynäcol.*, v., Bd. i., S. 150. [2] *Jahrb. f. Kinderheilk.*, 1873, S. 413.
[3] *Jahrb. f. Kinderheilk.*, 1875, S. 425.
[4] *Württemb. med. Correspondenzbl.*, No. 40, 1880.
[5] *Gaz. méd.*, No. 29, 1876.—*Jahrb. f. Kinderheilk.*, xxii., S. 248.
[6] *Arch. f. Gynäcol.*, vi., 1874, S. 369.
[7] *Cf.* Ziehl, *Wiener med. Wochenschr.*, 1883, No. 51.
[8] *Oesterr. Jahrb. f. Päd.*, 1872, ii., Anal., S. 205.

certainly in none of them did transmission take place from the child to the nurse or to others. Many others as well have observed this; and have therefore attempted to discover other causes besides infection. Thus, Bohn[1] connects this disease—which usually arises in the second half of the first week of life—with the exfoliation of the epidermis which is wont to begin about the third day, and terminates on an average by the end of the first week. He thinks that during this time any irritation of the skin—for example, that caused by the clothing but especially by baths—may transform the physiological into a pathological process resulting in the formation of bullæ; and justly cautions against judging of the temperature of a bath by the hand without the aid of a thermometer. Bohn refers to a case of pemphigus which had arisen in this manner from baths of 101·7° F., which were supposed by a nurse who had lost the sense of temperature to be 95° F.; and the child rapidly recovered when colder baths were used. Dohrn[2] is of the same opinion, and ascribes to the skin of new-born infants the property of responding to mechanical, chemical, or thermal irritation by an eruption of bullæ. The very exceptional occurrence of transmission to those in attendance, and the few attempts at inoculation which have been apparently successful can therefore scarcely be considered to have established the infectious nature of pemphigus neonatorum.

The treatment is extremely simple. I restrict myself to lukewarm baths, 90·5°—93·7° F., with the addition of bran and gelatine; and I consider it unnecessary to add corrosive sublimate, as is sometimes recommended.

Pemphigus cachecticus is to be distinguished from the simple acute condition by its affecting by preference the regions where the skin is thin—the neck, axilla, groin, and especially the soles of the feet and palms of the hands. The last, as we have seen above, almost always remain unaffected in the simple form. In a child eight days old, the tip of the nose was the seat of such a bulla. The bullæ, which rise on livid spots, are usually only half-filled and flaccid, and seldom exceed the size of a pea or hazel-nut. At the same time their contents appear less clear, often purulent, sometimes tinged with blood. New-born children sometimes bring traces of this eruption with

[1] *Jahrb. f. Kinderheilk.*, 1876, ix., S. 304. [2] *Arch. f. Gynäcol.*, ix., S. 3.

them into the world (which has affected them during fœtal life) in the form of bullæ which have burst and left behind them superficial ulcerations; and this condition usually leads to the supposition of congenital syphilis.

In fact this form of eruption may be held to be one of the earliest symptoms of syphilis, and I myself have records of cases which unquestionably prove this connection.

In a child of 6 months old the formation of bullæ had commenced immediately after birth, and during the last months had increased to such an extent that by this time on many parts of the body, also on the face and the back of the head, fresh bullæ were visible in some parts, excoriations and crusts in others.

The dirty complexion, the chronic rhinitis, and latterly mucous papules round the anus, proved that we had here to do with syphilis.

A girl of 6 days old, admitted 5th April, 1879, very atrophic, exhibited pemphigus bullæ on the whole body, especially numerous on the soles and palms, also under the nails. There was also rhinitis with scabs on the nostrils and lips and enlargement of the axillary and inguinal glands. Post-mortem: Osteochondritis syphilitica universalis; numerous small abscesses in the thymus.

A girl of 14 days, poorly nourished (13th December, 1881). Palms and soles covered with recent opaque bullæ and rounded excoriations, which were surrounded by a ring of epidermis (ruptured bullæ). A few also on the dorsal surface of the hands and feet and on the fingers and toes. There was also rhinitis, and intertrigo near the anus.

A child, 3 weeks old, with coryza, roseola, and pemphigus on the palms and soles, which had arisen 6 days after birth.

Are we then to consider this eruption of bullæ (which differs from the first form by an indefinitely chronic course) as a regular indication of syphilis, or to agree with Caillault[1] that this is only the expression of a deep-rooted cachexia such as one so often observes among the children of the poor, especially those who are wasted and debilitated? I freely admit that I formerly held this view myself, but lately, after having seen a good deal of the disease, I have changed my opinion. All the cases of this form of pemphigus which I have examined during the last few years have been due to syphilis; but owing to the wretched state of the children's general health, it was only in rare cases that specific treatment was able to avert death.

[1] *Traité prat. des maladies de la peau chez les enfants:* Paris, 1859.

Aphthæ of the Palate.

In a former lecture (p. 15) I drew your attention to miliary nodules on the mucous membrane of the palate, which are met with in many new-born children during the first four to six weeks of life. At this age if you examine the throat after depressing the tongue (which is not always easily done) you will very often find, on either side of the arch of the palate, just on a level with the pterygoid process and immediately behind the alveolar arch of the upper jaw (where the bone is visible through the thin mucous membrane), a round or rather more oval yellowish-white patch surrounded by a red border. These patches are usually quite symmetrical, though sometimes rather bigger on one side than the other; occasionally also they have evidently run together and their outline suggests the shape of a breakfast-roll. They seldom exceed $\frac{2}{5}$ of an inch in their greatest diameter. These "plaques," which readily bleed when touched with the spatula, are very often found in perfectly healthy children. They gradually lose their greyish-yellow colour, become red and disappear, leaving no trace behind. But in atrophic and cachectic children I have occasionally seen them increase in size and depth and pass into real ulcerations which may penetrate even to the bone. In such cases one often sees the mucous membrane of the mouth and palate simultaneously covered with thrush; and the children die in consequence of the general condition, or from the occurrence of complications.

These symmetrical "plaques" or "aphthæ" were formerly described, especially by French physicians, but had been forgotten; and Bednar[1] was the first again to draw attention to them. It is especially important to remember that these aphthæ have absolutely nothing to do with syphilis. I should not have mentioned this at all, were I not constantly seeing cases in which physicians unacquainted with the condition had made this diagnosis. I have always been of the opinion (now shared by others) that these aphthæ arise simply mechanically from the pressure and friction exerted on the mucous membrane, (which is very thin in those places) by the tongue in sucking either from the nipple or bottle. According to Parrot,[2] what

[1] *Die Krankh. der Neugeb. und Säuglinge:* Wien, 1850, i., S. 105.

[2] *Loc. cit.*, p. 207.

occurs first is a sponginess of the epithelium and a swelling of mucous membrane with proliferation of its nuclei; and afterwards a casting-off of this and the formation of a shallow erosion. It is not correct to regard these aphthæ as arising from ulceration of the already-mentioned miliary nodules in the palate, which almost always occur only in the raphé and its near neighbourhood, while aphthæ are situated laterally on the palate. Occasionally, however, ulcerations do also occur in the raphé, either superficial or deeply penetrating, and these may be regarded as possibly arising from the nodules. The ulcers which occur in this situation, however, are on the whole far less common, and, with comparatively few exceptions, I found them only in atrophic children. They resembled the aphthæ of which we are speaking, in every particular, but had sometimes a more elongated shape. They occurred generally on the arch of the palate and were sharply defined, rounded, and of a yellowish white or grey colour. Occasionally the subjacent bone was exposed. I cannot share the opinion of Parrot that all ulcers which occur outside the raphé are syphilitic in origin. For example, in a child of six weeks old who died in a state of extreme atrophy without showing a single sign of syphilis, I have seen the whole palate covered with such ulcerations, while at the same time there was an abundant growth of thrush in the mouth and on the palate. The ulcers which at first appear yellowish or greyish-white, become at last of a brown colour; and in some cases bare bone can be felt with the probe.

The aphthæ of the palate being a very frequent "decubital" symptom require treatment only if, under the influence of defective nutrition, they are increasing in size and depth. In that case I usually paint them with a solution of sulphate of zinc (1 in 10), or nitrate of silver (1 in 15). I have only three times seen these aphthæ after the first three months, in children of 5, 9, and 12 months respectively; in whom they were probably occasioned by too strong rubbing (during the cleaning of the mouth) of the parts of the palate affected. Parrot also mentions the case of a child of two and a half years old with measles, who besides other erosions and aphthæ in the cavity of the mouth presented two quite characteristic plaques on the palate.

Although Epstein[1] and Fischl[2] go perhaps too far in

[1] *Prager med. Wochenschr.*, 1884, No. 13. [2] *Ibid.*, 1886, No. 41.

thinking that many of the affections of the mouth in new-born children (stomatitis, plaques and ulcerations) are the result of mechanical injuries from frequent cleaning of it, it is well to observe the care in cleansing which they enjoin. In some cases I have, in fact, been able to observe an unusual spreading of the patches on the palate, due to hard rubbing. An appearance may result from this resembling a diphtheritic membrane.

> This happened, for example, in the case of two children in the first week of life, in whom in the first place two patches had appeared at the sides of the palate and gradually extended so far that they at last ran into one another, and the whole back part of the arch of the palate was covered by a continuous yellowish-grey membrane which ended in a sharply-defined line above the uvula. The latter, as well as the tonsils, was, however, normal; and this circumstance, as well as the mode of development which I have described, was sufficient to cast doubt on the diagnosis of diphtheria which had been made in one of the cases. As it turned out, the whole disease disappeared within ten days without leaving behind it any loss of substance.

Melæna Neonatorum.

I shall conclude the consideration of the diseases affecting new-born children exclusively or generally, with a few remarks on melæna neonatorum, a disease on the whole rare and which I have myself had an opportunity of observing in only a few cases. This complaint is characterised by hæmorrhages from the stomach and intestine commencing as a rule between the first and seventh days after birth, rarely later. Sometimes only a vomiting of dark blood on several occasions takes place; and after this, in spite of the extreme collapse at first, the children gradually recover. In other cases, however, the vomiting of blood returns more frequently and the diapers are saturated by blackish blood from the anus. Sometimes the vomiting of blood is entirely absent and only bloody stools occur following quickly on one another. These contain meconium or fæcal matter to begin with, but later consist solely of fluid and coagulated blood. Other morbid appearances may be entirely wanting, and the examination of the abdomen yields nothing abnormal. In most cases, owing to the repeated copious hæmorrhages there follow within 24—48 hours, death-like paleness, coldness of the skin,

disappearance of the pulse, and death. But a small number recover after the bleeding has ceased. The mortality, according to different authors, varies between 35 and 60 per cent.

The views as to the mode of origin of this dangerous malady vary greatly according to the pathological conditions which have given rise to it. Billard explains the hæmorrhages as due to the hyperæmia of the mucous membrane of the alimentary tract which is present normally during the first days of life, and may be aggravated by any chance disturbance of the venous circulation, *e.g.*, by an asphyxiated condition of the child at birth, atelectasis of the lungs, congenital malformation of the heart, or enlargement of the liver and spleen. Others (Kiwisch) blame premature ligature of the umbilical cord; while in recent times attention has been directed to little rounded ulcers of the mucous membrane of the stomach and intestine. These were known by the French authors, Denis, Billard, Rilliet and Barthez, Barrier and others, and were at a later period described by Vogel, Hecker, Buhl and others among ourselves. There is a difference of opinion as to the mode of formation of these ulcers (an anatomical description of which is to be found in Parrot's works[1]) for some ascribe to them an inflammatory origin, others (Bohn) hold that they proceed from an ulceration of the follicles or from a fatty degeneration of the small arteries (Rehn). Lastly, Landau,[2] arguing from a case of duodenal ulceration with thrombosis of the umbilical vein, believes in an embolic origin of the ulcers and thinks that they arise from thrombi which are driven from the ductus arteriosus, or from the umbilical vein into the small arteries of the gastric mucous membrane, and bring about gangrene of the affected area. At the same time, the corrosive action of the gastric juice upon the portion of skin which is excluded from the circulation, is held to promote this gangrene. Asphyxia and incompleteness of the first respirations are of importance in so far as they favour a stagnation of the blood column in the umbilical vein and the formation of thrombi in it. As a matter of course the ulcerations have in recent years been looked upon as parasitic and as occasioned by deposits of micrococci (Rehn[3]).

[1] *Loc. cit.*, p. 247.
[2] *Ueber Melæna des Neugeborenen u.s.w.:* Breslau, 1874.
[3] *Centralzeit. f. Kinderkrank.*, 1878, S. 227.

You see what a variety of views are held upon the pathology of melæna in infants; and hence you will conclude that this affection may be only a symptom, *i.e.* may be caused by various anatomical processes, just as in later life. It is most certainly a fact that ulcers of the gastric mucous membrane are pretty common in new-born children, while melæna is, on the whole, only rarely observed; and that, further, in the very many cases where multiple ulcers have been found post-mortem, neither vomiting of blood nor bloody motions were present during life. This is all the more remarkable because the contents of the stomach, as I have myself seen in such cases, appear bloody and blackish; and further, the little ulcers in the mucous membrane may be covered with a layer of mucus of a blackish colour, although during life no blood-evacuations had taken place. Even should we ascribe the melæna in isolated instances to ulcers in the stomach and intestinal canal (I have myself known one such case in which two ulcers were found in the duodenum[1]) yet we can by no means do so in the majority of cases.[2] Kling, in six cases which had ended fatally, found gastric and duodenal ulcers in but two, in all the others only venous or capillary hæmorrhages could be assumed. I should not, however, underrate the merit of Landau's work. His case of duodenal ulcer and the fact that hæmorrhage from the bowel may arise from embolism of the mesenteric artery,[3] really make it incumbent upon us to examine, in this particular, the arteries of the stomach and bowel in all cases of melæna in new-born children. On the other hand, we must acknowledge the possibility of hæmorrhages without ulceration when the venous pressure is much increased owing to interference with the respiration. Landau himself admits this, and Epstein's experiments prove it; for he caused blood-extravasations in the mucous membrane of the stomach in animals by suspension of the respiration.[4] Finally, I need not do more than mention here that cases of bleeding from the intestine may depend upon a hæmorrhagic diathesis, or very likely on "puer-

[1] Veit, (*Deutsche med. Wochenschr.*, 1881, No. 50). The child was seven weeks old, and had brought up only small quantities of coffee-ground material, and had never had regular vomiting of blood or bloody motions.

[2] "Ueber Melæna neonatorum": *Inaug.-Diss.*, München, 1875.

[3] Klob (*Zeitschr. der Gesellsch. der Wiener Aerzte*, 1859) has also observed in a child of eight days old a thrombosis of the mesenteric artery, with effusion of blood in the mucous membrane of the bowel.

[4] *Arch. f. experim. Pathol.*, Bd. ii.

peral infection"; because the bleeding in such cases forms only one link in the great chain of local and general symptoms. Two very interesting cases are mentioned (Rilliet[1]) of copious bleeding from the intestine in twins, who were almost simultaneously affected and were reduced to a state of extreme collapse. One might have been inclined in these cases to the diagnosis of a general hæmorrhagic diathesis had the disease not ended in recovery and both children remained afterwards perfectly free from hæmorrhages.

These cases, to which others might be added (Rahn-Escher, Silbermann),[2] also show that not only the slight attacks in which the vomiting of blood occurs only once or twice, but also the very severe ones in which the symptoms of collapse, general coldness, disappearance of the pulse, and turning upwards of the eyeballs appear, are still capable of cure. We must, therefore, (even when circumstances seem most unfavourable) always endeavour to arrest the exhausting hæmorrhages. Cold compresses or the application of an ice-bag to the abdomen, the arms and legs being at the same time wrapped in warm flannels, are to be recommended. The most suitable nourishment, when the children cannot take the breast, is iced milk, given with a teaspoon. In severe vomiting of blood this method of feeding is on the whole to be preferred to the breast; because when the latter is given the stomach is readily overfilled, and vomiting occasioned thereby. For medicine I should recommend first the liquor ferri perchloridi (gtt. ii every two hours in a teaspoonful of oatmeal-water), second ergotin (gr. $\frac{1}{2}$—$\frac{3}{4}$ internally or by subcutaneous injection). Enemata are not advisable, because they do not reach the higher portions of the intestine and are rather apt to produce slight tenesmus and fresh bleeding—as happened in the first of Rilliet's cases. As regards prophylactic measures, Landau warns against premature ligature of the umbilical cord; and it is always best not to tie it until the respiration is fully established and the children cry strongly.

In conclusion I may further remark that new-born children occasionally pass a little blood upwards or downwards, which has either been swallowed from sore nipples or an operation on the mouth or throat. This blood may also come from the nose

[1] *Gaz. méd.*, No. 53, 1848.
[2] *Jahrb. f. Kinderheilk.*, 1877, Bd. xi., S. 378.

and the neighbouring parts. Its amount, however, is always small, and it is scarcely possible to confound it with real melæna.

The following case stands quite alone :—

> A child of 5 days, admitted 1st October, 1881. Since the 3rd day of life, repeated vomiting of blood and black, bloody stools. The child sickly, shrivelled, anæmic. Extremities cold. Anal aperture covered with bloody fæces. Pulse imperceptible; temperature 87·8° F. Takes no nourishment. Death that evening. *P.-M.*—General anæmia. Spleen normal. Immediately over the cardia a ring of ulceration, 1⅝ inches long, surrounding the whole œsophagus. The submucosa remained free; it was swollen and infiltrated with greyish-white matter. The ulcer sharply defined above. Otherwise, everything normal.

We were unable to throw any light upon the origin and nature of this œsophageal ulcer.

SECTION II.

DISEASES OF INFANCY.

I.—*Infantile Atrophy.*

At no other period of life is the method of feeding of so much importance as during that which embraces the time from birth to the end of the first year. According to recent investigations (Baginsky[1]), the number of the glands throughout the intestinal wall goes on increasing from the fœtal period to the later stages of life, and the development of the glandular system advances *pari passu* while the lymph-vascular system steadily decreases in importance. Therefore, very young children are less able to assimilate substances, the consumption of which requires much chemical action on the part of the glandular system; but they are so much the more able to digest milk, which is essentially easy of absorption. Nature, for this reason, assigns the new-born child to the mother's breast. You know, however, that a number of difficulties may come in the way of the fulfilment of this necessary provision. Illness of the mother, poverty which compels her to work out-of-doors, and an undeveloped state of the nipple, are among the commonest excusable obstacles; while a number of mothers, generally those belonging to the upper classes of society, cannot combine that which they suppose their duty with what nature has appointed for them; and therefore, neglect the latter. At all events it is an easy matter in the latter class to replace the mother by a hired nurse; but in the lower classes, where on the score of expense the keeping of a nurse is out of the question—it is a different matter, and artificial instead of natural feeding must be introduced. I by no means deny that this, if only carefully and properly managed, gives quite satisfactory results in many cases; and every day brings us

[1] "Untersuchungen über dem Darmcanal des menschlichen Kindes": *Virchow's Arch.*, Bd. 89, 1882.

examples of children who have grown up perfectly healthy under these circumstances. In order, however, to achieve this result, the care and conscientiousness of the mother or nurse must be much greater than in the case of natural nourishment. Not only has the composition of the nourishment to be considered, but also the suitable intervals of feeding; and neither of these can be attained in practice among the poor in such a manner as would be necessary for the child to thrive. The struggle to obtain a livelihood, illegitimate birth, levity, want of judgment, and foolish superstition,—all these influences are disturbing elements. This explains why we find among the infants of the poorer classes such a great preponderance of disturbances of nutrition and, in consequence, such an enormous mortality; of this I have already spoken in the Introduction (p. 2). Deficient and unsuitable feeding is not the only thing to be blamed in these cases. Second to it—though still very potent—come the foul air breathed by these children in crowded rooms filled with emanations of every sort and into which it is impossible to bring fresh air regularly, deficient cleanliness, and neglect of the first stages of the diseases to which children are liable. Some of these causes are active in children's hospitals also—more so in foundling institutions—and in these, therefore, one has abundant opportunity of observing the various results of such unfavourable conditions. These results we group together under the name of atrophy; for I see no reason to exchange this old designation for that of "athrepsia," lately introduced by Parrot. The clinical picture of this morbid state, which may appear in its most terrible form at any period of infancy or even in new-born children, varies naturally according to the stage at which one sees it. The first sign is the arrest of development; which, of course, can only be accurately ascertained by carefully weighing the children every week.[1] Soon, however, it becomes evident even without this that the children are falling off; their fat steadily disappears, the skin on the face and on the whole body becomes flabby, wrinkled, yellowish; and, not unfrequently,

[1] The average weight of new-born children is about 7¼ lbs.; the daily increase in the first month about ⅞—1¼ oz., apart from the first 3—4 days (in which the weight usually diminishes about 8 oz.). At the end of the first month the weight has increased by about a third; in the fifth month it is doubled; in the twelfth month, tripled. Weaning, teething—still more, morbid states—arrest the increase of weight.

there is a branny desquamation of the epidermis. At this stage the organic functions, especially those of the alimentary canal, may remain quite uninterfered with or almost so; and by suitable nourishment and care we may still avert the threatened exhaustion and initiate recovery. In the majority of cases, however, the possibility of such a favourable turn is excluded by their poor circumstances; functional disorders of the digestive organs (especially vomiting and diarrhœa) are added; and finally the last stage developes which precludes all hope, and leaves to the physician when he sees a number of such children together (as *e.g.* in my wards), only sadness and resignation.

When the clothes with which the mother has wrapped up the child are thrown back, there looks out from them a yellowish pale face, with peaked chin and projecting bones, and numerous wrinkles (in all directions, especially round the nose and mouth and on the forehead) which become more pronounced on every movement of the facial muscles. The eyes are wide open and staring, or half shut with a dull expression, a perfect picture of languor, which from time to time is interrupted by painful distortion of the wrinkled features, by a feeble cry, or a hoarse whining sound. The movements are slow, or there are none at all. And yet the appearance of the face is only, as it were, the prelude to the horror excited by the examination of the naked body, which, when one considers the domestic circumstances causing it, is fitted to make a truly tragic impression. The shrivelled earth-coloured skin hangs in folds over the bones, which—especially the shoulder-blades, spinal column, ribs and ilia—distinctly mark out the outlines of the skeleton. On the neck and abdomen the skin lies in large folds; and these, owing to their loss of tone, retain their shape (as in Asiatic cholera) for a considerable time, as if they were formed of dough. The fatty tissue seems to have entirely disappeared; and the muscles (*e.g.* the gastrocnemii and the adductors of the thighs) feel like thin shrivelled bands. Not unfrequently the skin is erythematous on the genitals, anus, and heels; and in various situations—even on the scalp—it is the seat of abscesses and boils of various sizes. The mucous membrane of the mouth and palate is more or less extensively coated with thrush.

In all cases of atrophy occurring in infants at the breast or on the bottle, we must remember that the deficient nutrition of the

tissues may arise from different causes; and to treat fully of infantile atrophy would involve the consideration of no small portion of Pædiatrics. Even when all circumstances seem to indicate a simple atrophy, that is one which has arisen from faulty and insufficient nourishment, we must always inquire whether other causes may not also be acting. First among these causes I should place tuberculosis. Seeing that I shall have occasion later on to enter fully into this wasting disease, I shall only say here that in the first years of life, owing especially to the simultaneous implication of many organs which have the closest relation to blood-formation (the lungs, the lymphatic glands, the spleen, &c.), tuberculosis produces symptoms which differ substantially from those of the same disease in later life, inasmuch as the local symptoms of the various organs are of small importance compared with the general interference with nutrition. Certain proof of a hereditary predisposition to tuberculosis and the physical signs of a consolidation of the lung tissue are the only sufficient grounds for diagnosis; since owing to an accompanying catarrh, crepitations of different kinds may be heard in any case of simple atrophy, and if there is diarrhœa it may depend upon a chronic intestinal catarrh and its results, just as well as on a tuberculosis of the intestine. Although, as a general rule, atrophy in infancy is more commonly simple than tubercular; still, in any special case, repeated examination and observation of the course of the disease are necessary to establish the diagnosis; and Rilliet and Barthez[1] are certainly right in their maxim: "ni les symptômes généraux, ni les symptômes locaux ne peuvent offrir la lumière suffisante; le traitement seul est la pierre de touche du diagnostic."

I must now complete the general picture of atrophy which I have sketched, by adding a number of individual details. Very often disturbances of the digestion arise early, either repeated vomiting (immediately or some time after taking nourishment) or morbid alterations of the fæces, which are passed more frequently than in health, are more liquid, and instead of being bright yellow and of firm consistence present yellow or greenish lumps and streaks. At the same time the

[1] *L. c.*, ii., p. 377.

secretion of urine usually diminishes, so that the child's diapers often appear quite dry or at least considerably less wetted by urine than is the case with a healthy child. This circumstance results for the most part from the diminished appetite of the child. He takes less nourishment and struggles against receiving the bottle, or, impelled by thirst, drinks oftener than usual but always only a little; so that the amount of milk taken—and proportionately that of the urine passed—remains considerably below the normal. I occasionally, however, have met with cases of polyuria in atrophic children; but I have only exceptionally been able to discover a distinct sugar reaction in the urine—which was collected with some trouble, although others (Parrot,[1] Robin[2]) have been able to ascertain its presence (or at least that of a reducing substance) and that of albumen although the urine was scanty and dark in colour. Although the quantity of sugar has almost always been small, still cases have occurred where as much as 7 per cent. has been found in the urine, and which might therefore be looked upon as real diabetes. In addition to these symptoms the child is very fretful, cries much, and sleeps less than usual. As the disease progresses, all the symptoms increase in severity. The stools, which at first were only slightly altered, become more and more liquid, of a dirty-green colour, very offensive, and contain flocculi. Very rarely is the opposite condition, normal, or even diminished frequency of defæcation, observed. The appetite is quite gone; and the child loses even the strength to suck from the bottle or the nipple, and has to be fed with small quantities of milk out of a spoon. The respiratory organs—unless any complications are present—show no abnormal physical signs; only, the breathing becomes very shallow and weak, like the heart's action—which in the last stages may sink to sixty, or even fewer, pulsations in the minute. The body-temperature may finally fall to 85° F., or lower; and when the finger is introduced into the child's mouth, it feels a strange coldness. Owing to the weakness of the heart, the skin (which has hitherto been of a dirty-yellow tinge) presents on the extremities (lips, fingers, toes, nails) a slight cyanosis. Under these

[1] Other changes in the urine which Parrot has pointed out in regard to the amount of the urea, sediments, fat, &c., which it contains—are of more importance as bearing on tissue-change than for practical medicine.

Gaz. méd., 1886, No. 25.

circumstances we see that the great fontanelle sinks beneath the level of the surrounding cranial bones, whereby a depression about a line in depth is formed, and its size may be more or less diminished by the approximation of the cranial bones. The diminution in volume of the brain and the consequent decrease in the tension of the cranial capsule account for the fact that the edges of the bones are made to overlap one another. The half-shut eyelids, towards the end scarcely capable of the slightest movement, complete the picture of fatal collapse, which often comes on almost unnoticed, because in the last days the condition of the child may be already like that of a corpse—the pulse being imperceptible, the skin cold, and the breathing slow and extremely feeble. In new-born children, during the last period, that rigid condition of the body which I have described (p. 51) as true sclerema, may make its appearance.

One can make no definite statements with regard to the duration of atrophy; because it is regulated by the circumstances of the child, its original strength, the means of nourishment available, and especially by the complications which may arise. Thus, we often see new-born children die within the first weeks or months of life with the symptoms already described; while older children may prolong their miserable existence for many months, and succumb only to an exacerbation of the diarrhœa or to an intercurrent acute affection of the lung. Broncho-pneumonia is, under these circumstances one of the commonest causes of death; and in these as in other cases, it may be occasioned by an accidental chill or by food getting into the windpipe, especially when the child always lies on its back. I should draw your attention especially to the fact that, in these cases of extreme atrophy and debility, careless feeding, particularly the bad habit of leaving such children to themselves when feeding from the bottle, may result in milk being drawn into the air passages, and consequently in bronchitis and pneumonia—if indeed death does not rapidly ensue from asphyxia. This may also take place if the children bring up the contents of the stomach into the mouth, especially during sleep. I have once or twice unfortunately met with such cases in my ward, where, with the best intentions, it is yet impossible to keep up a constant oversight of each individual child; and Parrot (l.c. p. 67) mentions certain similar observations on cases of death from

asphyxia in which chyme was found in the lungs and by its chemical action had produced softening of the lung tissue and adjacent diaphragm.

In post-mortem examinations of children who have died of simple atrophy, we invariably find nothing but an almost entire absence of subcutaneous and perivisceral fat, attenuation and pallor of the muscles (including the heart muscle), and usually extreme anæmia of all parts.[1] There is often extensive atelectasis of the lung tissue, owing to the diminished power of inspiration. Among complications, the commonest are broncho-pneumonia, catarrh and follicular inflammation of the intestinal canal. Owing to the extreme weakness of the heart in the last stage of the disease, one sometimes finds venous engorgement and thrombosis—especially in the sinuses, in the dura mater, and in the renal veins. Such thrombi may occasion symptoms during life. I shall return to this in another place.

In atrophic children, our estimate of the danger must depend essentially on the degree of the disease and the possibility of removing the patient into more favourable conditions. If the atrophy is not too far advanced, and there are no serious complications, and if we can exclude any suspicion of tuberculosis and procure for the neglected child good nourishment and nursing, we may still give a good prognosis. It is astonishing in such cases how quickly the body may increase in bulk and strength, and the child which had the look of an old man may be transformed into a well-developed thriving infant. On the other hand, in practice among the poor, we can scarcely entertain a hope of attaining the same result by our directions and superintendence, however careful; and the younger the children the less hope there is. Thus, new-born children run the greatest risk; they supply most of the fatal cases, and the finer pathological anatomy of the disease is founded especially on the results of post-mortems of cases in which death had occurred in the first weeks or months of life. Although we are bound to acknowledge the very high merit of Parrot's contributions to this subject, yet

[1] According to the investigations of Ohlmüller ("Ueber die Abnahme der einzelnen Organe bei an Atrophie gestorbenen Kindern": *Inaug.-Diss.*, München, 1882.) the loss of fat is principally at the expense of the subcutaneous adipose tissue, the loss of albumen at the expense of the muscles (not including the heart), while the brain remains quite unaffected, also the heart and liver remain tolerably normal.

in my opinion his works do not authorise one to describe a new species of disease under the name of "athrepsia" of new-born children. This, as I have already remarked, is nothing more than our "atrophy," and the rapid course depends solely on the tender age and wretched circumstances present in the case of Parrot's patients. Thus we must explain the one-sidedness of his view, which puts down among the symptoms of athrepsia a number of pathological appearances which either have nothing at all to do with atrophy (such as trismus), or may also occur in children who are not atrophic (as thrush).

In turning to the treatment of atrophy, I do not fail to recognise the difficulties which stand in the way of thoroughly performing my task. Were I to undertake this in its full extent, I should far exceed the limits assigned me. Indeed, I should have to go into the whole treatment and care of a healthy child, from its birth to its weaning; because all the mistakes which are committed during this period in regard to feeding, cleanliness, clothing, &c., are at once reflected in the state of the child's nutrition. I should further have to enter into the spheres of social science and public health, since we can expect the removal, or at least the improvement of the unfavourable conditions in which atrophy is most apt to arise, only from comprehensive regulations imposed by the state and the various local authorities, promoting the general welfare of the poorer classes. Among such we may mention regulations concerning improvement of the dwellings, the providing of light and air for that first period of life which needs them so much, the possibility of the mothers feeding and nursing their own children and not being obliged to entrust them to strangers who for a small payment undertake a duty which they afterwards either neglect or worse than neglect. Such persons, indeed, ought to come under the rigour of the law, if their iniquity could only be proved. The humane efforts of our time and the wide-spread sympathy which the lot of these unhappy little creatures has called forth, are steadily doing away with the order of "angel-makers" I have just alluded to. Foundling institutions, crèches, and children's-refuge societies have been established in many places, in some munificently, and their beneficent action is worthy of all recognition. But all this is not nearly sufficient to grapple on a large scale with pauperism and its resulting

conditions. Thus the fulfilment of our apparently limited task —the treatment of infantile atrophy—remains closely bound up with the solution of this great social problem. You will soon become convinced in practice that treatment under the circumstances I have described can yield but small results; that all your directions are rendered useless by the simple fact that they cannot be carried out, or else by the evil intentions of those in charge. You must get accustomed to see every year a large number of such children pining away and sinking into the grave, without your being able to hinder it. This result is exemplified most sadly in institutions in which a considerable number of atrophic children are lodged together, hospitals, and all kinds of children's asylums. Only those foundling institutions which adhere to the boarding-out system, *i.e.*, giving the greater part of their children out to nurse in the country, can achieve better results.

Such being the state of matters, I must here confine myself to the discussion of a point which is certainly the chief one, and that which can be most readily dealt with from the purely medical side, namely, nourishment. I have very little to say on the natural mode of feeding—the mother's breast or that of a nurse. As I am not now lecturing to you on the dietetics of children, but on their diseases, I cannot enter more fully into the physiology of feeding, the qualities of human milk, the choice of wet nurses, &c. All these matters have to be considered by me only in their relation to pathological conditions; and therefore I must first tell you that even when fed in the natural way children may become atrophic if the milk continues to cause dyspeptic symptoms (*i.e.*, vomiting or diarrhœa), by which, naturally, the absorption of the amount of chyle necessary for normal nutrition cannot but be prevented. On the other hand, cases sometimes occur where the milk of a nurse disagrees with the particular child whom she is hired to suckle; and in that case the child suffers continuously from digestive disturbances, although these may not be present to a marked degree they retard its development. The same nurse after her dismissal may suckle another child with the best possible results; so that one must not assume the presence of some peculiar quality in the milk but rather a peculiar idiosyncracy of the first child, which in its turn may thrive extremely well with

another wet nurse. Strange things sometimes occur even when the mother suckles her own child. Thus, one sometimes notices that a mother who has already nursed one or more children most successfully, is obliged to take a subsequent child from the breast because her milk does not agree with it. And yet one may not be able to find any cause for this. Still, I may observe that it agrees well with infants in general to get good diluted cow's milk from a bottle once or twice daily—or at any rate during the night—in addition to their mother's milk. This practice, however, I consider justifiable only when the mother herself is suckling and not when there is a hired nurse. Even the commencement of menstruation in those who are nursing has in very many cases no disturbing influence, but it must always make us careful. Experience alone can decide in these cases. If the infant begins to have digestive disturbances (vomiting or diarrhœa) not only transient but continually recurring, and the body-weight ceases to increase or even diminishes, there must be no delay in making a change of nurse. In order, however, to recognise the loss of weight early, one must weigh the child carefully at least once every week, estimating the results most cautiously, with due regard to the influence of accessory circumstances (such as articles of clothing and the contents of the stomach, bowel, and bladder). These weighings can generally only be carried out in institutions or in private practice. In by far the greatest number of cases of atrophy which occur in connection with the polyclinic or in practice among the poor, we must be content with the observation of our own senses.

The symptoms which the children themselves show—dyspepsia and incipient atrophy—seem to me of far more significance than all the methods by which it has been attempted to estimate the quality of the mother's or nurse's milk. We certainly learn from the microscope, the number, form, and size of the milk globules; and it is doubtless a very good thing to find those fully formed and in proper number; because, when the globules are small and scanty, they do not as a rule afford the proper amount of nourishment.[1] But these investigations, although made by the most practised observers, give by no means uniform results as to

[1] Dogel (*Jahrb. f. Kinderheilk.*, xxii., S. 256) describes milk-globules capped with a crescentic mass of a finely granular, occasionally nucleated material, which are said to occur in large numbers in the milk of women whose children suffer from diarrhœa (?).

the influence of the various microscopic differences on the state of the child. More difficult still is the chemical estimation of the milk, which very few practitioners are competent to undertake for themselves in a sufficiently thorough manner. Also the results of the examination by no means always agree with the clinical observation, since, *e.g.*, an excessive amount of fat in the milk may excite dyspepsia in one child, and may be very well borne by another. I therefore advise you, above all, to make the condition of the child the standard whereby to judge the milk; just as in choosing a wet-nurse it is best to be guided by the condition of her own child. This, in my opinion, is the only proper and practical way. You may in this way, of course, be obliged to try three or even more wet nurses for the same child, but you must not be deterred by such difficulties, or by the inconvenience of repeated inspection of nurses. The success in the end and the consciousness of having done your duty will be your reward. I may also mention that an insufficiency in the amount of milk can be recognised not so much by feeling the breasts and noticing what can be pressed out of them as by the diapers' being dry and the child's continuing to cry after it has received the breast, when, had it been properly satisfied it would have fallen into a quiet sleep. In general the quantity of the milk diminishes from the beginning of the eighth month after confinement.[1]

Far more difficult, however, is the situation in the great majority of cases: where, from the reasons repeatedly given, the natural mode of nourishment is quite impossible; and the infant who is beginning to waste has to be put upon the bottle. It is inconceivable with what substitutes for milk the children of the poor are fed; but daily experience in the polyclinic is continually affording new proof of the stupidity and barbarity of these people. Thin oat-meal water alone, or that mixed with a little milk, or decoctions of meal of all sorts, form the wretched nourishment of many infants from the first days of their life. And even this is not given to them regularly, or as their hunger requires, just because the mothers or nurses have no time or inclination to discharge this duty. I have already mentioned to you (p. 15) the scantiness of the salivary secretion in the first months of life; and you will understand that during that period

[1] Pfeiffer, *Jahrb. f. Kinderheilk.*, Bd. xx., H. 4, 1883.

(*i.e.* till about the tenth week) absolutely no food which is composed of amyloids should be given, because those substances require saliva sufficient to change them into sugar. Can one be surprised, then, that with such a diet from the beginning the foundation of dyspepsia is laid, the stomach and intestine are surcharged with undigested masses, and tympanites and diarrhœa arise? And further, of course, these substances have a very small nutrient value compared with human milk. Where the latter cannot be procured we must order cow's milk as the only substitute suitable during the first three months. This does not, indeed, correspond entirely to human milk for it contains more casein and less sugar; and hence there is a greater tendency to acid-fermentation, so that cow's milk becomes sour more readily than human milk. The amount of fat in human milk is certainly liable to great variation, but is usually less than in cow's milk. A difference of the utmost importance lies in the fact that the casein in human milk is almost quite soluble, while that in cow's milk is so only to a small extent, so that the former is easily dissolved by artificial gastric juice and acids, the latter only with great difficulty; that, finally, the casein of cow's milk forms on coagulation a dense coherent curd which is difficult to dissolve, while that of human milk coagulates in small loose flakes (Biedert[1]). You will understand how important this difference must be for the child's stomach. The loose coagula of human milk are much more easily acted upon and dissolved in the stomach by the pepsin and hydrochloric acid of the gastric juice than those of the cow's milk. The fæces of children nourished with the latter will, therefore, always contain more undigested casein than those of children at the breast; and, on account of the greater amount of fat in the milk, they will also contain more fat. We cannot remove this drawback as we should wish, even by the much recommended addition of barley- or oat-meal water, gum arabic, lactin[2] and so on; although

[1] Soxhlet, (*Münchener med. Wochenschr.*, 1886, No. 15 & 16) is right in attaching great weight to this fact especially, that human milk in the breast is absolutely germless, while cow's milk is always rich in fermentitive agents, which have got into it in the stable during milking, from the excrements, &c. A repeated and thorough boiling is therefore absolutely necessary, and S. has constructed a special apparatus for this, which from personal experience I can strongly recommend to you.

[2] The addition of lactin, according to the experiments of Hryntschak (*Arch. f. Kinderheilk.*, 1882, iii., S. 421) is absolutely injurious.

we may at any rate make up for the other less important differences by suitably diluting the milk. In general, during the first three months you may take a proportion of one part milk to three parts water; during the second three months, one to two; during the third, half and half. From the ninth month onwards you may give 2 : 1 or quite undiluted milk, which like the water must always be boiled, in order, if possible, to destroy the germs of fermentation contained in it. It is quite evident that the proportion of dilution which we have given may be modified by the quality of the milk, which unfortunately often leaves much to be desired. The chance of recovery of atrophic children among the poor depends principally upon the procuring of unadulterated fresh cow's milk; and the public ought to give more attention than they have hitherto given to this point,[1] on which the well-being of the rising generation so greatly depends. Much more can be effected in this way than by all the recently recommended methods of preserving milk, however meritorious they may be. We must not forget that, in the whole question of artificial nourishment, we are chiefly concerned with the poorer classes, who are unable to bear the least additional expense; and that of all substitutes for human milk, fresh cow's milk is always the cheapest.[2] Asses' milk, which chemically most resembles human milk, is certainly the dearest substitute. The experiment which was successfully made in Paris[3] of using asses' milk for the feeding of infants in the first six to eight weeks, is therefore all the more deserving of recognition.

Since, however, insuperable difficulties lie in the way of the general employment of asses' milk, cow's milk forms the best substitute for the natural mode of nourishment, not only for the first month but for the whole period of suckling. I consider it allowable to give other substitutes only when good milk either cannot be procured in any way or does not agree with the children, *i.e.* when it causes continuous vomiting and diarrhœa. As a general rule, the latter circumstance does not often occur,

[1] *Cf.* Cnyrim, "Ueber die Production von Kinder-und Kuhmilch in städtischen Milchkuranstalten" · *Deutsche Vierteljahrschr. f. öffentl. Gesundheitspflege*, ix., 1879. —Kormann, *Jahrb. f. Kinderheilk.*, N. F., xiv., S. 238, and xv., S. 300.—Abstracts on the subject of "Recent contributions to the question of infantile nourishment" in the *Arch. f. Kinderheilk.*, Stuttgart, 1881, ii., S. 170.—Biedert, *Kinderernährung im Säuglingsalter:* Stuttgart, 1880.

[2] Hoffmann, *Jahrb. f. Kinderheilk.*, xvi., 1880, S. 143.

[3] Tarnier and Parrot, *Union méd.*, 1882, p. 101.

and one may not unfrequently remedy this state of matters (as I know from experience, and shall have occasion to refer to later on) by having the milk boiled and giving it to the child after it has cooled. There are always, however, a number of cases in which even this cold milk cannot be borne, probably on account of the firm consistence of its coagula, and the consequent difficulty of digesting them. We are then, in default of a wet-nurse, obliged to try other substitutes. Condensed Swiss milk which has recently been so much recommended, is apparently the most available and the best. If we put some of it under the microscope we see the field entirely covered with crystals of sugar of milk, which disappear as if by magic whenever we place a little water on the object-glass. We then see only innumerable well preserved milk-globules. Although I have seen condensed milk used with advantage for months in a few cases, still I cannot recommend this method of feeding; because the enormous addition of cane sugar which is necessary for the preservation of the milk (39—43 per cent.) frequently produces acid fermentation and diarrhœa. Very recently they have discovered how to diminish very considerably this addition of sugar, so as to avoid the injurious effects of condensed milk. Still, I have not yet seen any occasion to make use of this expensive preparation.[1]

Among the numerous artificial substitutes produced in our time, Nestlé's food, which is prepared in Vevey, has acquired special repute, and is most extensively used. This consists of wheat-meal, yolk of egg, condensed milk and sugar, in such proportions that there are 20 parts of nitrogenous matters and 7 parts of salts in 1,000. Usually one boils a tablespoonful of the food with 9 or 10 tablespoonfuls of water, and the fluid is given from a bottle. Nestlé's food may under certain circumstances become tainted, and then it is very injurious. Among others, I have seen one case of a child whom I was asked to see in the summer of 1878 on account of increasing atrophy with whom no cow's milk agreed, and in whom obstinate diarrhœa, naturally increasing the atrophy, persisted in spite of the administration for weeks of Nestlé's food, and of the most various remedies. I then discovered that the food which was contained in a tin box, had not (as it should have) a

[1] Hagenbach (*Correspondenzbl. der Schweizer Aerzte*, 1883, No. 1) and Banze (*Arch. f. Kinderheilk.*, iv., S. 212) recommend these varieties of condensed milk (Helvetia, Romanshorner Milch, 1 in 10 to 1 in 6 barley-water).

smell like that of a rusk, but smelt abominably—like old cheese. I had a fresh tin sent for at once, and then I found that the food prepared from it agreed very well. From my own experience I can recommend Nestlé's food as a suitable means of nourishment after the tenth or twelfth week of life, not earlier. But I am by no means altogether enamoured of it. From experiments which I instituted on other similar infant's foods, such as those of Gerber, Giffey, Liebig, Frerichs, and Kufeke, I am inclined to believe that the same value may be assigned to all of them, and to preparations from the manufactories at Cham and Vevey and Montreux. The lucrative character of this business, moreover, makes it probable that the world will continue to be favoured with new preparations of this sort; which will in turn excel one another in the endeavour to approach as nearly as possible to the composition of human milk.

Among the other well-known substitutes for mother's milk, I shall only mention here Liebig's food and the cream-mixture recommended by Biedert.[1] The former, once so much extolled, is now quite given up because its preparation is far too troublesome to allow it to be generally used in practice among the poor—whom we have chiefly to consider in discussing artificial nourishment. The same may be said of Biedert's cream-mixture, which I used in my ward for some time for a number of atrophic children without being able to convince myself that it was more efficacious than feeding with cow's milk or Nestlé's food. I have not myself sufficient experience of the "artificial" cream-mixture recommended by Biedert, which at any rate is more easy to use; but it is spoken well of by Monti[2] and others, though in this case also the price is a drawback owing to the poor circumstances of many of our patients.

An excellent aid in the nourishment of atrophic infants is wine, especially unadulterated tokay. Whether other kinds of wine, such as sherry and malaga which are frequently given, are to be regarded as of equal value I shall not decide. I myself always prefer to all other kinds the old Hungarian wine, of which my never-to-be-forgotten teacher Romberg used to say that it was not only a "lac senile," but also a "lac juvenile." In the first months of life we may give 20 to 25 drops three or four times

[1] *Virchow's Archiv*, Bd. 60. H. 3 und 4. [2] *Archiv f. Kinderheilk.*, Bd. ii.

daily, undiluted, or in a teaspoonful of water. In older childen we may increase the dose to several teaspoonfuls or more in the day. At the same time one should for the sake of cleanliness order a warm bath daily (93° to 95° F.), to which one may add, if the debility is increasing, aromatic infusions (the best being a handful of camomile and sweet calamus infused in hot-water). Well-ventilated sick-rooms, strict cleanliness, careful regularity in the nursing, all these are (and unfortunately too often remain) "pia desideria," which can be attained only in a small minority of the cases.

From drugs we can expect nothing in atrophy. It is only when it is distinctly complicated with disorders of the respiratory organs or intestines, that there is any indication for their use. And I must here remark that slight dyspeptic symptoms (vomiting or unnatural, offensive, badly-digested stools) may disappear without the use of medicines as the result of suitable dieting.

II. *Thrush.*

The younger the children the oftener they suffer from this affection of the mouth and throat. Thus it is commonest in new-born children and during the first months of life. But it also often occurs in the second half of the first year, and you will meet with it under certain conditions much later, even in adults. The appearance of the disease varies according to its degree and the circumstances in which you find it.

First Degree.—On the mucous membrane of the lips, tongue and cheeks, especially on the folds between the lips and gums and between the cheeks and the alveolar margin, we find separate, white, slightly-projecting points and spots. These can easily be rubbed off with the spatula, but if one uses force in doing this a drop of blood is left. The mucous membrane is otherwise unaltered, and there is no other disorder. This form of thrush occurs very often in perfectly healthy children if the necessary cleaning of the mouth has been neglected, owing to remains of milk being left behind in the above-mentioned folds of mucous membrane, and afterwards decomposing. Sometimes it is not easy at first sight to decide whether we have to do

with real thrush or only with remains of milk, as these have almost the same appearance; the difference is seen when we touch the spots with a spatula, by which the remains of milk (which lie loose on the surface) are at once removed while the spots of thrush adhere more firmly to the mucous membrane.

Second Degree.—The whole mucous membrane of the mouth, as well as that of the pharynx, is of a dark purplish-red colour and noticeably dry. All over it—but especially on the tongue, the cheeks, the lips and the hard palate—one sees a great many white points and spots of rounded irregular form, which here and there (especially in the above-mentioned folds and on the tongue) run together into larger patches. The cavity of the mouth appears to be tender to touch, as the children while sucking often distort their faces painfully, or refuse the breast entirely. At a still more advanced stage we find the tongue, cheeks and hard palate covered with a white membranous coating; while on the lips and gums, and further back on the soft palate and tonsils, spots of thrush are visible in large numbers. These extreme degrees occur only in atrophic children or in those exhausted by severe illnesses (diarrhœa, cholerine). Thus we may explain the circumstance that the mucous membrane, which was dark-red to begin with, gradually becomes pale from the progressing anæmia. In the last stages of the disease in such children I have found the spots of thrush adhering to a perfectly pale and slightly livid mucous membrane, and therefore less liable to be noticed than when the mucous membrane was very vascular. Further, the spots lose their milk-white colour more and more, and often appear dirty-grey or yellowish, the latter colour being due to bile-staining by vomited matter. Accordingly, one must look more narrowly to recognise the whole extent of the disease. The longer it lasts, the more firmly do the patches of thrush adhere to the mucous membrane. Among very many cases of this kind, I remember particularly that of a child of four months in a state of extreme collapse with congenital syphilis, and pneumonia of the right lower lobe; the whole of the pale mucous membrane of the pharynx as well as that of the mouth was covered with pearl-grey patches of thrush which were so firmly adherent that they could only be detached forcibly by means of a pair of forceps, and with some bleeding. New-born children with this disease

often present at the same time the ulcerations on the hard palate which I have already mentioned (p. 63). When we examine under the microscope a little piece of the thrush well teased out, we see that it is principally composed of a number of filaments and spores of fungi. When this was discovered in 1842, by Berg, a Swedish physician, all previous explanations of the disease as due to inflammatory exudation fell to the ground. We can only regard it as of parasitic origin. The filaments appear as long tubes, straight or bent in various directions, transparent, with a sharp contour, 50 to 60μ long and 3 to 4μ broad, and consisting of various segments articulated to one another. Almost all of the ripe filaments present one or more branches of the same form springing from those points of the stem-filament where the joints are marked by a septum. The interior of the filaments usually contains some molecular granules, as well as a few little oval bodies—probably spores in process of development. Round the origin of the filaments one almost always sees heaps of roundish or oval spores from which they arise.[1] Besides the fungous elements the microscope shows numerous epithelial cells, with a varying number of fat-globules and red blood corpuscles which have become entangled in the patches of thrush on being detached from the mucous membrane.

That is all that thrush shows clinically. All the symptoms which were formerly ascribed to it — especially the violent diarrhœa, vomiting and collapse, of which earlier French authors particularly spoke—do not belong to thrush but to the original disease of which it is a result. I have, therefore, only a few anatomical and pathological remarks to add. Thrush is by no means confined to those areas of the mucous membrane which are accessible to our clinical examination, it also frequently occurs (as the post-mortems show) further down—especially in the lower part of the pharynx, and often in the œsophagus, particularly its lower two-thirds; there it occurs either in the

[1] Authors still differ widely concerning the botany of thrush. The name "oïdium albicans" which has been attacked by Grawitz (*Deutsche Zeitschr. f. prakt. Med.*, 1877, No. 20) is indeed given up. *Cf.* Plaut (*Beitr. zur system. Stellung des Soorpilzes:* Leipzig, 1885), Stumpf (*Münchener med. Wochenschr.*, 1885, S. 627), Baginsky (*Verein f. innere Med.*, 30th November, 1885), Klemperer, (*Centralb. f. klin. Med.*, 1885, No. 50), Plaut, (*Neue Beitr. zur system. Stellung des Soorpilzes in der Botanik:* Leipzig, 1887). Plaut regards the fungus as identical with that which we find growing on rotten wood, fresh cow-dung and sweet fruits—Monilia candida.

same way as in the mouth, or forming a more or less perfect cylinder which, owing to the projecting folds of the mucous membrane, looks like a piece of bark. Thrush of the œsophagus is not usually of a pure-white colour but pearl-grey or yellowish and ends just above the cardia in a sharp line. I have found it on the mucous membrane of the stomach only in one case, where it occurred in the form of isolated and somewhat prominent patches. I must, however, admit that such a careful examination of the stomach, as is necessary here was not always made; and, of course, a large number of our atrophic children showing thrush in the mouth did not come under post-mortem examination. I mention this because Parrot[1] has not unfrequently observed thrush in the stomach. To recognise the patches we must first remove by a stream of water the thick layer of mucus which covers them; they then come into view in the form of little papillæ, isolated or aggregated, some of which can only be made out with a lens. The larger patches often present a central depression; and from this, as well as from their generally yellow colour, they acquire a decided resemblance to a favus-crust. Most commonly the disease is found on the posterior wall of the stomach, along the lesser curvature, and in the neighbourhood of the cardia. Here the thrush is so markedly adherent that it is difficult to remove it by a stream of water or by scraping. Beyond the stomach thrush only very rarely occurs. The observations of Valliex and Seux, made without the help of the microscope, are not conclusive. But those of Robin and Parrot may perhaps be so; the former having found it in the small intestine, the latter in the cæcum on two occasions. In this region, as in the stomach, the acidity of the contents is to be regarded as a condition favouring the growth of the fungus. However this may be, we must in all these cases assume that the germs or filaments of the fungus must have found their way down from the pharynx or œsophagus. It is remarkable that the disease, however strongly it is developed in the pharynx, never extends into the back part of the nasal cavity, even in cases of cleft palate where a direct communication exists between the cavities of the mouth and nose. It may, however, be found occasionally on the mucous membrane of the glottis in the form of little

[1] *Loc. cit.*, p. 223.

patches or streaks. Since this is the only part of the respiratory mucous membrane which is affected by thrush, we must agree with Berg and Lélut that only squamous and not ciliated, epithelium affords a suitable soil for the growth of the fungus. Thrush has been found in the lungs only in very rare cases, and it has then probably developed from germs inspired from the pharynx (Parrot, Birch-Hirschfeld).

On examining more closely the relation of the fungus to the subjacent mucous membrane, we find that a part of it lies superficially between the epithelial cells ; another part penetrates more deeply into the tissue, so that the filaments can be distinctly seen to enter the mucous membrane perpendicularly (Wagner[1] and Parrot). This fact explains also the very considerable resistance which one occasionally meets with in the attempt to detach the spots. The observations of Zenker and Ribbert[2] on certain rare cases in which it was found in the brain, seem to indicate that the fungus may be carried into other parts of the vascular system.

Thrush does not seem to develope in a perfectly healthy mouth, or at least it never spreads to any considerable extent. Even in the cases of our first degree, we must assume a certain amount of irritation of the mucous membrane from the remains of milk, which decompose and prepare a favourable nidus for the development of the germs. This is more distinctly seen in the cases of the second degree, which are far commoner. In these it is always preceded by a marked dryness and dark-red colour of the mucous membrane of the mouth ; the tongue becomes rough from projecting papillæ, and it is in these places that the growth of the fungus begins, being favoured by the deficient alkalinity of the mucous membrane. To this feature I have already drawn your attention. The exceedingly small amount of the salivary secretion in the first months must favour in a high degree the formation of acids in the mouth and dryness of the mucous membrane. This view need not for the present be shaken by the cultivation-experiments of Kehrer,[3] according to which saliva seems to be an excellent medium of nourishment for the thrush-fungus. The main influences, however, which favour the germination of the spores are the child's weakness and

[1] *Jahrb. f. Kinderheilk.*, 1868, i., S. 58. [2] *Berliner klin. Wochenschr.*, 1879, S. 618.
[3] *Ueber den Soorpilz:* Heidelberg, 1883.

atrophy; and in proof of this fact I may adduce Delafond's[1] experiments on animals. He was never able to transmit thrush by inoculation to the mucous membrane of the mouth of a healthy well-nourished sheep with copious salivary secretion; but he succeeded at once when he had weakened the animal by hunger, or had chosen for his experiment an animal already diseased and with acid saliva. In accordance with this is the clinical observation that eruptions of thrush, quite similar to those occurring in atrophic infants and those exhausted by diseases of all kinds, occur not uncommonly at a later age, in the last stage of phthisis and in severe cases of typhoid. Among other cases I found in a girl 13 years of age who had died of severe typhoid, not only the pharynx but also the œsophagus as far as the cardia covered with a coating of thrush, which from its dirty-grey colour and the difficulty of closely examining the pharynx had been mistaken during the last days of life for diphtheria. Thus many cases of "diphtheritic complication" of typhoid which are not examined post-mortem are really cases of thrush of the pharynx; and this mistake is all the more likely to be made as thrush may occasionally spare the mucous membrane of the mouth and attack only the palate and pharynx.

Although the spores usually reach the mucous membrane of the mouth along with the food (milk and other fluids) or inspired air, yet direct transmission by the bottle is possible (should its mouth-piece not be repeatedly cleansed every day with the utmost care) and may occasion repeated attacks of thrush in the same child. Be particular, therefore, that the india-rubber mouth-piece of the bottle is carefully washed, left lying in water, and daily cleansed inside with a small brush. Whether thrush may be transmitted from the child's mouth to the nipple of the mother or nurse is a question on which different observers are by no means agreed. Seux[2] says that out of more than 1,600 cases of thrush, he did not once observe its transmission to the nurse's nipple; but others — especially Mignot[3] — on the strength of a few observations, express themselves in favour of such a possibility, chiefly when the nipple is excoriated; and Delafond, in his above-mentioned inoculation of sheep, found that the

[1] *Gaz. hebdomad.*, 1858, p. 909.

[2] *Recherches sur les maladies des enfants nouveau-nés:* Paris, 1855, p. 29.

[3] *Traité de quelques maladies pendant le premier âge:* Paris, 1859, p. 223.

oïdium might be transmitted by a lamb to its mother's teat. We must, therefore, in all circumstances warn those who are suckling of the possibility of such a transmission; and impress upon them as a duty the utmost cleanliness, and especially frequent washing of the nipple with alkaline fluids.

In cases where one has doubts as to the diagnosis of thrush—and these are extremely rare—the microscope alone can decide by showing the characteristic filaments and spores. I have already mentioned that remains of milk-curd on the mucous membrane are readily distinguished from thrush, because they can be easily wiped off. There is, however, another condition which is sometimes mistaken for thrush by the inexperienced; namely, a membranous desquamation of the epithelium of the mucous membrane of the tongue, and especially of the gum, in the form of thin greyish-white layers. The microscope in such cases at once proves the error, by showing only epithelial cells and an amorphous granular mass but no fungus elements. In a few cases we see these accumulations of epithelium only under the tongue, where they become rolled-up and form a transverse cord of a milk-white colour.

> I have found this in two infants; one of whom was thriving and well-nourished, the other atrophic, with many cutaneous abscesses and a bed-sore on the elbow. Neither of the children had any teeth, but the mucous membrane of the mouth was reddened all over and bled readily when touched. The white layer under the tongue could be pretty easily removed, only at the frenum it was somewhat more firmly adherent, and left a drop of blood behind it. Under the microscope I could recognise only fat-globules (really remains of milk), epithelial cells and an amorphous connecting mass, but no trace of the thrush-fungus; and it seems to me that the free desquamation of the epithelium resulting from the hyperæmia of the mucous membrane had assumed this form of a convoluted cord from the continual gliding of the under-surface of the tongue over the alveolar border during the process of sucking.[1]

It is obvious that the local treatment of thrush affords hope of success only in cases of our first degree. In these a mere mechanical wiping-off is generally sufficient. The nurse must

[1] The affection of the frenum linguæ described by Riga (*Glosso-frenulite membranacea*: Napoli, 1881) appears to me to belong to the same category as these cases, and its fatal termination to be mainly due to the atrophy and weakness of the patients. I see no reason to set this down as a special epidemic disease.

not hesitate to rub off the patches of thrush which she sees on the mucous membrane, with a piece of fine linen wrapped round her finger and dipped in cold water—even although it causes a little bleeding. Whenever new eruptions make their appearance, this proceeding must be repeated, and the cavity of the mouth very carefully cleansed in the same way after each nursing; the affection will thus soon be got under. It is a very different matter in cases of the second degree in children who are atrophied and exhausted. Here also, it is true, you will readily succeed in removing the thrush by simply cleansing, as above; or, even better, if you neutralise the acid reaction of the mouth by dipping the linen rag in an alkaline solution instead of in water only (*e.g.* pot. chlorat., ac. boric., borax, or sod. benzoat., 5 p.c. solutions in water; or common salt, a large pinch dissolved in a glass of water). In this matter the experience of practitioners hitherto has been quite at variance with the results obtained by Kehrer in his experiments. For, according to the latter, we should expect the remedies named to favour the growth of the fungus. The general morbid condition which favours the growth of thrush is always the most important matter; and consequently you will continue to have fresh outbreaks taking place in these cases. When this occurs, I have often obtained a good result from painting the whole mucous membrane of the mouth with a solution of nitrate of silver (1 or 2 p. c.) after the patches have been wiped off.

III.—*Hereditary Syphilis.*

During the period in which we most frequently observe the beginning of atrophic conditions and the development of thrush, we have also the most abundant opportunity of becoming acquainted with the phenomena of hereditary syphilis. As this disease occurs in very various forms, it seems most suitable to give you first of all a clinical picture of it as you will most frequently see it in practice; and to discuss later on its varieties and less common conditions.

The children are brought to you usually in the second or third month of life, and appear well- or ill-nourished according as they have been suckled or hand-fed. An extreme degree of atrophy

is by no means one of the necessary features of infantile syphilis; for a large number of children brought to me—especially those on the breast—were well-nourished and of a healthy complexion, although those that were hand-fed certainly showed a tendency to atrophy. Extreme degrees of this latter condition were not, however, to be attributed to syphilis alone; but also to other factors—hunger and all kinds of misery—working along with it.

One of the earliest symptoms is a snuffling character of the respiration, which is caused by swelling of a part of the nasal mucous membrane lying beyond the reach of inspection; and it is often called "a cold in the head" by mothers. At a later stage, the nostrils become blocked by yellowish or brownish crusts, and sero-mucous discharge sometimes slightly blood-stained (coryza syphilitica), and the nose may become somewhat swollen externally. This coryza—which varies very much in degree—I hold to be one of the most constant symptoms of the disease, either preceding the other symptoms, or almost always accompanying them. It is only absent in exceptional cases. Soon we have in addition bright-red patches—usually with a brownish tinge—rounded or irregular in shape, varying between the size of a threepenny-piece and a sixpence. These appear at first singly, and their favourite positions are the region of the eyebrows, the chin and naso-labial fold, the neighbourhood of the anus, and the palms and soles (roseola syphilitica). Many of these patches present a branny desquamation of the epidermis, or are covered with large fragments of it; others—and in many cases, nearly all—have a glazed and almost varnished appearance when looked at from the sides. The patches situated on the chin and nates become gradually macerated by the repeated action of the secretions from the mouth or the urine and fæces. And when the epithelium is shed they are changed into moist red excoriations which, taken apart from other symptoms, have not in themselves any distinct specific character, and may, indeed, be obscured by an erythema surrounding them (intertrigo). In every case, however, the distribution of these excoriations, the patches with unbroken skin which occur along with them, and the presence of the coryza are sufficient indications to warrant a suspicion of syphilis and to justify specific treatment.

If not so treated, the further progress of the disease soon dispels

any uncertainty. The patches now spread over a large part of the body—especially over the forehead, all round about the mouth, and over the extremities. In many places they coalesce and form large dusky-red or brownish-yellow and more or less desquamating patches, covered here and there with scabs owing to the drying-up of moist excoriations. The palms and soles are generally diffusely reddened, covered with fragments of desquamated epidermis, and often (the heels especially) present a glossy redness and tension. There also occur whitish excoriations at the angle of the mouth, and fissures and cracks in the mucous membrane of the lips (rhagades), which readily bleed on sucking and on crying. These, along with crusts which cover the eyebrows and with coryza, present a picture which can scarcely be mistaken any longer by the least experienced and which justifies the diagnosis of syphilis without any confession from the parents. In many cases the picture is rendered still more characteristic by the falling-out of the hair, especially the eyebrows, and even the eyelashes. Trousseau's observation of a brownness of complexion peculiar to congenital syphilis, I can confirm only for a series of cases where the patients were atrophic; while among many other well-nourished children I have observed a complexion just as white as in health.

You must not expect, however, that all the features of this disease are generally as well marked as I have just described to you. Often only some of them are present, while others are wanting or very slightly indicated. Thus, *e.g.*, I have sometimes seen the genital and anal regions quite free from eruption, while the upper parts of the body (sometimes, indeed, only the face) were most typically affected. Further, variations from this typical description of the disease are by no means rare. Thus, instead of roseola, I have repeatedly observed dark-red rounded papules on the soles of the feet, the lower extremities, and round about the anus; or, here and there, dull-red infiltrated spots covered with thin whitish scales, occasionally "figured,"—occurring especially on the glabella and on the eyebrows but also on the cheeks and nates. These bordered partly on psoriasis, partly on condylomatous formations. Occasionally—though only in children in the first weeks of life—we find the remains of bullæ (p. 61) in the form of red spots or

excoriations surrounded by a dry ring of epidermis; sometimes also there are on the soles and palms recent, usually flaccid, bullæ with turbid purulent contents. In many cases, especially in very young children, I have found along with the signs of syphilis almost the whole skin diffusely reddened and covered with large yellowish scales of epidermis mixed with sebaceous matter. Least frequently I have observed vesicular and moist (eczematous) forms of eruption as the expression of syphilis; and these have usually seemed to me as if they had been brought about by maltreatment of the papular and macular eruptions, especially by scratching or the contact of irritating secretions and excretions. In a child six weeks old an eczema which developed along with a copious roseola on many parts of the body, turned out to be simply the result of very abundant perspiration and had therefore nothing to do with syphilis. I have more frequently observed deeper ulcerations, covered with scabs, to develope out of the above-mentioned excoriations in the neighbourhood of the anus and on the scrotum, and also on other parts of the skin (*e.g.* about the eyebrows, or around the navel), just as the intertrigo of the inguinal region, which is often present at the same time, shows a tendency towards the formation of whitish-grey ulcers with red infiltrated margins. On the other hand, I have not been able to convince myself of the correctness of the view[1] that it is only the condyloma latum (mucous papule) which justifies a diagnosis of congenital syphilis. On the contrary, I can affirm that in a considerable number of cases and in spite of the most careful examination we could nowhere find this condition. I by no means consider the mucous papule as one of the earliest symptoms of the disease; for, except in isolated cases, I have never observed condylomatous formations till at a later stage—in children already some months old or suffering from a relapse of the disease. Under these circumstances, certainly, mucous papules occurred frequently enough—especially at the angles of the mouth, on the tongue, under the chin, in the inguinal folds, round the anus, on the scrotum and vulva; sometimes also on the inner and uppermost part of the thigh; most commonly, on the alæ nasi and at the outer angles of the eyes. Thus generally they are found in situations where the folds of skin lie in contact with one another, and irritation is caused by

[1] Caillault, *Traité prat. des maladies de la peau chez les enfants:* Paris, 1859.

pressure and by accumulation of secretions. Their appearance was the same as that of those in adults, and their tendency to become macerated by secretion (saliva, urine, fæces, sweat), was very marked; the epidermic covering of the condylomata being consequently shed, they turned gradually into greyish-white fissured ulcers. In rare cases the condylomata formed continuous masses, which—especially when they occurred on the labia majora—presented a nodular appearance which reminded one of elephantiasis. Onychia was also frequently observed, with thickening and claw-like deformity of the nails, which were finally cast-off by suppuration of their matrix.

In addition to all these various affections of the outer skin, the mucous membranes may also present morbid appearances. In addition to the almost constant coryza, I have observed conjunctivitis with purulent secretions (but in no case iritis, which seems to be one of the rarest of all the manifestations of congenital syphilis), fluor albus, occasionally also redness and swelling of the urethral orifice with pain on micturition. On the dorsum of the tongue there occur, as already mentioned, condylomatous (or rather, perhaps, gummatous), hard, dark projections, especially towards the back; and also the tonsils are sometimes the seat of flat ulcerations arising from condylomata. I cannot, however, regard these affections of the mouth and throat as common, since in the great majority of my cases these parts presented nothing in the least degree morbid; and I here warn you once more against regarding the repeatedly-mentioned palate-ulcers of new-born children as syphilitic in nature. Sometimes we have, along with the syphilitic affections of the skin in children, an alteration of the voice—a more or less pronounced hoarseness, which in extreme cases may go on to complete aphonia. In the following case this loss of voice constituted almost the only symptom of syphilis which could be ascertained:—

> Carl C., four months old, brought to my polyclinic 14th March, 1867, had suffered for two months from hoarseness, and latterly from complete aphonia. We saw the child crying, but scarcely heard any sound. No cough; breathing normal. In the pharynx and on the epiglottis nothing abnormal. Examination with the laryngoscope unsuccessful (Waldenburg attempted it). The child was healthy, well-nourished, and thriving; but there were brownish scars round the anus. On further investigation

it was found that at the age of two months he had suffered from coryza, with a desquamating macular eruption, which was cured by calomel. Diagnosis.—Syphilitic affection (condylomatous ulcer?) of the vocal cords. I ordered mercur. solub. (Hahnem.) gr. $\frac{1}{10}$ twice daily. By the 23rd—that is, after 29 days—the voice was clearer; on the 18th April quite normal. After-treatment with syr. ferr. iod. No return of the disease by December.

As to the nature of the laryngeal affection in this case, I shall not hazard an opinion. I have no experience of perichondritis of the epiglottis or caries of the thyroid cartilage, such as have occasionally been described. Just as little have I seen of the syphilis of the intestine in new-born children, which has recently been spoken of a good deal. This consists in gummatous indurations of the muscular and mucous coats, sometimes ring-shaped, which encircle and narrow the lumen of the small intestine, and usually correspond in position to Peyer's patches, partly also in condylomatous growths and ulceration of the patches and in cellular infiltration of the smaller arteries to their obliteration and causing anæmic gangrene.[1] In the meantime, these conditions do not appear to have any clinical importance, since a case of this kind reported by Schimmer[2] (recovery of a case of diarrhœa under specific treatment) cannot be held to have demonstrated this.

Slight enlargements of the lymphatic glands (from the size of a pea to that of a bean), which are moveable, may often if not always be found on close examination. Sometimes there are only a few behind the ears or at the lower end of the upper arm, or a number massed together in the cervical, axillary and inguinal regions. These masses of glands are always among the most intractable features of the disease, and also often persist after it is cured. In these cases, certainly, it is doubtful whether these glandular enlargements do not form a chance complication depending on other causes. I can by no means agree with Bednàr, who regards the swelling of the lymphatic glands as extremely rare, and says that he himself has only once observed it.

Syphilitic affections of the osseous system were formerly believed to be very rare. A few cases of destruction of the bones

[1] Oser, *Archiv f. Dermat. u. Syphilis*, 1871, S. 1.—Jürgens, *Jahrb. f. Kinderheilk.*, 1881, xvii., S. 126.—Mracek, *Vierteljahrschr. f. Dermat. u. Syphilis*, 1883, S. 209.

[2] *Archiv f. Dermat. u. Syphilis*, 1873, No. 2.

of the nose (vomer and turbinated bones), or of periostitis of the femur and other long bones, have been described; but there seems to have been no idea that these conditions occur in early childhood just as often as in adults, and under certain conditions even oftener. A case of this kind was observed and described by me in the year 1861[1] :—

Anna B., 2 months old; atrophic, although on the breast; brought to my polyclinic on 4th April, because she had not moved her arms for 14 days. Both upper extremities lay flaccid and motionless, even when the child moved its legs and body in different directions. Not the slightest movement of the fingers could ever be made out. If one lifted up the left arm and then let it go, it fell down without any resistance, like that of a dead body; while, if the same were done to the right arm, there were still observable some slight traces of resistance. Sensibility and temperature of both arms normal. Both condyles and the entire lower third of the left humerus much swollen; on the inner side of it a moveable gland about the size of a pea is felt. Cervical, axillary and inguinal glands partly swollen and hard. The soles of the feet—especially about the heels—red, glazed, slightly desquamating. Nostrils obstructed; breathing snuffling; sometimes a slight bloody and purulent discharge. The mother owned to having suffered repeatedly from her throat and from a skin eruption during her pregnancy, and had marked alopecia. Treatment :—merc. solub. (Hahnem.) gr. $\frac{3}{5}$ twice daily; inunctions of ung. pot. iod. into the swollen part. On the 11th (in 8 days) the swelling of the bones had disappeared, the coryza was less, and the arms moveable to a very slight degree. Under the continued use of the medicines along with camomile-baths and tokay wine, rapid improvement ensued. On the 16th the mobility of the arms was once more quite normal and the coryza entirely gone. The mercury was now changed for syr. ferri. iod. (gtt. v., twice daily). On 21st May I found that all syphilitic affections had disappeared, although the atrophy still continued. Further history unknown.

The following cases observed by me recovered in just the same way :—

A child of 6 weeks, brought to the polyclinic on 14th February, 1879, with brownish colour of the skin. Hand-fed, but pretty well nourished For three weeks coryza, fissures on the lips, and onychia on all the fingers and toes. All the nails much thickened, deformed, and already much loosened from their beds. The terminal phalanges covered with scales of epidermis; much desquamation of the soles, less of the palms. The left arm, which

[1] *Beiträge zur Kinderheilk.* : Berlin, 1861, S. 192.

had been hanging flaccid for a week, now incapable of movement. The lower third of the humerus much swollen and tender. The right testicle larger and harder than the left. All functions normal. Treatment:—Calomel gr. $\frac{1}{7}$ twice daily. On 26th mobility of arms returned, swelling diminished by about one half; fissures and coryza almost healed. The nails have almost all fallen off, the new nails growing under them. To continue the treatment.

Child of 8 months, brought to polyclinic 20th May, 1876, with a relapse of syphilis. Papular and macular eruption on the chin and the upper lip; severe snuffles and coryza. Swelling of the lower epiphysis of the right humerus, with difficulty in moving it and pain on pressure. The left arm normal. Mercurial treatment. Further course unknown.

While in these cases only the lower end of the humerus was the seat of the syphilitic periostitis and ostitis, the following cases show that other long bones may also be attacked in the same way:—

Child of 10 weeks, brought 18th November, 1877, with coryza, obstruction of the nostrils by scabs, and glazed, red, flat umbilicated papules round the anus and on the nates. Tender swelling of the lower epiphyses of the radius and ulna on the left side; also of middle phalanx of left middle finger, and of first and second phalanges of right finger. Mercurial treatment. 27th December:—With exception of epiphysial swelling, child has almost quite recovered. Phalanges of fingers almost quite normal. Treatment continued.

Child of 3 months, brought to the polyclinic on 7th June, 1875; well-nourished and thriving. Intertrigo with erosions round the anus and genitals. Coryza almost since birth, with purulent discharge and crusts at the nasal apertures. For 4 weeks swelling of upper epiphyses of bones of the right forearm. Tender on pressure. Joint unaffected. Right arm hangs flaccid and is very little moved. All other bones apparently normal. Mercurial treatment. Marked improvement by end of June. Further course unknown.

Child of 12 weeks, brought 18th June, 1879; coryza, enlargement of lower epiphyses of the radius and ulna on both sides; most marked on the left. Both arms incapable of movement. Roseola on the whole body. Fissures on the palms and desquamation of the soles. Course unknown.

Child of 3 months, brought 28th November, 1879. Well-nourished, by mother. Swelling of epiphyses of all extremities; complete immobility of the arms. Legs flaccid. No other syphilitic symptoms. Mercurial treatment. Movement of arms improved after 6 days. Swelling of epiphyses also soon diminished. Did not return for treatment.

You see that not only may the epiphyses of the different long bones be distinctly enlarged, but also those of the digital phalanges. Such cases closely resemble osteomyelitis (pædarthrocace)—*i.e.*, a hard swelling, covered at first by skin of normal colour which is not adherent to it, but in the course of time becomes red, breaks out in little fistulous openings and after suppurating for years heals at last with a funnel-shaped cicatrix. I have seen this several times in addition to case 4, especially in relapses of hereditary syphilis in the first and second years of life; but in every case on the fingers, never on the toes. In a child of four weeks, who presented no signs of syphilis except coryza, there was considerable enlargement of the middle phalanx of the third finger on the right hand and swelling of the upper epiphyses of the left humerus and radius with paralysis of the left arm, only the fingers of which could be moved. In another child of six months there was enlargement of the first phalanges of three fingers besides other syphilitic symptoms—all the epiphyses of the upper extremities being normal. Other authors [1] have recently treated of this "dactylitis," which must always be regarded as a comparatively rare condition. Still, you must not forget in the cases of osteomyelitis which you meet with in future that this affection is not always a scrofulous one, but may also be due to congenital syphilis. On the other hand I must warn you against being too ready to regard epiphysial swellings as syphilitic, especially those at the lower ends of the radius and ulna, even when other suspicious symptoms are present; they may be due to rickets, especially in infants who have passed the first half year. In these cases the enlarged epiphyses are unaffected by mercurial treatment, while the essentially syphilitic symptoms disappear.

Child of 7 months, brought 29th January, 1876. Well-nourished, pale. Coryza since birth. Eight weeks after birth a macular eruption, cured by baths (?), but always returning. Now, slight roseola on the face, head, hands, and feet. Numerous condylomata on the inner surface of right thigh, round the anus, on scrotum and nates. For some weeks, marked enlargement of lower epiphyses of bones of the lower arm on both sides. Cranial sutures still open with very soft borders. Epiphysial swelling at the junction of the costo-chondroid articulations. Mercurial treatment. On 17th February everything recovered from, except the swellings of the epiphyses, which remain unaltered.

[1] Taylor, *Syphilitic lesions of the osseous system:* New York, 1875.—Lewin, *Charité-Annalen, Jahrg.* iv.

You must always try in such cases to investigate carefully whether a combination of rickets and syphilis is not present; although this is certainly unusual during the first six months. In the first few months you may have less hesitation in regarding and treating such enlargements of the epiphyses as syphilitic. I cannot regard as of much significance a difference in the form of the swelling (Taylor characterises the syphilitic as having a "sudden, abrupt" commencement); but certainly the fact (which I have often observed) that the epiphysial swelling may occur on one side only in syphilis—which is never the case in rickets—is of importance.

In most of the cases here given you will have noted a difficulty in movement or a complete immobility of the upper extremities, so that when the arms were raised and then let go they fell heavily as if lifeless (syphilitic pseudo-paralysis). The first author, as far as I know, who appreciated this symptom was Bednàr[1], in whose table of 68 cases of hereditary syphilis, paresis of the arms is noted sixteen times, that of the legs once, that of all the limbs twice. His description agrees entirely with the symptoms observed in our cases. Bednàr seems inclined—though he nowhere asserts it definitely—to regard this paresis as a myopathic affection entirely due to a relaxed state of the muscles. I am not able to give a satisfactory explanation of this paralysis. It is certainly not a central affection; but at the same time the view that the immobility is caused by pain is open to doubt. Because in not a few cases of this kind I have not been able, either by passive movements of the affected limb or by pressure on it, to elicit any expression of pain. This much is certain, that in all my cases the diminution of the swelling was rapidly followed by a return of the mobility of the limb. One might, of course, on the contrary lay stress on the fact that Bednàr does not mention enlargement of the epiphyses in any of his cases of paresis, also that in my first case the arm which was not swollen was likewise paretic, and that I have frequently seen paralysis of one arm only while the epiphyses on both sides were markedly enlarged. I can even adduce from my own experience two or three cases in which paresis existed apart from any observable affection of the bone.

[1] *Krankheiten der Neugeborenen u.s.w.* Wien, 1853, iv., S. 227.

Child of 6 weeks, with yellowish-red, somewhat desquamating roseola on the arms and legs, face and body; dark-red glazed desquamating palms and soles; coryza and conjunctivitis. Both arms lay completely flaccid; only the fingers showed some slight movement. Nowhere any swelling of the bones. The mercurial treatment, which had been begun in the University polyclinic on 10th July, 1860, had already by the 16th caused a disappearance of the eruption, and freer movement of the upper limbs.

Child of 3 months, brought to my polyclinic 15th January, 1879. The mother had already aborted 4 times. Arms and legs lying immobile and flaccid, almost since birth. Coryza with "snuffles" and discharge; a few spots of roseola on the face and round the anus. No swelling of the bones. Mercurial treatment. On 4th February coryza and spots cured. Arms and legs freely moved, but the latter cannot be fully extended at the knee-joints, owing to resistance of the flexors. Treatment continued.

Child of 6 weeks, brought 24th July, 1879. Coryza, soles red, glazed, and desquamating. Roseola round the anus. Epiphyses not enlarged. For the last 8 days, arms flaccid and immobile. Every passive movement excites crying. Further course unknown.

Child of 8 weeks. Slight roseola; intertrigo ulcerosa; fissures of the under lips; coryza. Both arms paralysed, flaccid. Epiphyses not swollen. Did not come back.

By the researches of Wegner[1] we are brought somewhat nearer to the explanation of these "pseudo-paralyses" affecting by preference the upper extremities with or without swelling of epiphyses. In syphilitic new-born infants and young children one finds—according to his investigations—almost invariably in the long bones, at the point of junction of the diaphysis with the cartilage of the epiphysis, a morbid process consisting in an excessive proliferation of the cartilage cells, and a retarded ossification of the already calcified substance. Along with this the formation of new blood-vessels in the bones is either altogether arrested or takes place very imperfectly; and from want of nutrition the cells are gradually destroyed by fat-metamorphosis and shrivelling. The result of this process is seen on section, as a narrow, somewhat jagged line of yellowish or orange colour running along the margin of the epiphysial cartilage. According to Wegner it is formed by necrotic tissue and separates the diaphysis from

[1] *Virchow's Archiv*, Bd. 50, S. 305.

the epiphysis, and may lead to a complete separation of the latter by an "inflammatory suppurative complication." The process always makes its appearance in several places at once, particularly often at the lower end of the femur, in the bones of the legs and forearms, and in the ribs; and sometimes in all the long bones. Meanwhile the ossification of the epiphysial cartilage proceeds irregularly, and the cartilage cells—which in healthy bones are arranged in rows—are partly put out of order or are completely disintegrated and replaced by groups of small cells. These observations were confirmed by Waldeyer and Köbner;[1] but they, as also Taylor, regard the yellow zone not as a necrobiosis caused by deficient vascularity, but as a gummatous process due to the enormous proliferation of new cells which, by compressing the vessels, occasions the death of the intermediate tissue and the consequent separation of the epiphysis from the diaphysis. Whatever its correct explanation[2] may be, the important fact (from a clinical point of view) remains, that we have here a morbid process at the epiphysial line which, although it certainly occasions recognisable symptoms during life in only a very small proportion of the cases (swelling, pain, immobility), yet has an influence on the mobility of the affected limbs which, even where other symptoms are wanting, must not be underestimated. A separation of the epiphysis observable during life is rare, and is manifested by abnormal mobility at the epiphysial line and an unusual "dangling" of the hand (Köbner and Waldeyer). I have myself been able only in one instance[3] to make out crepitation at the affected place. Moreover the change described at the line of junction of the epiphyses does not always affect all parts equally. In a child of two months, the epiphyses of whose forearms were distinctly smaller during life, it was well-marked in these situations only while in the other bones it was merely indicated. In a child of thirty days old there was but little of it to be seen in any of the bones examined.[4] Perhaps in this

[1] *Virchow's Archiv*, Bd. 55.

[2] According to Haab and Veraguth (*Virchow's Archiv*, Bd., 84, Heft 2) we have to do, chiefly, with an inflammatory process in the cartilage, which causes fissures to appear in it.

[3] Troisier, (*Union méd.*, 1883, No. 104) and Kremer ("Beitr. zur syphil. Epiphysenlösung": *Dissert.*, Berlin, 1884) describe such cases.

[4] According to Köbner and Waldeyer even in the cases where there are no naked-eye change in the epiphyses, these may be certainly recognised by the

case the mercurial treatment which had been carried on for twenty days with marked success (all eruptions were already cured), had acted beneficially on the bones.

I am not myself quite sure of having ever observed the affection of the joints whether following on disease of the epiphyses or not. On the other hand, some writers[1] mention having seen purulent inflammation of joints or periarticular abscesses as the result of congenital syphilis. Without denying the correctness of their observations, I must point out that, at least in a number of the cases, they may have had to do with inflammation of the joints accidentally complicating hereditary syphilis. Of the subacute form of congenital syphilitic joint affections, of which Somma[2] describes six cases, I have hitherto, in spite of my large amount of clinical material, met with only one case, and that not altogether free from doubt. Still, I admit that I have not yet directed my attention to the joints as carefully as I consider necessary after these recent communications.

Bouchut and Parrot[3] have also found the diaphyses of the long bones often unusually dense and hard, and frequently the seat of periostitic deposits. Wegner has in rare cases found a gummatous periostitis on the inner side of the cranial bones; or little gummatous nodules in the pericranium. The nature of the following case of bone-disease affecting the sternum does not seem to me quite clear.

> In October, 1878, an apparently healthy and thriving child of 8 weeks was brought to the polyclinic. No signs of syphilis. In the region of the ensiform process there was a wound the size of a shilling with a grey coating; in the middle of it a fistulous

---

microscope.—Lomer (*Zeitschr. f. Geburtsh. u. Gynäcol.*, x., H. 2, 1884) was unable to find them in 13 out of 43 macerated fœtuses, some of which were undoubtedly syphilitic.

[1] Güterbock, *Langenbeck's Archiv*, Bd. xxiii., Heft 2 and Bd. xxxi., Heft 2.—Schüller, *Ibid.*, Bd. xviii., Heft 2.—Parrot.—Heubner, *Virchow's Archiv*, 84, Bd., 1881.—*Klin. Wochenschr.*, 1884, S. 548.

[2] *Su di una forma morbosa articolare per sifilide ereditaria:* Napoli, 1882. The characters of it, according to Somma are:—Very early commencement; cachexia; crying on movement; fever (to 102·2); enlargement of several joints (especially the knee-joints) with slight local redness and rise of temp. Duration 18 days to 2½ months. Recovery possible under specific treatment (inunction of ung. hydrarg. and pot. iod.). In two cases there was found at the post-mortem, inflammation of the synovial capsule, sero-purulent exudation into the cavities, necrosis of the cartilage, hyperæmia and rarefaction of the neighbouring bones. Gummata are also mentioned.

[3] *Archiv f. Kinderheilk.*, Bd. ii., S. 433.

opening, from which on expiration there issued pus along with a few air-bubbles, which had evidently entered from outside. A probe touched rough bare bone (sternum). According to the mother's statement, an abscess had formed a week after birth and had opened. I did not see the child again till 21st February, 1879. The fistula had completely healed after the exfoliation of a piece of bone; but the child now had coryza, fissures of the lips and angles of the mouth, spots of roseola, and erosions round the anus and on the genitals.

Whether the necrosis of the sternum in this case was really to be regarded as a manifestation of syphilis I shall not venture to decide; because I have never hitherto observed a specific bone disease coming on soon after birth and preceding all other symptoms of the disease by months. Also, the bone affection was recovered from without specific treatment.

Infantile syphilis does not limit its action to the skin, mucous membranes and bones. Other organs also, as in adults, may be affected; amongst these the testicles and liver may be specified as parts when implication is discoverable during life as well as post-mortem. The affection of the testicle was partially unknown until very recently. Hennig and Taylor mention it only incidentally; and Désprès[1] was the first to describe carefully three cases, in children of from seven months to three years of age, one of whom was examined post-mortem by Cornil and found to have hypertrophy of the tunica albuginea with interstitial orchitis and epididymitis. In the comparatively short time since 1874 I have myself met with at least twelve cases, of some of which I have already elsewhere[2] published accounts. Never neglect, therefore, in every case of infantile syphilis to examine the testicles carefully. The testicle thus affected is more or less enlarged, hard and firm; likewise somewhat uneven and nodular. The size varies from that of a hazel to that of a chestnut. I have found both testicles affected in four cases, the left alone in four, and the right alone in two. The youngest child was three months, the oldest was suffering from a relapse of syphilis and was two and a half years old. Only one case was examined post-mortem.

Boy of 2½ years, brought to the hospital in the end of September, 1876, with mucous papules at the anus and psoriasis

[1] *Bullet. de la soc. chir.*, 1875.

[2] *Deutsche Zeitschr. f. pract. Med.*, 1877. No. 11.

syphilitica. Both testicles markedly enlarged and nodular. Treatment by inunction (grs. 10 ung. hydrarg. daily). After thirty inunctions, all the symptoms disappeared, except that the testicles remained unchanged. Death on 25th December from cholera infantum. *P.-M.*—Both testicles very large and firm. The microscope showed an extensive hypertrophy of the interstitial connective tissue in the testicle, most marked in the corpus Highmori. Gumma nowhere to be discovered.

There was therefore in this, as in one of Désprès' cases and in others recently observed by Hutinel,[1] interstitial orchitis, and partial epididymitis; and of course when this has gone on to the formation of fibrous tissue, it will resist all treatment. Only in an early stage you may expect that resolution may occur, although perhaps not always complete; and of this I have fully convinced myself in four cases. In just the same way the liver may also be attacked by an interstitial inflammation, with or without formation of gummatous nodules; but this in a number of cases is only recognised post-mortem.[2]

A girl of 7 days, illegitimate, born in the Charité. Father syphilitic. On examination of the child we found roseola, and psoriasis of the palms and soles, thighs, legs, and nates. Extreme atrophy, no enlargement of liver. Death from collapse, 25th November, 1875. *P.-M.*—Interstitial hepatitis; liver somewhat enlarged, very tough, smooth. Acini not visible; whitish bands consisting of connective tissue passing through the parenchyma in all directions. Cortical substance of kidneys extremely firm. Hæmorrhages in fundus of stomach, both outside and in; its mucous membrane covered with a coherent membrane-like layer of blood-stained mucus. The yellow zone in the epiphyses of several of the bones; periostitic deposit on the right humerus. All diaphyses extremely hard.

While in this case the interstitial hepatitis was only revealed by the post-mortem and even the hæmorrhagic catarrh of the fundus of the stomach—possibly a result of engorgement of the portal vein—caused no symptom; in other cases an enlargement of the liver was noticeable, which confirmed the diagnosis.

Felix L., 3 months old, was affected at the age of six weeks with a macular eruption which gradually spread over the whole body. In places blebs of the size of a pea occurred, filled with

---

[1] *Revue mensuelle*, 2, 1878.

[2] *Cf.* v. Bärensprung, *Die hereditäre Syphilis:* Berlin, 1864.

turbid fluid. Intertrigo on the scrotum and in the neighbourhood of anus. About four weeks previously, also coryza and hoarseness. On 15th March, 1864, I discovered a considerable enlargement of the liver, along with all the usual symptoms of congenital syphilis. The liver reached down to the level of the umbilicus, where its sharp margin could be distinctly felt, and was visible at each respiration through the wasted abdominal walls. Surface smooth; not tender to touch. The liver-dulness extended on the left side right over to that of the spleen. Mercurial treatment, with no results. The atrophy increased and death ensued on 25th. At the post-mortem the liver was found to be considerably enlarged, with numerous whitish-yellow patches and bands of various sizes scattered through it. Microscopic examination of these by Prof. Klebs showed the appearance of interstitial hepatitis. Spleen and kidney normal on microscopic examination.

Child of 9 weeks (7th February, 1881). Moderate jaundice since birth, sclerotic and mucous membranes affected. Fæces and urine contain bile. Liver prominent and smooth. No signs of syphilis. Advancing atrophy. Treatment with calomel unsuccessful. Collapse and death, 28th February. *P.-M.*—Liver very large and thick, olive-green and tough. Acini separated from one another by white bands of connective tissue, the immense number of which is better seen under the microscope (interstitial hepatitis). Scattered effusions of blood in the mucous membrane of the stomach and bowel. Characteristic syphilitic zone in all the epiphyses of the ribs. No signs of syphilis elsewhere. Soon after this case, I had another identical one which was further remarkable, because the mother had already lost three children from this same liver-affection with jaundice.

In these cases there is usually only a moderate degree of jaundice or none at all; but if the process of induration affects not only the interstitial tissue but also the porta hepatis, the jaundice may reach a very high degree and present a greenish tint. I have seen one such case, which must have arisen during intra-uterine life,[1] in a child ten weeks old, who since birth had had a hard uneven liver, intense jaundice with quite colourless motions, and an enlarged spleen, and whose gall-bladder and bile-ducts were found post-mortem to have been entirely transformed into thick fibrous masses filling the portal fissures. It is

[1] *Cf.* Beck's case (*Prag. med. Wochenschr.*, 1884, 26):—A fœtus of eight months. Fibrous masses in the liver, on the bile-ducts and gall-bladder, and in the pancreas, with miliary gummatous deposits, interstitial orchitis and epididymitis.—de Ruyter, "Einige Fälle von Syphilis congenita": *Diss.*, Berlin, 1885.—P. Meyer, "Aus der Kinderpoliklinik der K. Charité zu Berlin." *Berliner klinische Wochenschr.*, 1886, No. 16.

only in the minority of the cases, as far as my experience goes, that interstitial or gummatous disease of the liver can be made out clinically; and even at the post-mortem they may in many cases be overlooked on merely naked-eye examination. Ascites also, which is such a usual symptom of interstitial hepatitis (cirrhosis) is almost always absent in these cases. The case published by Depasse,[1] in which the fluid in the abdominal cavity communicated with the tunica vaginalis and was evacuated by these punctures (one through the scrotum), is therefore all the more remarkable. This case is also noticeable owing to the success of the specific treatment (which usually has no effect) even although in the 8th year the liver was still considerably enlarged.

The spleen is often also affected in congenital syphilis by hyperplasia, induration, and perisplenitis adhesiva; and I have myself repeatedly seen it more or less considerably enlarged in such children, and once in a very atrophic child of six weeks, and again in one of two months, with roseola and palpable enlargement of the liver. One must not, however, forget that the spleen is not unfrequently found to be hypertropied in infants who are not syphilitic, and, therefore, the combination of syphilis and enlargement of the spleen may in many cases be accidental.[2] Hypertrophy of the connective tissue occurs also in the kidneys, supra-renals and pancreas; but has no more clinical interest than the gummatous nodules which are sometimes found in the thymus, in the lungs, and even in the heart. Abscesses of the thymus-gland on which P. Dubois laid great weight I have seen twice, in the form of multiple collections of pus scarcely the size of a pea. The children presented at the same time many pemphigus-bullæ, especially on the palms and soles, and they died in the first weeks of life.

The implication of the nervous centres, especially of the brain and its vessels, by syphilis has aroused much interest in our time; but my own experience seems to indicate that it occurs much more frequently in adults than in children.[3] In rare cases I have seen contractures improved or cured by specific treat-

[1] *Revue mens.* Août, 1886, p. 360.

[2] Haslund, *Archiv f. Kinderheilk.*, Bd. iv., S. 297.

[3] Chiari (*Wiener med. Wochenschr.*, No. 17 & 18, 1881) describes a case of Endarteritis syphilitica of the vessels of the brain in a child of 15 months with hereditary syphilis.—Barlow (*Lancet*, 1877) gives a similar case.

ment. My first case of this kind[1] was that of a boy aged fourteen months, who was brought to my polyclinic on 24th Nov. 1867.

> On examination he was found to have contracture of the right arm at the elbow-joint, of the fingers of the right hand, and of both lower extremities at the knee-joints. Unable to stand, sit, or grasp with the right hand, Biceps brachii and flexors of the legs extremely tense. Every attempt to extend the limbs caused violent crying. The child had also papules round the anus and on the scrotum, excoriations of the alæ nasi and of the angles of the mouth, coryza, enlargement of the clavicular and axillary glands. The child was said to have suffered for months from severe coryza, from an "eruption of blebs" and ulcers; and, when three weeks old, from epileptic fits for several days. The contractions were said to have gradually developed after these. When the child had taken mercury for about a month it could on 23rd December open the right hand and also bend the knee. Gradual improvement till 3rd February, 1868. On 27th, change to pot. iod. On 30th March, began to walk and to use right arm. Further progress unknown.

The influence of the anti-syphilitic treatment is here unmistakeable. Still, it is questionable whether the contractures should really be regarded as a cerebral affection and connected with the fits which the child had formerly had, or as a myopathic affection quite independent of the nervous system and caused by an interstitial myositis such as occasionally occurs in syphilitic adults. That the latter may occur in congenital syphilis seems to me to be proved by the following case:—

> In a syphilitic child of 4 months (brought to the polyclinic October, 1874), there was a stiff contraction and hardness of the flexors at the back of both thighs, so that the limbs were held continuously in a state of semi-flexion. The leg could be only partially extended at the knee. The use of mercury for several weeks brought about complete recovery; first of the skin eruptions, and finally of the contractions also.

I have never been able to observe essentially cerebral symptoms in infantile syphilis,—neither the chronic meningitis described by Somma,[2] nor paralysis of one or more extremities, nor convulsive seizures. And although such things may occur, it is still very doubtful whether one is justified in attributing them to syphilis. The following is a case in point:—

[1] *Beiträge zur Kinderheilk., N. F.:* Berlin, 1868, S. 421.
[2] *Clinica pediatrica di Napoli*, 1877.

In a child of 2 years (admitted to one of the children's wards 6th November, 1877) there was—along with osteomyelitis—an unusual psychical condition, alternating precocity and stupidity, but without any interference with motility. At the *P.-M.* (after death from diphtheria) we found several nodular tumours about the size of a cherry under the pia mater, and in different parts of the cerebrum and cerebellum. They were of a grey colour, and transparent at the periphery; the centre partly fatty, partly calcified. A similar deposit was found in the upper part of the left kidney. Since tubercle was nowhere present and periosteal deposits were found on both tibiæ, I was inclined to regard the brain-tumours as syphilitic gummata; and they were considered such on being examined at the pathological institute of the Charité.

Considering that the diagnosis between gummata and tubercle cannot be made with perfect certainty with the microscope alone, and that the clinical symptoms with the progress of the case and finally the result of the treatment have a much higher value from a diagnostic point of view, we must be very sceptical in judging of such cases. At any rate it seems strange that, in spite of the great number of children with congenital syphilis I have seen, I have practically never been able to find cerebral symptoms which could with certainty be referred to syphilis. Any connection between chronic hydrocephalus and congenital syphilis seems to me extremely improbable on account of the ineffectiveness of mercurials in the former disease. Even the case of "diffuse insular sclerosis" which Buss[1] has described seems to be by no means beyond a doubt as far as its connection with hereditary syphilis is concerned.

In other regions of the vascular system changes have occasionally been found in new-born children, which recall "the syphilitic affections" of the blood vessels of the brain. Thus, Schütz[2] describes the small arteries of the kidneys and skin as being much narrowed, their walls considerably thickened by hypertrophy of the muscular coat and adventititia; and he ascribes to this the numerous little ecchymoses which, in his case of a premature child, he found in the skin, subcutaneous connective tissue, muscles, kidneys and other parts. Fischl's[3] researches, however, render it very doubtful whether

[1] *Berl. klin. Wochenschr.*, 1887, Nos. 49 u. 50.
[2] *Prager med. Wochenschr.*, 1878, Nos. 45, 46.
[3] *Arch. f. Kinderheilk.*, viii.

these vascular changes are really caused by syphilis. He regards this state of the small arteries in new-born children as the normal condition and thinks it has nothing to do with hæmorrhage. Mracek[1] on the other hand says that in children with syphilis hæmorrhagica he has found the walls of the small and medium-sized veins thickened by a proliferation of their nuclei, and the lumen narrowed or even obliterated. While the matter is thus undecided we cannot, in the meantime at least, recognise any real anatomical foundation for the view which Behrend[2] has endeavoured to advance, viz., that there is a hæmorrhagic form of syphilis neonatorum.

The progress and termination of the case in congenital syphilis depends, according to all experience, less on the nature of the symptoms than on the state of the patient's nutrition. Syphilitic infants fortunate enough to receive their natural nourishment from the mother or wet-nurse, usually thrive well when treated specifically, and have the best prospects of complete recovery. But all hand-fed children, especially such as have from birth been weak and atrophic, I consider to be in great danger; indeed these latter may almost be given up for lost. While in private practice, and even in the polyclinic, out of a very large number of syphilitic children I have lost only a few, and that from chance complications, in the children's wards of the Charité, where every one of the cases was extremely atrophied, almost all I have seen ended fatally. Not uncommonly death came quite suddenly. Trousseau has already drawn attention to this; but in my opinion it is nothing very strange, since sudden death is a common enough event in atrophic children. Under propitious circumstances, the disease often takes a favourable turn with surprising rapidity. One is astonished to see eruptions, condylomata and bony swellings diminish within five or six days under the influence of mercury, and after a few weeks disappear entirely. But I should here warn you against over-estimating the results of your treatment. Recurrence of the disease is extremely common in infantile syphilis; and, especially in polyclinics where the children are so

[1] *Jahrb. f. Kinderheilk.*, xxvii., S. 191.

Vierteljahrschr. f. Dermatologie und Syphilis, 1884. I will only remark that among Behrend's cases there are two in which there was an enlarged spleen with purpura; and these often occur together where there is no syphilis. *Cf.* also Petersen, *ibid.*, 1883, S. 509.

often removed from further observation on the first disappearance of the symptoms, one has opportunities of convincing oneself of this fact.

A child of 6 weeks, brought 7th January, 1874, with many symptoms of syphilis. Recovery towards end of February, under mercury. Brought again 10th April, with symptoms of recurrence of the disease which have existed three days. Recovery on 28th. Recurrence on 18th June.

Boy of 2 years, born of a syphilitic mother, all of whose children were infected. Infantile syphilis in the 2nd month. A few weeks later treated in the polyclinic for erosions at the angles of the mouth and on the tongue. Recurrence on 15th May, 1874:—For 8 weeks condylomata at the anus and on the dorsum of the tongue, which towards the back appears hard, infiltrated and of a dark-red colour, and towards the front is covered with a greyish-white coating. At the end of June, recovery under mercury. On 14th November, recurrence of the condylomata at the anus. On 9th January, 1875, recurrence again, requiring new treatment.

Girl of 5 years, with mucous papules at the anus, and enlargement of the inguinal glands. First outbreak of syphilis at the 5th week; 2nd, at 1½ years; 3rd, at end of 5th year.

One should not, therefore, at once discontinue the treatment on the disappearance of all symptoms, but should always carry it on for some weeks. But even this does not remove all danger of recurrence. In most cases, however, one succeeds in curing the disease completely within the first or at least the second year; and I have gathered a sufficient number of observations from private practice to be able to assert that by this time the thing is really at an end. You must, however, always be prepared for a fresh outbreak of the disease, even in the later years of childhood. And under these circumstances doubts may arise as to whether we have a recurrence of congenital syphilis, a direct infection, or a so-called "syphilis tarda" (to which I shall return later). But even in the cases where the disease has been thoroughly cured by continuous treatment from the very beginning, there yet not uncommonly remains a derangement of the constitution favouring the development of rickets. I have frequently seen this disease come on after recovery from congenital syphilis in children situated in the most favourable circumstances and nursed with the greatest care. But I must protest against the incomprehensible opinion of Parrot, who regards rickets as invariably the result of syphilis.

The difficulty of establishing with certainty the origin of infantile syphilis is often very great, although its diagnosis is so easy. All cases which have arisen within the first two months of life, must, with extremely few exceptions, be regarded as hereditary. I have already stated (p. 61) that hereditary syphilis sometimes appears in the form of pemphigus, even during the first days of life; and some of the cases already given show that as early as the first two weeks other syphilitic skin affections and coryza may make their appearance. Much oftener, however, the children present no striking peculiarity during the first four to six weeks; and it is only later that symptoms begin to be observed. After the second—or still more after the third month—it is rare for them to appear for the first time;[1] and when they occur still later, it is always doubtful whether it is not a case of recurrence or of direct transmission. The latter is certainly not easy to establish; and, especially under those circumstances which render confession on the part of the parents difficult or impossible, an attempt is often made to turn the physician's thoughts from the subject of heredity, and deceive him by false statements about a syphilitic wet-nurse or other attendant having infected the child. I by no means deny the possibility of such infection; yet, out of the cases of this kind which I have myself met with, there has not been a single one so certainly proved that I was able absolutely to exclude a hereditary origin. In poor families, however, I have certainly observed the direct infection of infants by syphilitic women living in the same house and, consequently, brought much into contact with them; perhaps sometimes due to the use of sponges and other toilet articles in common. On the other hand, the infection of the child during birth, from the genital organs of the mother being affected with syphilis (syphilis adnata), which was formerly often assumed,—is very doubtful: for example, Trousseau's case, in which he ascribed an "indurated chancre" on a child's nates to contact with the ulcerated vulva of the mother. I have not myself seen any case of this kind; nor yet one of infection by vaccination which in our time has raised so much dust, under the name of "syphilis vaccinalis." Since the contagiousness of secondary

[1] Roger found, in 249 cases, the earliest symptoms 118 times in the first, and 217 times before end of third month; but only 32 times after that.

syphilis has been proved beyond doubt, one can certainly no longer dispute the possibility of the transmission of the disease by inoculation with vaccine lymph derived from a syphilitic child, whether any blood is mixed with it (Viennois) or not. And it cannot be denied that many of the cases of vaccination-syphilis which that author gives seem to prove it. Still, the point remains a matter of controversy, and I do not consider myself called upon here to give a definite judgment upon it—all the more because, as I mentioned before, I have never myself met with a single well-authenticated case. I have, indeed, seen many cases in which, after vaccination, ulcers appeared at the sore, and various eruptions, which might very easily have been mistaken for syphilis by inexperienced and superficial observers, but which had no connection whatever with it. Of the frequency of these errors I am perfectly convinced; and I would refer, as a positive proof of this, to the work of Joukoffsky[1]; he saw fifty-seven children who had been vaccinated from eleven syphilitic infants remain absolutely free from the disease. I should also remind you that syphilis does not interfere with the regular development of the vaccine vesicle; but that where hitherto latent it may become manifest owing to an injury, such as vaccination is,—and a false assumption of transmission by the lymph may thus arise. I am even less afraid of transmission by the milk of a syphilitic nurse, so long as her nipple is healthy. At the same time, one would of course be as unwilling to choose a suspected nurse as to make use of vaccine lymph from a child which then or previously had presented symptoms of syphilis.

With rare exceptions, therefore, all the cases of syphilis occurring during the first months of life, are to be regarded as hereditary. The study of this heredity has long been pursued with especial zeal[2]; and if in spite of this medical writers at the present day are as yet by no means unanimous and differ widely on many points, the fact merely proves how difficult it is to get rid of all doubt in matters which from their very nature can only become known by the confession of those interested.

[1] *Oesterr. Jahrb. f. Pädiatrik*, v. 2, S. 139.

[2] Köbner, *Klinische und experimentelle Mittheilungen aus der Dermatologie und Syphilidologie:* Erlangen, 1864.—Kassowitz, "Ueber Vererbung und Uebertragung der Syphilis," *Jahrb f. Kinderheilk*, Bd. xxi., 1884, S. 53.

Every day brings us new examples of the fact that, especially in syphilis, those concerned are but seldom to be fully trusted, and the physician, in spite of the utmost care, is here liable to vexatious deception. I have myself met with cases where not only was the diagnosis of congenital syphilis beyond doubt, but the post-mortem, also, gave the fullest confirmation of this—and yet both parents persistently denied ever having been syphilitic. We know for certain that syphilis may be inherited from the father as well as from the mother. The father transmits the disease immediately through the semen with which he impregnates his wife,—the mother, through the ovum from which the fœtus developes[1]; in this case, the parents must be the subjects of secondary syphilis. Primary affections can only have an influence in infecting the child in so far as they lead to the development of secondary symptoms in the mother during her pregnancy—an origin of congenital syphilis which by many writers (*e.g.* Kassowitz) is positively denied. I do not consider it by any means settled whether they are right in doing so, or whether an infection of the fœtus by the blood which nourishes it is possible in the case of a mother becoming syphilitic during pregnancy; but I think the latter is very probable. Those who deny such transmission by the blood naturally discredit the possibility of a non-syphilitic mother becoming infected through the blood of her fœtus which derives its syphilis from the father; others hold this to be certainly possible. Hutchinson and Fournier allege from their experience that women who have married syphilitic men frequently do not become infected until they conceive and not so long as the marriage remains unfruitful. Some recent observations of Behrend[2], also, seem to be in favour of the view that such a "placental infection" does take place sometimes, but is by no means a necessary occurrence. Be that as it may, this much at any rate is certain—that syphilitic mothers are exceedingly liable to abortion, or to give birth prematurely to non-viable infants, whose epidermis, often macerated and detached, is sometimes mistaken for the product of a fœtal pemphigus. This tendency

[1] The streptococci in the capillaries described by Kassowitz and Hochsinger (*Wien. med. Blätter*, 1886, 1—4) are regarded by most authorities as not pathogenetic.

[2] *Berl. klin. Wochenschr.*, 1881, S. 107.

to abortion is due to endometritis decidualis, thickening of the placenta, or circumscribed gummatous growths in it (Virchow), —perhaps also to atheroma or endarteritis syphilitica of the umbilical vein (Winckel). The knowledge of this is of importance for diagnosis, because in cases where the presence of congenital syphilis is doubtful, it helps to turn the scale in its favour.

By time, and by repeated specific treatment, the disease in the parents may be weakened or temporarily cured. And this explains the fact that at the earlier period of such marriages the tendency to abortion is strongest, and gradually decreases as time goes on; also, that the first-born children are apt to be the most severely affected, and the later ones may be quite healthy. Not uncommonly we also observe a remarkable alternation of healthy and syphilitic children, which can only be explained by the fact that the syphilis in the parents manifests itself afresh periodically, and at other times remains in a condition of latency which does not endanger the health of the fœtus. In this manner the possibility of hereditary transmission may continue for a very long time. Kassowitz estimates it at ten to fourteen years; but the following case of my own shows that even twenty years may have passed.

> The father of the child at the time of his marriage had a chancre not yet completely healed. The first child, born a year after the marriage, was said to have suffered repeatedly from swellings on the tibiæ; and I myself observed in the same child, when she had grown to a girl of 17, another extensive periostial swelling on the left humerus. The mother herself had suffered repeatedly during her twenty years of married life from suspicious sore throats and obstinate ulcers in the neighbourhood of the knee-joints, which always required treatment with pot. iod. and decoct. sarsœ co. (Germ. P.) to remove them. During this long time she bore two other children who were quite healthy; but subsequently she had several abortions. In the 20th year of her married life she was delivered of a boy who, 14 days after birth, was affected by well-marked manifestations of congenital syphilis, and had to be subjected to a long course of mercury. Later, he became extremely rickety, suffered frequently from convulsions and laryngeal spasm, but in the end—thanks to first-rate nursing—grew up a healthy youth.

I am at present uncertain whether it is possible to recognise from the form of infantile syphilis whether it originates from the father or mother. The opinion of Bärensprung, Hecker and

Keysel[1], that syphilis of the internal organs—especially of the liver—proves heredity from the father's side, appears to me by no means certainly established, for one has to bear in mind the insuperable difficulties in the way of our obtaining a reliable history here.

I come now to the treatment of the disease. I may sum up the result of my large experience in this department in this short sentence—the only reliable remedy in infantile syphilis is mercury. Its action, as I have already mentioned, is often really wonderful, and its rapidity extremely surprising. Neither iodide of potash nor iodide of iron, which are recommended by many, are comparable to mercury. Of its preparations I prefer to all others, for patients of this early age, calomel and mercurous oxide (Germ. P.) in doses of gr. $\frac{1}{7}$—$\frac{1}{4}$, morning and evening. The latter occasionally—and especially at the beginning of the treatment—causes vomiting. Any other mode of giving the mercury—such as the mercurialisation of the nurse or even of a milk-giving animal—I consider inadmissible; all the more so, as it is by no means certain that the mercury is transmitted by the milk. At any rate, certain experiments in this direction undertaken by Kahler[2] showed that the milk of three mothers under treatment by inunction was completely free from mercury. Inunctions of mercurial ointment or subcutaneous injections of perchloride of mercury are only indicated where there are no extensive syphilitic eruptions, or where intestinal complications (diarrhœa and vomiting) forbid the internal use of the drug. All the patients with whom I have used inunction were already more than two years old, and were suffering from a relapse of syphilis, which generally manifested itself more by condylomatous formations than by extensive skin eruptions (inunction of grs. x.—xx. unguent. hydrarg. daily). I have injected perchloride of mercury subcutaneously in these cases, with good results; I shall return to this in considering the syphilis of older children. Perchloride of mercury baths (grs. xv. to a bath) I have used frequently, but with no constant effect. I therefore recommend them only for those cases in which advanced atrophy, vomiting or diarrhœa make the internal administration of mercury inadvisable.

Condylomatous excrescences are to be dusted with calomel, or,

[1] *Bayer. ärztl. Intelligenzbl.*, 1876, No. 21. [2] *Aerztl. Correspondenzbl.*, 1875, No. 23.

if they are already ulcerated, painted daily with a solution of nitrate of silver (grs. xvi to the ℥i). I also recommend this latter to you for the nasal mucous membrane, should the coryza obstinately resist internal remedies. In most cases, however, internal treatment suffices to cure it.

The extreme importance of the natural method of nourishment for syphilitic infants has been already mentioned. Any artificial method is objectionable for such children, although unfortunately it is often unavoidable; and it may also be well borne as long as we have to do with strong children.[1] It will be readily understood that if the mother herself is syphilitic she need have no hesitation in nursing her own child. It is another matter when there are absolutely no signs of the disease to be found on the mother, and when any previous syphilitic affection is denied. Under these conditions—which are not at all common—the mother should be allowed to nourish her child only if its lips and mouth present no morbid appearances (rhagades, or condylomata). The same holds true in the case of a wet nurse; for there can be no doubt that such a child may transmit syphilis to the excoriated nipple of a healthy nurse, and that specific ulcers on the breast may arise in this way and be followed by secondary symptoms. Even the secretion from coryza must not be altogether disregarded as an unimportant matter in deciding whether the child shall be allowed to take the breast (Roger). Certainly the observations of Günsburg[2] seem to be quite against such an infection, since out of thirty-one wet nurses of syphilitic children (one nurse within two years suckled as many as eleven), he did not see a single one become affected. Thence he concludes that congenital syphilis is never transmitted to the person who suckles, and that all the cases in which this is said to have taken place are to be explained by the fact that the children were suffering from acquired syphilis. This opinion, however, seems to me to be somewhat forced; and as cases have been observed of healthy wet-nurses being infected by children who were indubitably suffering from congenital

[1] In the "Hospice des enfants-assistés" in Paris, experiments have been made recently (at Parrot's suggestion) in nourishing syphilitic children with asses' milk—the children sucking the ass's teat; and the results of this were much better than those of hand-rearing. *Cf.* Wins, "L'allaitement à la nourricerie de l'hospice des enfants-assistés," *Thèse:* Paris, 1885.

[2] *Oesterr. Jahrb. f. Kinderheilk.*, 1872, ii., S. 169.

syphilis, I consider it extremely problematical; and therefore I advise you to exercise caution. To my thinking, the physician is bound to point out to the wet-nurse the possibility of an infection. It then lies with herself to decide whether she will expose herself to this danger for the sake of remuneration. In this way, certainly, the most awkward family secrets may be disclosed, and the physician accused of indiscretion; still, I think that all these considerations must not induce us to expose a healthy nurse to the risk of syphilitic affection without her knowledge. It is not, of course, necessary to use the name "syphilis" to the nurse; it is enough if one explains to her that it is an infectious skin eruption. Almost all nurses are quite willing to enter into the engagement on this understanding and in most cases they remain free from syphilis. I myself, at any rate, have as yet never known of any nurse becoming infected in this way, although several of the children nursed were affected in a high degree with congenital syphilis. The greatest cleanliness and, still more, the most careful attention to any excoriations occurring on the breast, are to be impressed on the wet-nurse as a duty. The child may have difficulty in sucking owing to fissures on the lips and severe coryza; still, I have never seen danger in the matter of nutrition arise from this.

Finally, a word or two on the physician's conduct to the parents. While in poor practice and in that of the polyclinic a candid statement by the physician has scarcely ever any bad result, in the upper classes of society such a statement is apt to lead to serious consequences in the family. I therefore advise you if you are not confided in spontaneously, and if you are sure that the mother is quite innocent, to take the father only into your confidence. Fortunately the disease is so characteristic that confession on the part of the parents is unnecessary for diagnosis, and the proper treatment may be entered on at once. Still it is always a matter of the greatest importance to ascertain the parents' state of health, for it is only by thorough specific treatment that we can prevent the subsequent offspring from becoming likewise syphilitic.

In spite, however, of this characteristic group of symptoms, cases do occasionally occur in which even the most experienced physician is unable to make the diagnosis of syphilis with

certainty. In such cases it would be highly indiscreet to agitate the parents by obscure hints and questionings. Suppose, for example, one were to observe intertriginous redness about the anus and genitals, with superficial rounded excoriations here and there in the middle of it. This intertrigo, in spite of cleanliness, gradually spreads over the lower part of the back or over the greater part of the body, while the reddened skin becomes covered with yellowish-white scales, consisting of desquamated epithelial cells mixed with sebum. Or there may arise in the intertriginous folds of the skin—especially in the inguinal region—deep elongated ulcers covered with a greyish-white coating. Perhaps, also, coryza or red spots in various situations may appear—still further unsettling the diagnosis. In most cases of this kind you will be guarded against error by the fact that the lips and the angles of the mouth remain free. But it will do no harm whatever if, to quiet your professional conscience, you begin mercurial treatment; and this will very soon show whether there is any syphilis present.

I close this chapter with a few remarks on the syphilis of older children, of which I have seen a considerable number of cases, especially in my department in the hospital.

The thirty-nine children, on whose cases I have founded the following description, were between two and fourteen years of age, and (with the exception of eight) were all girls. On the most careful questioning it was found with certainty in only six cases that the syphilitic symptoms were to be regarded as due to a recurrence of congenital syphilis which had already shown itself in the first months of life. In all other cases no connection of this kind could be certainly traced; and therefore, under the circumstances, we were left in doubt whether we had to do with a hereditary disease or with one acquired by later infection and intentionally concealed by the relatives. In any case I should rather acknowledge this doubt than assume a so-called "syphilis tarda,"—a form which is said to be hereditary, although it only makes its appearance for the first time in older children between the eighth and twelfth years or even later. That such syphilis tarda may possibly occur I shall certainly not deny, for the theory has the support of conscientious observers; but I have never in my own experience met with a single indubitable case of it. Further, I should recognise as such only

a case in which I myself had been able to verify, by continuous observation from birth, the absence of all syphilitic symptoms in early life; and, at the same time, the absence of syphilis in the parents. For the statements of the latter are almost always unreliable, and often even intentionally misleading.

In eight girls, between four and twelve years, the symptoms could be referred with perfect precision to an assault, or at least to an attempt at one; but the statement of the eldest of these children (12 years) that she was assaulted by a man while asleep on a stair seemed very questionable, owing to the extremely bold air of the patient. Only in two cases was the hymen found torn; in all the others it was intact, so that a complete immissio penis could not have taken place, although the whole neighbourhood of the hymen as far as the inner surfaces of the labia was in many reddened and tender, and there was more or less fluor albus.[1] In two sisters (of 9 and 11 years) the disease was said to be derived from a syphilitic nurse. One of them had been infected by her at the age of two years, and had then transmitted the disease to her sister, who was continually with her. As the parents here were undoubtedly worthy of credit, this case may serve to impress strongly on you the necessity for caution in the choice of servants and nurses. I have also known children from two to five years of age become infected from having to do with others who were affected with congenital syphilis, or with prostitutes who had taken lodgings with poor families. The source of infection in all such cases lies partly in the caressing of the children by syphilitic persons, partly in the use in common of sponges and other toilet articles and household necessaries, or in their sleeping together.

The symptoms with which syphilis begins in later childhood do not essentially differ from those in adults. The only thing worthy of note seems to be the predominance of condylomatous forms. Although I can by no means agree with Violet[2] that under these circumstances syphilitic eruptions never occur; yet I must allow that he is right in saying that mucous papules on the skin and mucous membrane constitute

[1] In three girls, of 4, 6, and 12 years, I observed, as the result of an attempted assault, not indeed syphilis, but a more or less considerable inflammation of the vulva with fluor albus and numerous warts on the labia.

[2] *Syphilis infantile:* Paris, 1874.

by far the commonest form of manifestation of the disease at this age. The mucous papules appear in more or less thick masses about the anus or on the labia majora, not uncommonly —partly softened and ulcerated—on their inner surface. In the latter situation they sometimes form quite nodular masses, disfiguring the whole labium. In two girls, of 12 and 13, I have seen a thick mass of mucous papules curving backwards on each side and extending from the commissure of the labia majora as far as the anus, and laterally reaching the folds of the groin. Also, the inner surfaces of the thighs, the nates, the folds of the skin between the neck and chest, and even the outer layer of the prepuce, were sometimes the seat of these growths; besides which, there also very frequently appeared, at the corners of the mouth, on the mucous membrane of the tonsils and of the adjacent palate (less commonly of the cheeks) whitish condylomatous growths partly eroded and partly cleft with fissures (rhagades). The upper and under lips were likewise sometimes the seat of rhagades, with infiltrations round them. Especially frequent, however, were gummatous changes on the dorsum of the tongue, in the form of round or more angular infiltrations of the mucous membrane, varying in size; which, by their darker colour and greater resistance, contrasted with the surrounding tissue. They sometimes projected above the surface, and in such cases (which were comparatively rare) they were somewhat white and opaque, or else eroded at their most prominent part. In two sisters, of 9 and 11, the almost exact correspondence in the gummatous affection of the tongue was very striking.

The relative rarity of syphilitic eruptions has been already mentioned. That they may occur is proved, however, by several cases in which a fine scaly roseola of the forehead, of the hairy scalp, of the body and extremities, with psoriasis palmaris and plantaris, was observed. In a girl of six years old and one of four years, with condylomata of the uvula, pharyngeal ulcers and a gumma of the tongue, there was psoriasis guttata extending over almost the whole body. There was a similar eruption in a boy of 7 years, who presented at the same time condylomata at the anus, on the tonsils, and in the middle line of the palate. The lymphatic glands were usually slightly enlarged and moveable, and in several cases most of the visible glands (the

cervical, occipital, cubital and inguinal) were distinctly swollen. I have often observed affections of the osseous system.

A girl 12 years old, brought 26th June, 1879, had complained for a year of violent pains in the right upper arm, especially during the night. The humerus was swollen to twice its usual size, largest towards the middle, uneven and angular, very tender on pressure. At the age of three years, syphilitic infection; later, affections of the throat (?). A few glands in the neck and in the axilla enlarged. Child previously treated, but disease always recurs. Further course unknown.

A girl of 11 years, brought 3rd November, 1874. For 1½ years very tender swelling of considerable size on the right tibia; and violent pains at night. Glands under the jaw enlarged. No other syphilitic symptom. Pot. iod. On 25th, marked improvement. On 20th July, 1875, no trace of former trouble. In the course of the following year (the girl was under treatment at the polyclinic for mitral incompetence) repeated slight relapses, requiring the renewed use of pot. iod.

A boy of 7 years, brought 15th February, 1876. Mother syphilitic. During the last 8 weeks a somewhat pointed exostosis has gradually been growing on the spina mentalis. It is now the size of a pigeon's egg—scarcely tender, and has already occasioned an abscess of the superjacent integument. Swelling of the bones of the nose, dry coryza, enlargement of glands. Already he has had repeated syphilitic symptoms. Did not return for treatment.

Considerable defects in the pharynx, complete destruction of the uvula, adhesion of the soft palate to the back wall of the pharynx, destructive ulceration of the nasal septum and of the hard palate, I have only exceptionally observed. The alteration of the teeth which has been strongly emphasised by Hutchinson (the upper incisors short, narrow, widely separated, and notched) and is said to be connected with an alveolar periostitis, I should not regard as a certain sign of syphilis tarda—all the more because this condition of the incisors occurs not uncommonly in children who are absolutely free from syphilis. In the same way the further working out of this idea by Parrot[1] seems extremely questionable; I should be much more inclined to regard the alterations in the form of the teeth as rachitic. Syphilitic caries of the bones of the skull, and the formation of gummata in the brain I have never observed;[2] but I have

[1] *Gaz. des hôpit.*, 1881, No. 74, 78, 80.

[2] *Cf.* Demme, No. 20. *Jahresbericht*, &c., S. 80.

certainly often seen amyloid degeneration of the liver and kidneys, of which I shall speak later.

The treatment in every case was mercurial, except in the very rare instances in which there was nothing but a bone affection. In these cases we first tried iodide of potash, which rapidly relieved the pains and reduced the swelling of the bones, but hardly ever prevented relapses. In other cases we at once had recourse to mercury, either in the form of inunction with mercurial ointment (grs. x.—xx. daily), of which, on an average, ℥i or ℥ii were used; or else injections of perchloride of mercury (gr. $\frac{1}{16}$—$\frac{1}{13}$), which were continued for about a fortnight, and only once, in a boy of 4 years old, caused a moderate degree of mercurial stomatitis. Mucous papules were effectually treated by touching with nitrate of silver, or by dusting with calomel.

IV.—*The Dyspeptic Conditions of Infants.*

Before turning to the morbid conditions which I class together under the term "dyspeptic," I must direct your attention to a symptom which appears, certainly, to be pathological, but which occurs so frequently, that we can scarcely regard it as such—I mean the vomiting of infants. This is entirely due to over-greedy sucking either of the breast or of the bottle, whereby the stomach becomes overloaded, and then gets rid of the surplus milk by a kind of regurgitation without much apparent effort. According as this takes place immediately after sucking or after an interval of some minutes, the milk returns either uncurdled or, more frequently, mixed with curds. This may be repeated after each sucking, or may occur more rarely; depending upon the amount of nourishment which the child takes. Movements, *e.g.*, rocking the child on the arms &c., favours the process, which, as I have said, occurs in innumerable children and seems to be an appointment of Nature's to guard against the development of dyspeptic conditions by the speedy discharge of the surplus quantity of nourishment. This regurgitation is favoured by certain characteristics peculiar to the stomach of infants up to about the 10th month; namely by its more vertical position as well as by the small development of the fundus and of the greater curvature as compared with their development in later

years—owing to which the capacity of the stomach is relatively smaller. So long, therefore, as the children remain, in spite of this vomiting, healthy and thriving in other respects, there is no occasion for medical interference. We may reassure the anxious mother, advise her to give the child the breast or bottle at longer intervals and for a shorter time, to let it lie quietly in bed immediately after sucking—and especially forbid all violent movement of the child. Improvement will generally soon take place, and will be favoured in some degree also by the further normal development of the stomach.[1]

Not uncommonly, however, cases occur in which the vomiting, which at first seemed to be merely of this simple form, assumes more serious significance, while at the same time weighing the child shows that it has ceased growing, and very soon the signs of incipient atrophy prove that we have to do with something more than a mere regurgitation of surplus milk. Under these circumstances vomiting takes place even after comparatively small quantities of milk. It is only after much persuasion on the part of the nurse that the child can be got to suck for a short time; and even then there is vomiting immediately or shortly afterwards, of uncurdled or but slightly coagulated milk. In such cases the physician may remain for some days in anxious doubt as to whether he has to do with a dyspeptic condition or with the commencement of a cerebral affection, especially of tubercular meningitis. I intend to return to this in describing that disease, and shall only mention here that the vomiting of dyspepsia is usually preceded and accompanied by eructations which betoken an amount of gas-formation in the stomach, unusual at this age and that it may have a sour or offensive smell. As a rule, the vomited milk is mixed with more or less tough mucus—a feature which I consider of especial importance. In the first days, or even weeks of this condition (which I call dyspepsia gastrica) the motions may retain almost their normal condition; or at most present a greenish or brownish colour. But generally they also are mixed with mucus and have an unusually offensive smell. Their

[1] Uffelmann (*Handbuch der privaten u. öffentl. Hygiene des Kindes:* Leipzig, 1881, S. 233) gives a case of vomiting in an infant which was caused by washing out its bottle with leaden shot. The milk contained lead and traces of arsenic. In obstinate cases, bear such possibilities in mind.

frequency, however, is usually not increased. As a rule, these children suffer much from flatulence, and before this is got rid of the abdomen is generally much distended, especially in the region of the transverse colon.

In another set of cases (dyspepsia intestinalis) there is either no vomiting at all, or it is so infrequent as to be of secondary importance. The dyspeptic symptoms manifest themselves in connection with the intestines. Many children take violent fits of screaming, writhe, turn up their eyes, and exhibit "lightning contractions," or convulsive trembling of the arms and legs, and do not become quiet until some of the flatus has been discharged with a loud noise (colica flatulenta[1]). The motions, which at first had the appearance described above, soon become looser and more frequent, and contain a quantity of yellow or greenish coloured flakes and lumps, consisting of casein, lime-salts and fat, with more or less tough mucus. They have a greenish (even a spinach-green) colour (biliverdin), and have either a sour, or oftener a highly offensive ammoniacal smell.[2] In the 24 hours there may be 15—20 such stools, for the most part with a strong acid reaction; but usually their number is limited to 5 or 6, at least in the early stage of the disease. The appetite is diminished; the tongue is sometimes clean, at other times covered with a greyish-white fur. The secretion of urine is diminished.

Whenever such symptoms are observed in an infant, you must at once carefully investigate their causes. For only by their removal, and not by medicines, is the dyspepsia to be permanently cured. In the first place we have to consider how the child is fed; because as a matter of experience improper feeding is almost always the cause of such derangements. Hand-fed infants are, as a matter of course, the most frequent subjects of this dyspepsia. Bad quality or adulteration of the milk is often to blame; still oftener, feeding with unsuitable farinaceous substitutes for milk at a period when the secretion of saliva is not

[1] That infants may also have colic from other causes—*e.g.*, from lead-poisoning—is shown by some cases published by Loewy (*Wiener med. Presse*, 1883). The causes were:—the use of rouge containing lead by the nurse, lead-fomentations to sore nipples, and a lead stopper lying in the bottle.

[2] Whether the colour is due to excessive production of acid, as is usually supposed, or to alkaline decomposition (Pfeiffer) is not yet settled. The influence on the colour exerted by the bacillus which has been described by French writers (Hayem and others) is even more in need of confirmation.

sufficient to justify their use. You must direct special attention to those feeding-bottles, so much in use among the poor, the mouth-pieces of which communicate with the interior of the bottle by a narrow indiarubber tube. Owing to insufficient cleaning of this tube, so that remains of milk-curd are left in it, the milk taken by the child is charged in passing through the tube with the germs of fermentation and the causes of dyspepsia. I have observed this so frequently in the polyclinic that I absolutely forbid the use of such feeding-bottles, unless assurance of the most careful cleanliness can be given. But even children on the breast are by no means exempt. An alteration (even although incapable of chemical or physical demonstration) of the milk of the mother or nurse—whether due to disturbances of temper, or excessive bodily exertion, want of nourishment, or recurrence of menstruation—may, as experience shows, produce dyspepsia in the child. From among many others I may mention as a striking example, a child of 4 months who throve splendidly with his nurse, until she got suppurative tonsillitis, which caused her very great pain and kept her from sleep. The child forthwith had diarrhœa, 5—6 loose, green, fœtid motions daily, until the tonsillar abscess burst. From that day the child's dyspepsia disappeared. I have already mentioned that the most inconceivable errors in the feeding of children are of quite common occurrence among the lower classes, although comparatively rare among educated people. Little children who are being fed from the breast or bottle are often allowed to share in the ordinary food of the family—potatoes variously cooked, cabbage, peas and beans, apples, grapes or plums are very often given to these children; and I have also had cases where sausages, pancakes, &c., had been used as food. In such circumstances one cannot wonder that dyspeptic conditions are amongst the commonest of infantile diseases, especially among the lower classes. This disease is particularly apt to occur at weaning, when there comes a change of food—whether this takes place only at the end of the first year or, through the force of circumstances (arrest of mammary secretion, or illness) a few months after birth (diarrhœa ablactatorum).

What, then, is taking place in the stomach and intestine? This question has received different answers at different periods. The view generally prevalent in former times, of an "acid-

formation" in the digestive organs, founded upon the sour smell of the mouth and on the acid condition of the green stools, gave place, when pathological anatomy came more to the front in our science, to the anatomical explanation that a "catarrh" of the gastric and intestinal mucous membrane was the cause of the dyspeptic symptoms. At a later period, there was a return to the chemical theory—which in my opinion is the only correct one. We have here, obviously, fermentative and septic processes in the contents of the stomach and intestine, the final result of which is the excessive production of lactic and fatty acids. The exact manner in which this process takes place cannot yet be laid down with certainty. Although the action of certain bacteria, which gain entrance to the stomach along with the milk and excite fermentation especially in the sugar contained in it, is rendered very probable by the most recent researches,[1] still we must also recognise that food which is difficult of digestion or even irritant, may by direct irritation cause in the first place a catarrhal condition of the stomach and intestine, with copious secretion of mucus. Then, through the alkaline nature of this mucus, the hydrochloric acid of the gastric juice which is necessary for normal digestion is neutralised, so that it can no longer operate upon the contents of the stomach in the normal manner; and there result fermentative processes, with the excessive production first of lactic and finally of butyric and fatty acids. These processes either come to an end in the stomach (d. gastrica), or (which is more common) extend still further downwards into the intestinal canal (d. intestinalis). For we can easily understand that if all the fermenting contents of the stomach are not evacuated by vomiting, the fermentation must pursue its course as soon as the abnormal contents with their germs of fermentation reach the intestine and come in contact with its contents. The sourish smell from the mouth, the masses of mucus in the vomit (which also generally smells sour), the fœtid evacuations, their irritating character (which is apt to occasion erythema round the anus), the flatulence and passage of fœtid gases by the anus, as well as the flatus discharged from the stomach—all these symptoms constitute the clinical manifestations of the abnormal chemical process. I shall here say nothing at all of microscopic inspection of the vomited matters and the

[1] Escherich, *Die Darmbacterien des Säuglings*, 1886, S. 116.

motions; because, in spite of many researches, some of which are most worthy of recognition, we have not yet been able to establish with certainty the forms of the micro-organisms with which we are here specially concerned. Besides, for the practical physician this difficult and tedious examination is unnecessary, since the clinical and etiological relations are all that is required for diagnosis. Sometimes such an enlargement of the stomach occurs as to be distinctly recognisable by the eye and by palpation. In such cases I have observed offensive eructations and flakes of a yellow (butter) colour in the mass of milk and mucus which was incessantly being vomited. The introduction of a simple stomach tube (Nélaton's catheter), which I have repeatedly tried in those cases, and always easily managed, at once brought about the evacuation of these masses, and invariably caused a rapid collapse of the greatly distended epigastrium. These fermentative processes are, however, by no means peculiar to early infancy. At a later age, also, even in adults we often enough see similar processes occur owing to overloading of the stomach with food and drink, injurious in its quantity or quality. These conditions are described under the names of status gastricus, biliosus, saburralis, diarrhœa stercoralis, &c. But while in older children and adults the morbid process generally ends with the discharge of the fermenting substances upwards or downwards, and therefore almost always is quickly over; this rapid termination occurs in infants only when the diet is at once regulated as it should be. Limiting the amount of food by less frequently giving the breast, substituting for it boiled water with a little gum-arabic dissolved in it, feeding with a solution of white of egg or with greatly diluted cow's milk, often suffice to remove the complaint in a few days. But, unfortunately, the conditions are frequently ill-adapted for protecting children from fresh attacks of the same kind. Only too often the dyspeptic symptoms are disregarded for a long time, and among the lower classes usually referred to teething, with which they have nothing at all to do. Without calling in a medical man, the mothers attempt to remove them by giving farinaceous food—oatmeal-water, gruel, &c.—and in this way matters grow worse. Thus the unnatural fœtid evacuations, and often the vomiting also, last for weeks, resulting in steadily increasing atrophy, as I have described (p. 71). The further

course is determined chiefly by the patient's circumstances, *i.e.* by the possibility of obtaining suitable feeding and treatment. The case may go on alternately getting better and worse for months, according as the physician's orders are followed more or less completely. Finally, an anatomical change is added to what was originally only a chemical one, since the prolonged irritation of the fermenting contents must necessarily induce a permanent catarrhal affection of the mucous membrane. At the post-mortem of such children we find areas of hyperæmia and swelling of the mucous membrane, in which both the solitary glands and the Peyer's patches project more than usual above the level of the mucous membrane—in a word, the appearance of chronic intestinal catarrh, to which I shall refer more particularly further on. In judging of this in any given case, we must never lose sight of the fact that we have here to do not with a primary disease of the mucous membrane, but with a secondary affection which must be regarded as arising from a chemical process. Sometimes too although the disease has lasted for months the change in the mucous membrane is extremely slight, and only discoverable on careful examination.

A special kind of dyspepsia has been recently described by Demme[1] and more especially by Biedert[2], under the name of "fat-diarrhœa." This is characterised by the copious discharge of motions, poor in bile, with a shining, fatty look or even an asbestos-like appearance. The chemical examination of these reveals a great increase in the amount of fat (40 to 67 per cent. of the dry substance); while even by the microscope a considerable increase of fat is made out. This condition, which may occur with either natural or artificial feeding, and which if chronic must lead to atrophy, is referred by Biedert to a catarrh of the duodenum hindering the fat-digesting secretions (bile and pancreatic juice) from entering the bowel, so that most of the fat in the food is discharged in an undigested state and nutrition suffers materially. Although I have myself repeatedly observed such fatty motions, yet in the absence of chemical and anatomical research I am not in a position to criticise the propriety of regarding this "fat-diarrhœa" as a separate form of dyspepsia. I shall only remark that the absence of jaundice

[1] *Jahresber. des Jenner'schen Kinderspitals von*, 1874, 1877, 1880, 1882.
[2] *Jahrb. f. Kinderheilk.*, Bd. xii., xiv., &c.

seems to me to tell against Biedert's view. Indeed, the considerations against it formerly brought forward (by Uffelmann) have received fresh support from recent researches[1] into the variations in the amount of fat and its occasional presence in large quantities in the fæces of healthy infants and those suffering from diarrhœa or from febrile affections. The whole question, then, is not yet ripe for judgment, in spite of seeming therapeutic results—to which I shall return soon.

When the dyspepsia of infants is acute from the beginning, it commences, sometimes, with such violent symptoms that after some days a critical and even fatal state of exhaustion may ensue. The clinical picture is then very similar to that which you will become acquainted with later on, in the description of cholera infantum. But the cases to which I here allude all occurred sporadically, and in the winter time,—that is to say, at a time when true cholera does not usually appear. Here also the cause may almost always be found in faults in the feeding of a very obvious kind; and this also happens in well-to-do families, where utterly undigestible dainties are given with the best intentions to little children by indulgent relatives or by servants. Violent vomiting, profuse, loose, fœtid evacuations (following one another in rapid succession and becoming more and more clear and colourless), intense thirst, alteration of the features, a very marked sinking-in of the eyes, low temperature of the skin, disappearance of the pulse and depression of the fontanelle, and finally convulsive fits, occur as in cholera—where, however, these symptoms are due to an epidemic and presumably infectious influence. The cause of the rapid collapse lies probably in the violent watery diarrhœa and vomiting, caused by the irritating action of the fermenting substances on the mucous membrane, and by the reflexly-increased peristalsis. This very great loss of water explains on the one hand the rapid re-absorption of the fluids of the body, which causes the sinking in of the features and the depression of the fontanelle, and on the other hand the extreme weakness of the heart which finds expression in the apathy and somnolence (arterial anæmia and venous hyperæmia of the brain) with the disappearance of the pulse and the fall of the temperature. Such cases may be just

[1] Tschernoff, *Jahrb. f. Kinderheilk.*, Bd. xxii., S. 1.—Kramsztyk., *ibid.*, S. 270.

as fatal as epidemic cholera in the summer months. Still, as a matter of experience, their prognosis, generally, is more favourable, because when the deleterious contents of the bowel have been expelled with violent symptoms, the disease usually ceases and the child again recovers strength. In the event of a fatal issue the post-mortem shows, as a rule, either extremely slight catarrhal changes in the mucous membrane of the stomach and intestine, or none at all; at times only an extreme paleness, corresponding to the general anæmia, with perhaps slight swelling of the follicles.

Under these circumstances we must always be prepared to meet with the peculiar alteration of the stomach which, under the name of "gelatinous softening of the stomach (gastromalacia)" has occupied physicians for many years. The slightest degree of this—and we meet with it pretty often—consists of a pulpy softness of the mucous membrane of the fundus and also of the posterior wall of the stomach, so that it can be scraped away with the handle of the scalpel like a thick solution of gum. Thus, the parts affected are just those which in the usual position of the dead body, are most exposed to the action of the stomach's contents. Less frequently, the softening affects all the coats of the stomach, and they are then transformed into a kind of grey, reddish, or dark brownish semi-transparent jelly, which has the smell of butyric acid and reddens litmus-paper. Generally, they are still held together by the serous coat; but this also may give way previous to the post-mortem; and we then find in the situation of the fundus, nothing left but a few fragments mixed with jelly-like masses and the contents of the stomach. There is not a trace of any inflammatory process to be found anywhere; and the microscope shows in the softened parts only some epithelial cells mixed with a mucus-like substance, and a few blood-vessels still intact and filled with dark clots. The question so long disputed as to whether gastromalacia is really a disease or merely a chemical alteration of the stomach which takes place after death, is now unquestionably settled in favour of the latter view. We have here to do with a post-mortem digestion of the coats of the stomach by its contents, and we can therefore only expect to find it where food had recently been taken and death ensued during digestion. Thus also is explained the fact that some-

times not only the fundus of the stomach but also the contiguous organs—spleen, left kidney, omentum and diaphragm, and even the lower lobe of the left lung, are found more or less digested and softened. We can easily explain how this condition was in former times regarded as morbid and furnished with a complete symptomatology, corresponding exactly with that of acute dyspepsia, or cholera. For in these diseases abnormal fermentative processes of the stomach's contents form the chief feature, and hence after death a destructive influence on its walls will be much more easily exerted than in other morbid conditions.

The fatal results which we have seen ensuing in dyspepsia neglected at its commencement, make it our duty to enter at once upon serious treatment of the case, which can only be carried out with a fair prospect of success where the circumstances of the little patients are favourable and our orders are carefully attended to. To the children of the poor, our aid often comes too late; and even when it is sought in time we meet with hindrances hard to remove—chief amongst which is the lack of proper nourishment.

In acute cases, we often reach the sick-bed only after nature has by violent vomiting and diarrhœa already got rid of the injurious contents of the alimentary canal. We now find the child simply exhausted, and we have nothing further to do but to superintend the regulation of the diet. If the child is on the breast, we must first—if no positive defect can be found in the diet—keep in mind the possibility of an injurious change in the milk. Changes of temper and over-exertion on the part of the nurse, occasion only a temporary change in the milk; and the child may therefore be put back to the breast whenever the dyspeptic evacuations have ceased. We must, however, especially guard against over-feeding, which is only too often to blame in cases of dyspepsia. Mother's milk requires two hours, at least, for its digestion; cow's milk certainly longer. And these intervals must therefore be carefully observed, before the child is fed again.[1] Unfortunately in practice one often meets with foolish obstinacy on this point; but the researches of

[1] Although Epstein, (*Archiv f. Kinderheilk.*, Bd. iv.), found on washing out the stomach of several healthy children of several weeks old, who had drunk 1—2½ oz. of their mother's milk, that the stomach was usually empty after 1—1½ hours—still, I cannot make up my mind to change from the practice above recommended.

Biedert[1] (who proved that the amount of nourishment taken in the first months, especially by hand-fed children, often far exceeds the amount really needed) show how very necessary it is for us to do all we can to check this foolish popular error, and to reduce the quantity.[2] Under these circumstances I have seen attacks resembling collapse in infants, also pallor, and symptoms like those of fainting, and these rapidly disappeared when the superfluous milk was vomited up. Restriction of the amount of nourishment is all the more necessary when dyspepsia already exists. It is therefore always well to forbid the breast entirely for 24—36 hours; or only to allow it to be taken less frequently than usual; or to give instead of it a little gruel or barley-water, or, still better, the solution of white of egg, recommended by Demme (the whites of 2 eggs to 1½ pints of water with a little sugar and cognac). Should the recurrence of menstruation in the nurse always occasion dyspepsia in the child, there remains no remedy except a change of nurse or weaning. However, in the majority of cases I have observed no bad effects on the milk from menstruation; and therefore I have but seldom had occasion to dismiss a nurse on this ground. It is the same with acute diseases of the nurse; which as I have shown you from a striking example (p. 127) may possibly originate dyspepsia, but by no means do so invariably. It is only when the acute disease of the nurse is presumably to be a short and slight one, that we may put the child who is suffering from dyspepsia on the bottle for the time being. But if such is not the case, you must at once try to procure another nurse. Should the child, however, be hand-fed, you will—after the attack is over—cautiously try again its usual food, if you consider it suitable. If relapses occur, a change of food must, naturally, be tried; and in this case the first question for consideration is whether we should now have a wet-nurse, instead of the artificial feeding which has been used since birth or for some time past. If the parents' circumstances allow it, you should advise a nurse. It is true that there are many difficulties to be met in such a case; for the children, having become accustomed to the bottle and the ease with which the milk flowed from it, prefer it to sucking the

[1] *Jahrb. f. Kinderheilk.*, xvii., S. 251, 288; xix., S. 291.

[2] Excessive quantities of milk will naturally also cause an increase in the amount of urine. Polyuria results, which occasions obstinate intertrigo in the neighbourhood of the genitals and anus.

breast, to which they are unaccustomed and which they often positively refuse. Still, if we only have patience, we shall generally succeed in getting over this difficulty and accustoming the child to the breast. I have seen children even 3—4 months old, who had been hand-fed from birth, take to the breast without much ado. Of course the thing is not always at an end even then. For the nurse's milk may, for various reasons (p. 127), disagree with the child and occasion dyspeptic symptoms; so that a new nurse has to be provided. Cases are by no means rare of such a child having three or more nurses in succession, before a suitable one was found.

The guiding rules for the dietetic treatment of infantile dyspepsia can only be laid down in a very general way. For you will often come upon cases in which, through obscure causes, the application of these rules becomes impossible, and such must be treated on some other method. Thus, I have sometimes had cases of dyspepsia which persisted in spite of a repeated change of nurse, and yielded only on the children being weaned. With others who have hitherto been exclusively hand-fed, even cow's milk (which I have always regarded as the best substitute for the breast: p. 81) caused dyspepsia, so that one had to give it up or replace it by some other form of nourishment such as the above-mentioned (p. 84) infant foods. At the same time the idea of many physicians that good cow's milk is not digested under such circumstances, is not generally justified. I advise you to be guided here, less by theoretical opinion than by practical experience, and to make repeated trials with cow's milk before having recourse to any other substitute. How frequently have infants with dyspeptic diarrhœa been brought to me, who, through dread of cow's milk, had been fed only on oat-meal water and thin gruel, and who were in consequence becoming more and more wasted. I confidently advised that they should be put again on cow's milk, and I have very often seen the motions and the general condition improve every day when they did so. Experience, however, has taught me that in these cases the milk is often more easily digested cold than warm—probably because it is in this state less liable to ferment. It should therefore be allowed to cool after it has been boiled; and, especially in acute dyspepsia, should be put in ice and given to the children quite cold. Most children take it

willingly, many even greedily; and whenever they begin to refuse the cold milk and again show an inclination for the warm, I regard it as a sign of returning health. As long, however, as dyspeptic vomiting continues it will be well to give the children cold milk from a spoon, because drinking it from the bottle is apt to cause overloading of the stomach and vomiting.

Child of 10 months, weaned six weeks before, suffering 1½ weeks from diarrhœa, for which hydrochloric acid had been used with varying success. On 19th December, 1864, sudden exacerbation, numerous loose bright-yellow motions; occasional vomiting. Continual restlessness, slight sinking of the features, abdomen normal, but tender on pressure. Latterly only veal-tea had been given instead of milk; but neither this nor small doses of opium nor calomel had any favourable result. Within 24 hours there were about 20 motions and frequent vomiting; at the same time high fever and unquenchable thirst. Milk and arrowroot given on 22nd caused repeated vomiting and still more severe diarrhœa. I now ordered 2 or 3 dessert-spoonfuls of iced milk every hour, and to quench the thirst little pieces of ice frequently and ice-cold water slightly sweetened. As medicine an emulsion of almonds, likewise iced, was ordered in teaspoonful doses. On the following day, already a marked improvement; rest and sleep for several hours; pulse and temperature normal; thirst considerably lessened. Vomiting had only taken place once, after violent crying, and the 3 motions which had been passed were perfectly normal. On 24th, complete convalescence; and the child now refused the cold milk which it had hitherto taken greedily, and again showed a desire for the usual lukewarm milk mixed with arrowroot. The anorexia, which still continued, with a thick white fur on the tongue, yielded in the course of a week to small doses of tinct. rhei.

Child H., one year old, suffering from dyspeptic diarrhœa which had followed on weaning 14 days before. On 12th November, 1873, I found the child collapsed, cold, with scarcely perceptible pulse. Milk and all other drinks were at once vomited; 12—15 loose, brownish, offensive motions daily. Treatment:—iced milk in spoonfuls, 2 camomile baths daily, bismuthi subnitratis gr. ¾ every 2 hours. On 14th no more vomiting; cold milk is taken greedily and well borne. Still 6—7 evacuations daily, with a putrid smell. Treatment changed to creasote gtt. ivss., aq. ℥ ii., a teaspoonful every 2 hours. Recovery after four days.

Such examples (of which I have now collected a large number) certainly encourage the trial of iced milk as a form of nourishment in the acute dyspepsia of infants. Even in this form, however, the milk has not always a favourable effect; and it is

then necessary to substitute other drinks—solution of white of egg, soups, barley water, decoctions of salep, arrowroot, or "infants' food." In persistent vomiting, we may also attempt to administer the nourishment per rectum, and I have twice or thrice tried this by means of enemata of peptone (about a teaspoonful in half a cup of beef-tea). I have, however, had no success from this, probably because the very active peristaltic movements of the bowel were still further increased by the enemata, which were at once rejected almost unaltered. I have myself no experience of peptone given by the mouth, which is praised by Escherich. The washing-out of the stomach recommended by Epstein and others[1] in obstinate vomiting of young children, which according to my experience is generally easy to perform (p. 129), I consider worth a trial even in older children, especially when the stomach is evidently distended, and gross errors in diet are known to have been committed. I have not yet had sufficient experience of this method to justify me in speaking decisively about it. It is certain that recovery often occurs without washing-out of the stomach; this proceeding, however, cannot do any harm whatever, and may accelerate recovery by rapidly getting rid of fermenting materials. Still we should be on our guard against over-estimating this method of treatment. In many of my cases, indeed, a single washing-out was sufficient to arrest an obstinate attack of vomiting; but far oftener the treatment was unsuccessful, although frequently repeated. The miserable condition of the majority of the patients in my children's ward may, however, be to blame for this want of success.

As to medicinal treatment:—in recent cases of dyspepsia (that is, such as have not lasted more than a week), whether the dyspepsia is shown by vomiting or by diarrhœa, or by both, I should recommend calomel as the first remedy. This should be given, according to the child's age, in doses of gr. $\frac{1}{13}$—$\frac{1}{7}$ every three hours with pulv. acaciæ, grs. viii. (Form. 2). Although nothing definite can be said as to the way in which this medicine operates, its action is probably anti-fermentative. The statement that the calomel is changed into perchloride by the

[1] "Ueber Magenausspülungen bei Säuglingen," *Archiv f. Kinderheilk.*, Bd. iv. *Jahrb. f. Kinderheilk.*, xxvii. S. 113.—Lorey, ibid. xxvi. S. 44.—Ehring, ibid. xxvii. S. 258.

chloride of sodium in the contents of the stomach and bowel, is correct only in so far that such a change takes place very gradually, and only when large quantities of calomel remain in the bowel for a long time. In the present cases, however, neither of these conditions is fulfilled. Let us, therefore, hold to the therapeutic action which has been ascertained practically. Cessation of the vomiting and improvement of the motions (diminution of the fœtor, and more pulpy consistence) occur frequently by the second or third day of its use, and in many cases there is no need of any other remedy. Perhaps the purgative effect, although it is but slight, which even such small doses of calomel have upon infants, may be regarded as a favourable accessory action; since in such cases, the first point is to remove the abnormal contents of the bowel as quickly as possible from the body. Should the affection have already lasted a week or longer, we cannot promise ourselves such good results from calomel as in perfectly fresh cases; still, even in this case, the medicine is worth a trial, for I at any rate have never observed any injurious effects from its use.[1]

Next to calomel, in my experience, stands hydrochloric acid (Form. 3), which in not quite recent cases may also be given with good effect. The action of this medicine, as the experiments of Schottin[2] prove, is strongly anti-fermentative. He showed in the case of fermenting fluids in a hot chamber, that the lactic acid—as well as butyric acid—fermentation is immediately arrested by adding sulphuric acid, and does not begin again until the acid has been neutralised by an alkali. "Hydrochloric acid acts much more favourably, because it is also able to dissolve the proteids in the stomach, and take the place of the gastric juice which is wanting." In fresh cases you must not add any opium, for its constipating effect is apt to cause great distension of the bowel with gas. But if several days have elapsed and the loose motions still continue, you may then assume that after the injurious contents have been got rid of, there remains an irritated condition of the mucous membrane, and an increased peristalsis. When this is so, the addition of tinct. opii (about gtt. iv.—v. to the mix-

[1] *Cf.* on the action of calomel on fermentative processes, &c., Wassilieff, *Zeitschr. f. physiol. Chemie*, vi., S. 112.

[2] Köhler, *Handb. des physiol. Therapeutik:* Göttingen, 1876, S. 882.

ture) is very beneficial—doubtless because this, by lessening the peristalsis, affords time for the hydrochloric acid to take permanent effect.

The results which I obtained with calomel and hydrochloric acid and published some time ago[1], have since then received confirmation in innumerable cases. Nevertheless, there are still many physicians who prefer alkaline remedies, especially bicarbonate of soda, to acids. But, although this medicine may temporarily neutralise the acid of the fermenting contents of the stomach, it cannot reach the fermenting process itself, and I can therefore recommend neither it nor other alkaline medicines. I have not experimented sufficiently with benzoate of soda[2] (which is praised as an antiseptic) to be able to give a definite judgment as to its value. Being contented with the success I obtained with calomel and hydrochloric acid I have not looked for other remedies. Where these remedies fail, however, I should certainly recommend creasote on account of its strong anti-fermentative action, especially in cases in which vomiting is a prominent feature. But, if only given in sufficient doses (Form. 4), it is also effectual in those cases where, after the violent symptoms are over, there still continue to be thin, offensive motions which are not improved by hydrochloric acid. The following cases show that we need not be afraid even of large doses.

A boy of 7 months, hand-fed. For some days back, vomiting of milk partly fluid and partly curded, with a sour smell. Also frequent sour-smelling motions, resembling "weiss Bier." Hydrochloric acid alone, and also along with tinct. opii., was unsuccessful. I next tried creasoti gtt. vii., syrupi simpl. ʒ iii., aquam ad ℥ ii., a teaspoonful every 2 hours. After 2 days, cessation of the vomiting, but persistence of the diarrhœa, which was afterwards cured by small doses of opium.

A girl of 6 weeks, hand-fed. During the last 24 hours, diarrhœa and vomiting after every drink. The vomited matter smells very sour. Creasote, gtt. iv. in ℥ ii., a teaspoonful every 2 hours. After 4 days, only 1—2 normal motions; no more vomiting.

In children, therefore, of six weeks and seven months respectively the dose was ¼ and ½ drop; since ℥ii. of fluid represent about 16 teaspoonfuls. Besides the drugs I have

[1] *Beiträge zur Kinderheilk., N.F.*, S. 293.

[2] Escherich, *Centralbl. f. Bacteriologie u. s. w.*, ii., 1887, No. 21.

named, which in my opinion occupy the first place among the remedies for this disease, I have also made trial, both in the hospital and in private practice, of other medicines which have a high reputation for their antifermentative action—namely, chloral hydrate (1 per cent. solution or more), carbolic acid, aqua chlori and resorcin. The first of these was successful (although not invariably) in cases of dyspeptic vomiting. The other three I have quite given up; and I consider the continued use of carbolic acid especially, as not unattended with danger. Naphthalin, which has been occasionally praised very recently, I have not seen occasion to use from the reports given of it. Just as little did the pepsin (so much recommended of late) meet my expectations; and this may perhaps have been because we are unable to determine the indications for its administration in each individual case. This remedy, however, can evidently be of use only where the dyspeptic fermentation is produced either by diminished secretion of gastric juice or at least by a deficient amount of pepsin in it. These changes can only be estimated (and that merely approximately) when the contents of the stomach are removed by a tube and examined chemically, which generally is quite impracticable in ordinary practice. Under these circumstances, the use of pepsin in infantile dyspepsia must always remain an experiment which we can try either at the beginning or after other remedies have been used without result, but one whose success is to be regarded merely as a happy chance. I prescribe pepsin either alone (gr. i.—iss.) or along with hydrochloric acid (Form. 5) in the form of the essence of pepsin to be had of any chemist. Naturally, pepsin can only exercise its effect where substances containing protein—especially milk—are still being taken. It must therefore always be given half-an-hour before or after food.

Richard K., 10 weeks old, hand-fed, poorly nourished, brought 7th December, 1866. For some days back, no sleep, frequent colic, daily 10—12 loose green motions, excoriating the anus; slight flatulent distension, no vomiting, no fever. Calomel used, without result. Pepsin, (gr. i. 3—4 times daily) brought about recovery after 12 doses. On 13th April, 1867, brought again to the polyclinic on account of vomiting whenever food was taken. This had lasted for some weeks. Thrush in the mouth. Pepsin. gr. i. 4 times daily. By 16th marked abatement of the vomiting; on 23rd, complete recovery.

Girl of 15 weeks, brought 6th May, 1873—hand-fed. For 4 weeks past, vomiting (especially frequent after taking milk) and diarrhœa. Great restlessness, a certain amount of wasting, great thirst, motions very offensive. Pepsin, gr. i., 4 times daily. Recovery on 14th. Now only 3 normal motions daily.

Boy of 6 weeks, on the breast, brought 19th Jan., 1874. Violent vomiting after each drink; frequent green offensive evacuations. Calomel without effect. On 24th, pepsin. grs. xv., aq. destill., syr. simpl., ana 3 vii., acidi hydrochlor. gtt. x., a teaspoonful every 2 hours. On 27th, vomiting much less frequent, and not till 10 or 15 minutes after taking the breast. Motions better. Pepsin increased to grs. xxiii. in the mixture. Recovery on 31st.

Thus we see that under certain circumstances pepsin has good results; and we may in dyspepsia have to try, one after another, all the remedies which are accredited by previous practice. In one case one drug, in another another, will prove the more effectual, without our being able to discover the reason of this difference. Besides the remedies already named there are several others, which will be described under the heading *Diarrhœa;* especially subnitrate of bismuth (magisterium bismuthi). The suitable time for the administration of this drug is, I think, the moment when the presence of particles of mucus in the motions indicates that the chemical processes are beginning to cause a catarrhal condition of the mucous membrane of the intestine. To children in the first year bismuth. subnit., gr. $\frac{3}{4}$—gr. iii., with pulv. acaciæ, grs. viii., may be given 5—6 times daily; and when the disease has lasted for a week I have often seen an increased effect from the addition of extr. opii., gr. $\frac{1}{3^{1}2}$. Later on, also, if the symptoms of chronic intestinal catarrh are becoming constantly more apparent, bismuth proves to be one of our most reliable remedies. Nitrate of silver, also, (gr. $\frac{3}{4}$ in ℥iiiss.) undoubtedly does good service in many cases of dyspeptic diarrhœa, and is therefore always worth a trial when the disease is very obstinate. After recovery, I recommend rhubarb as a tonic for the digestion. It should be used for some weeks in the form of vinum rhei (gtt. v.—xv., 3—4 times daily, according to age).[1]

[1] I have no experience of transfusion, which Demme (18. *Bericht über die Thätigkeit des Jenner'schen Kinderhospitals.*, 1880, S. 42) has tried frequently (5 grammes of blood, 5—8 times) and with partial success. Demme himself speaks very reservedly about this method, which is said to raise the depressed nutrition.

V. *Coryza of Infants.*

The extreme sensitiveness of the mucous membrane of the nose in infants is shown to a marked degree in the new-born child, in whom soon after birth and in the first weeks of life contact with the air excites frequent reflex sneezing. Any chill affecting the child, especially from carelessness in washing or bathing it, readily occasions a coryza with snuffling breathing and watery mucous discharge which, if cleanliness is not carefully attended to, dries into yellowish-brown crusts about the nostrils, and interferes with the entrance of air. This tendency to coryza is also found in infants throughout the whole of the first year. After what I have already said (p. 93), you will understand that in all such cases a suspicion of hereditary syphilis occurs to the physician—especially as coryza may form the very first symptom of syphilis and precede all its other manifestations by weeks. For this reason we are obliged in every protracted case of coryza, to examine the child and its parents in regard to this matter; so that, should our suspicion be confirmed, specific treatment—which in that case alone is of any use—may be commenced.

Now, although syphilitic coryza may bring with it the same risks as any ordinary non-specific coryza—still, this very seldom happens. In most cases it is only one link in a chain of symptoms, and does not claim to be of specially great significance. Serious symptoms, which may become fatal in various ways, occur far more frequently in the simple coryza due to a cold. The danger to the child lies chiefly in the fact that the coryza may at this age extend downwards with great rapidity, to the mucous membrane of the larynx and trachea, and even to that of the bronchi. Hoarseness of the cry, coughing, fever and dyspnœa often develope within a few days. Examination then shows more or less wide-spread bronchitis or broncho-pneumonia. On the other hand, the catarrhal swelling of the mucous membrane of the nose which causes considerable contraction of the child's already sufficiently narrow nasal cavity, may result in more or less extreme dyspnœa. This gives an alarming character to any tracheal or bronchial catarrh which is combined with coryza, even when percussion and auscultation do not seem to justify

apprehension. But even in cases of simple uncomplicated coryza we sometimes have sudden attacks of dyspnœa, which are apt to perplex the physician who has been hastily summoned and is unacquainted with the child's previous condition. Bouchut describes symptoms of asphyxia as having occurred in the following way:—The child was unable to breathe through the obstructed nostrils; it had, therefore, to breathe through the mouth with such force that the tongue was suddenly jerked back during the process, and the lower surface of its tip pressed against the hard palate, thus necessarily obstructing the passage of air into the throat. This explanation of the sucking-in of the tongue by violent inspiration is held by many authors—among others, by Kussmaul and Honsell;[1] and the possibility of its occurrence, especially when the frenum is long and loose, cannot be denied. Personally, I have only once met with this indrawing of the tongue. It was not in a case of coryza, but in a violent attack of spasmus glottidis in which I could only with difficulty reach the root of the tongue with my forefinger, it being firmly pressed against the palate and curled upon itself. In coryza of very young children I have always been obliged to regard the dyspnœa as the result of the blocking of the nasal cavity; and in very acute cases I have known it reach such a degree that it might have been mistaken for croup.[2]

In March, 1861, I was called to see a child of 7 weeks, who had been attacked by violent dyspnœa about 1½ hours previously. According to the account given by the alarmed parents, the child had been perfectly well a few hours before and had been taken out in a strong east wind. Almost immediately after returning, the attacks had come on without any evident occasion—not even that of sucking. As the worst of it was over by the time I arrived, I thought it might have been an attack of spasmus glottidis, and in order to decide this I had the child put to the breast. At once a fresh and even more violent attack resulted, almost as severe as one sees in croup. With an expression of extreme anxiety on its cyanotic face, with open mouth and violent action of all the inspiratory muscles, the child gasped for breath; and at each gasp a whistling noise was heard which obviously proceeded from the nose. The cavity of the pharynx was completely free. After a few minutes, a gradual cessation took place, sleep soon following—

---

[1] *Henle's und Pfeuffer's Zeitschr. 3 Reihe.*, Bd. xxiii., S. 230, 1865.

[2] In a case of syphilitic coryza given by Hassing (*Jahrb. f. Kinderheilk.*, xxiii., S. 466), it was even necessary to perform tracheotomy.

during which both inspiration and expiration were accompanied by a snuffling noise. The lower part of the nose was somewhat swollen. During the next 12 hours I had the child fed only with the spoon, had warm oil rubbed over the bridge of the nose, and gave calomel, gr. $\frac{1}{4}$, every 2 hours. During the following days a muco-purulent discharge made its appearance from the nose, but disappeared again after a few days.

In cases of this kind—which are always rare—the rapid development of catarrhal swelling of the mucous membrane is especially noteworthy, being analogous to that which so frequently occurs during the night in adults in the course of a violent cold in the head (especially when in a recumbent position) and interferes with breathing through the nose. Here also the secretion is arrested when the swelling increases; and, as a rule, raising oneself to a sitting posture is the first thing to bring relief, as everyone has probably experienced for himself. In the case just mentioned also, the dyspnœa was best relieved by carrying the little patient about in an upright posture. To my mind, there is a decided analogy between these cases of acute coryza, so-called false croup, and certain very acute attacks of bronchial catarrh to which I shall have an occasion to return later on. According to the recent experience of specialists, it is conceivable that catarrhal irritation of the mucous membrane of the nose may also excite reflexly a spastic contraction of the bronchial muscles; and this may give rise to such violent symptoms as in the case just given. Another danger lies in the interference with sucking. The child during this act has to depend upon breathing through the nose, and finding this no longer possible, has to let go the nipple or mouthpiece of the bottle frequently in order to breathe through the mouth; and in this way its nutrition is in course of time seriously interfered with. For the same reason, in severe coryza it is during sucking that the violent attacks of dyspnœa occur.

Coryza nearly always attacks both nasal cavities at once. It is but rarely limited to one side. For example, I observed, in June 1874, a child of 8 weeks, formerly perfectly healthy and certainly free from any suspicion of syphilis, which had suffered for about a fortnight from a yellowish watery discharge from the right nasal cavity, while the left was quite unaffected. Pressure on the right side of the nose promoted the discharge. Along with this there was snuffling respiration and dyspnœa during

sucking, so that the child was often obliged to let go the nipple. Brushing out the right nasal cavity with a solution of nitrate of silver effected a cure in 14 days.

The examples given contain all I have to say to you on the treatment of coryza. The nourishment of the child demands your attention above everything else. If sucking is interfered with by dyspnœa, you must either have the mother's milk artificially drawn off, or have cow's milk given with a spoon; and I have always found this satisfactory. A case recorded by Kussmaul is likely to remain unique:—a child of 6 months having to be fed for a whole week by means of an œsophageal tube, owing to the drawing-in of the tongue already spoken of. For internal use in very acute cases of coryza, I recommend calomel, gr. $\frac{1}{7}$—$\frac{1}{4}$, every 2 hours, even where there is no suspicion of syphilis. In slighter attacks, however, we need do nothing beyond keeping the lumen of the nostrils free by applying oil and removing the scabs. If the disease takes a more chronic course, good effects will be gained by painting the inside of the nose with a solution of nitrate of silver (2 per cent).

We shall discuss later on diphtheritic coryza, which is by no means rare during infancy. I only remark here in regard to it that in every case of coryza in a young child, if one wishes to guard against surprises of a very serious nature, a daily examination of the pharynx is indispensable.

VI.—*Retro-pharyngeal Abscess.*

The reason why this disease is still practically unknown to many physicians, lies chiefly in the fact that its occurrence is very uncommon. In spite of the large amount of clinical material at my disposal, I have records of only about 65 cases. Thus it is that those physicians who do not see any large number of sick children are generally unacquainted with this disease, and so usually fail to recognise the first case presented to them. On the other hand, any one who has had the opportunity of watching closely even a single case of retro-pharyngeal abscess is tolerably well insured against future error in diagnosis. For the clinical picture of the disease is indelibly impressed upon his mind, and the recollection of this single experience makes the diagnosis easy to him.

This disease consists of an abscess in the connective tissue between the cervical spine and the pharynx, which almost always developes somewhat insidiously and gradually forms a tumour projecting more or less into the cavity of the pharynx, thus occasioning interference with deglutition and in a greater degree with respiration.

My first case of this kind I observed as early as 1850,[1] and I readily admit that I owed my diagnosis of it entirely to the circumstance that I had chanced a few days previously to read two cases of this kind published by Fleming in the Dublin Journal for Feb. 1850. This first one, along with two other cases, will be found recorded in the book published by Romberg and myself ("Klinische Warhnehmungen und Beobachtungen": Berlin 1851, S. 120), and the description then given has since required no alteration in spite of numerous subsequent observations. In almost all my cases, the children were still in their first year or but little beyond it. The majority were much less than a year old, the youngest being only four months. In only two cases were the children aged 2 and 3½ respectively; and these, as it chanced, came to the polyclinic on the same day (26th July 1880). The disease in its early stages is very obscure; crying, restlessness and frequent refusal of the breast or bottle are the first symptoms, and from these alone no diagnosis can be made. We may, indeed, assume that there must be pain in swallowing from the beginning. But dysphagia is a symptom which cannot be made out at first in children who are too young to complain; although only a pained expression of the features during drinking may arouse suspicion. But this is often absent, even when the tumour is fully developed as is also the regurgitation of liquids. The first symptom which I regard as really suspicious is a snoring character of the breathing, especially during sleep; and this very symptom causes the inexperienced to regard the complaint as a cold in the head—which, indeed, does at times, though by no means always, accompany it. The inspection of the pharynx—which in these circumstances every conscientious physician ought to make—usually reveals nothing, or, at most, a swelling and redness of the mucous membrane of the throat, which is covered by mucus; and one is satisfied with the diagnosis of a catarrhal swelling of the turbinated bones.

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

Generally it is from 10 days to a fortnight or more before the abscess by its size seriously interferes with the breathing. Next the sleep is disturbed; the child sleeps with its mouth open, but wakes often and gasps for breath. Gradually however a fresh set of symptoms commences, which is apt to mislead one unacquainted with the disease by its resemblance to severe laryngeal catarrh or even croup. The respiration becomes laboured, the accessory muscles of inspiration act strongly, while each inspiration and expiration is accompanied by a snoring noise. When the child attempts to drink, attacks of choking may occur and the liquid is often rejected again from the mouth and nose. In extreme forms of this disease the countenance is distressed and may present a cyanotic hue. Formerly the absence of cough and the quite normal sound of the voice appeared to me very important symptoms, for I thought that I found in them an essential difference from croup. Further experience, however, has taught me that these are by no means constant, and that cases sometimes occur in which hoarseness and cough are present owing to an accompanying catarrh. The duty of examining locally therefore becomes all the more imperative. In many cases of retro-pharyngeal abscess a diffuse swelling is visible on one or both sides of the upper part of the neck; and several swollen lymphatic glands may also be felt, which from their superficial position look as if forced outwards from within. The external jugular veins are often much distended. All these symptoms, however, are in no way characteristic; a sure diagnosis can only rest on an examination of the pharynx by means of the finger introduced over the tongue into the throat, and on that alone. In infants who have teeth this examination is more difficult, because they often bite the finger; and in these cases I generally use a metal ring as a protection. You must also be prepared to find in very extreme dyspnœa that not only may symptoms of asphyxia but even convulsions, be excited by the local examination, as Fleming has noted. Still, I have managed in every case, and without great difficulty, to feel the abscess quite distinctly as a swelling in the throat projecting forward from the spinal column. It is situated either at the upper part, so that one comes upon it just behind the velum, or else (which is much more undesirable) deeper down at the level of the epiglottis or even lower. The swelling is generally of a

rounded form, more rarely oval, distinctly fluctuating, about the size of a walnut, and situated either in the median line or a little to one side of it. Whenever you feel this you may be sure of the diagnosis. For other fluctuating swellings with the symptoms described and having an acute course, occur only exceptionally in this region in such young children.[1] The diagnosis being made, there can be no question about the treatment. I should strongly recommend you not to delay for a moment the incision of the swelling as soon as fluctuation has been distinctly made out. For, although the dyspnœa, which has arisen owing to the entrance of air into the larynx being obstructed, may not as yet have reached an imminently dangerous degree, still you can never be sure that the tumour may not burst of its own accord and some of its contents be drawn into the larynx with the inspiration. It happened in my own experience that a colleague, who for the sake of a clinical demonstration wished to "preserve" a case of this kind till the following day, paid the penalty of this delay in the sudden death of the child from suffocation during the night. Cases such as this and the one given by Noll—where the abscess was allowed to remain unopened for 7 days after it was discovered, and ended fatally by bursting into the œsophagus, and by extension of suppuration—must be adduced as warnings and examples.

Thus, then, there is only one remedy—immediate incision. In all the cases I have had hitherto, I have performed this with a straight bistoury or, if the abscess was situated low down, with a curved one, or else with a tenotomy knife enveloped almost to the point in paper or sticking-plaster. The head of the child, who ought to be sitting upright, is to be firmly held by an assistant or nurse. The tongue must then be depressed by the forefinger of the left hand, which may be protected by a metal ring when the children have teeth; in such a manner that the point of the finger touches the swelling and feels it plainly. Then, using the finger as a director, the knife is to be carefully guided along it to its tip—that is, to the tumour, which is then to be boldly incised. The cavity of the throat becomes at once filled with yellow matter and a quantity also is expelled from the

[1] *Cf. e.g.*, the case of a lipoma behind the pharynx (Taylor, *Lancet*, 1876, ii., p. 685), or that of an abscess between the tongue and epiglottis (Pauly, *Klin. Wochenschr.*, No. 22, 1877).

nose. The small wound is to be enlarged in withdrawing the knife. To facilitate the expulsion of the matter, the child's head should at once be bent forwards. When the incision has been made, the trouble in the majority of cases is at an end, and a more speedy and surprising change can scarcely be imagined than that from the extreme dyspnœa, threatening immediate death, to a feeling of perfect well-being. Almost always, I have seen the difficulty of breathing vanish as by magic, the swelling on the neck speedily disappear, the turgidity of the jugular veins diminish, and—even after a few minutes—the child which had seemed past recovery now looking about it brightly, and willingly taking the breast which it had so long refused.

The matter is not always, however, disposed of so speedily and smoothly. In several cases I have met with much greater difficulty—due for the most part, to the abscess being situated deep down. In these cases I could only with difficulty reach it with the point of the forefinger, and get the curved bistoury down far enough. Especially in very young children, in whom the mouth and throat were extremely small, I have often found it very difficult, because every time an operation was attempted, violent attacks of suffocation were caused by the passage of the finger over the larynx.[1] The breathing stopped, the child became cyanotic, the eyes turned up, the pulse became irregular and small, and there was nothing for it but to withdraw the finger quickly and so restore respiration. I have never, however, given up the attempt, and have always been fortunate enough to gain my point in the end; except in one case where the abscess was situated so low down behind the lowest part of the pharynx that I was doubtful of the result from the very beginning. For opening these very deeply situated retro-pharyngeal and retro-œsophageal abscesses, a guarded pharyngotome is to be recommended; but I have never used it myself. The greater facility of introduction, the less danger of wounding other parts of the mouth and throat, and the possibility of reaching a much greater depth with the instrument, ought to make it decidedly preferable for this kind of abscess. I have also repeatedly observed cases in which a single incision of the abscess was not sufficient. It very often re-filled even on the

[1] I have already published one such case in my "*Beiträge zur Kinderheilkunde*," *N.F.*: Berlin, 1868, S. 269.

following day, probably owing to the opening being too small. The symptoms were renewed, and a second operation had to be performed which almost always resulted in a complete cure. Only in one case was I obliged to open the abscess a third time, but I should mention that the second time I had made use of my finger-nail instead of a bistoury—a method which is occasionally employed, but which I cannot recommend. After incision, I advise you to have the cavity of the nose and throat syringed with tepid water. There certainly is a possible danger of these fluids being drawn into the larynx during the operation,[1] but nothing of the kind occurred in any of my cases; nor yet have I ever observed any bad results from milk getting into the incision.

If the operation is not performed at once, as I have already said, a spontaneous rupture may take place during sleep and the pus be drawn into the air-passages, causing fatal suffocation, or, as I have once seen, rapidly fatal pneumonia. Or again, the matter may gravitate downwards behind the pharynx or œsophagus, even as far as the mediastinum, and death then ensues from exhaustion due to the extensive suppuration. In the following case the suppuration spread at the same time down to the outer part of the neck.

A weak, emaciated child of 10 months was brought on 2nd April, 1875, to my polyclinic. It was said to have been unable to swallow properly for about a fortnight. Also there was snoring and in places a wheezing breath-sound; a large amount of mucus in the throat, and diffuse swelling in both submaxillary regions—in which a few enlarged lymphatic glands (the largest being of the size of a walnut) could be felt. Veins on the temple unusually prominent. On introducing the finger, I felt, on a level with the epiglottis, a fluctuating tumour, the size of a walnut, projecting into the pharynx from behind. This I at once incised, and copious discharge of pus followed. In the next few days marked improvement of all the symptoms, but the discharge of pus from the wound still persisted, the external swelling diminished very little, and the enlargement of the glands was unchanged. On the 9th, I could make out on each side of the upper cervical region a large fluctuating swelling. The left of these was opened at once, the right on the 11th, after the child had been taken into the Charité. From both incisions there was an enormous amount of pus dis-

[1] For a few cases of this kind which ended fatally owing to pneumonia following inspiration of pus, see Témoin, *Revue mens.*, Avril, 1887, p. 172.

charged; but the wounds did not heal, the suppuration continued both inside and outside, while emaciation and collapse advanced daily. Death on the 19th. At the post-mortem a large collection of pus was found behind the pharynx and œsophagus, which extended on both sides into the submaxillary regions, and had here been opened externally. Further, there was limited broncho-pneumonia, enlargement of the mesenteric glands, and small tubercles in the liver. Vertebral column normal.[1]

You see from this that suppuration originally confined to the retro-pharyngeal connective tissue may also extend laterally, and penetrating through between the muscles may appear externally on the neck. I have only once had an opportunity of observing a rupture of the abscess into the pharynx.

Pale, emaciated child of 15 months brought to my polyclinic 10th January, 1865. Complete aphonia during the last 8 days; coughing and hoarseness for some time previously. Breathing noisy, especially during sleep. Pharynx red and full of mucus. No tumour to be discovered either inside or outside. Catarrh of the bronchi; dyspnœa; inability to continue sucking for any time. No dysphagia; moderate fever. Death on 14th with difficulty in breathing. *P.-M.*—On separating the larynx from the hyoid bone, a large quantity of yellow pus spurted out, seeming to come from a hole (the size of a pea at least) in the back wall of the pharynx. This hole had quite the appearance of a round gastric ulcer, and was situated just at the junction of the pharynx and œsophagus. Pus continued to flow out of it. When the œsophagus was dissected we found an extensive collection of pus between it and the vertebral column, extending from the atlas to the sixth cervical vertebra. This whole extent was covered with fragments of gangrenous connective tissue. The spinal column showed no morbid alteration. Little fimbriated masses were situated upon and under the vocal cords, and turned out to be tubercular. There was also caseous degeneration of the bronchial glands, and tuberculosis of the lungs.

This case shows that if the retro-pharyngeal abscess ruptures into the pharynx, its diagnosis may become impossible; for then the pus for the most part escapes through the ruptured point into the œsophagus and is swallowed. Therefore, no swelling need necessarily occur, either externally or into the pharynx.

Still more uncommon—in my experience—than retro-pharyngeal abscesses, are those which form on the lateral walls of

[1] In such cases—which are very rare at any time—paralysis of the facial nerve may occur from pressure of the pus on the region of the stylomastoid foramen (Bokai).

the pharynx, between it and the soft parts of the neck, consequently forming a fluctuating tumour on the right or left side behind and under the tonsils. In two cases a rupture of the abscess took place into the external auditory meatus. But this is certainly an extremely rare occurrence.

On 10th April, 1874, a medical friend of mine consulted me about an affection of the neck from which his child (æt. 15 months) had suffered for several days. The chief symptoms were fretfulness, dysphagia, crying at each attempt to swallow, moderate fever, and noisy breathing during sleep. The left tonsil was somewhat enlarged and much reddened. Immediately behind and beneath it on the lateral wall of the pharynx a red fluctuating tumour could be seen and felt. Also externally, under the mastoid process, there was a diffuse swelling. No difficulty of breathing observable. When on the 12th I examined again with a view to incising the abscess and pressed rather firmly on the swelling, a stream of yellow pus streaked with blood suddenly spurted out of the left ear, whereupon the tumour at once disappeared, so that no operation was necessary. On the 13th the pus continued to flow in moderate quantity from the ear, especially on pressure beneath the mastoid process. The child was perfectly well, slept without snoring, the tonsil was almost normal, and no trace of the tumour was any longer perceptible. No disturbance of the hearing resulted.

As the nurse said that she had noticed a discharge of matter from the ear some days before, it may be taken as certain that the abscess on the lateral wall of the pharynx had gradually worked its way through the loose connective tissue to the meatus auditorius and had broken through it at a number of points. Squeezing the tumour had suddenly completed the rupture. The second case, which was observed at the polyclinic (May, 1881), had a quite analogous course. Bokai[1] also describes a similar one: only in it the abscess had already been opened from the inside and had since re-filled; and on pressure it at once discharged through the left ear, after which complete recovery ensued.

It is rare for phlegmonous abscesses of the connective tissue of the neck to open into the pharynx. But I have observed this in one boy of 5 years old, admitted into the hospital (11th April, 1881) with a very large hard infiltration extending from the angle of the jaw on the right side to the scapula, and in

[1] *Jahrb. f. Kinderheilk.*, x., 1876, S. 151.

front as far as the second rib. Scarlet fever and diphtheria could be excluded. The pharynx was reddened; its right lateral wall was pressed inwards and the uvula displaced towards the left. Dysphagia, copious secretion of saliva. Evening temperature, 104·2° F. On the 12th, spontaneous rupture of the abscess into the pharynx, the child spitting out a quantity of offensive pus and blood, and fragments of tissue. Temp. normal. On the 13th an incision made into the neck, on account of fluctuation, and some offensive pus let out. Drainage. On the 25th, recovery.—In two other cases I have seen a submaxillary phlegmon (which had developed as the result of scarlet fever) rupture into the pharynx before an incision was made. Of this I shall again have to speak in treating of scarlatina.[1]

With very few exceptions, all the cases I have observed belong to the class of idiopathic abscesses, *i.e.*, to those which occur in perfectly healthy children independently of any other illness. A few children were perhaps somewhat atrophic, but there were no abscesses in any other part of the body. Nor yet was there any disease of the cervical vertebræ or any general condition owing to which the abscess could have developed. The etiology of all these cases is, therefore, involved in complete obscurity, and the supposition of Bokai and others, that the inflammation and suppuration of the retro-pharyngeal connective tissue originated in the lymphatic glands in front of the spinal column, is by no means certainly proved. I myself, indeed, had one case of a child of 3 years who still showed distinct scars of scrofulous glandular abscesses in both submaxillary regions. Still I do not consider this sufficient to warrant us in referring the abscess with absolute certainty to retro-pharyngeal adenitis.

I only twice observed the formation of an abscess arising from spondylitis of the cervical vertebræ. In a child of 1½ years, which since the beginning of December, 1874, had been observed to move its head with difficulty and pain and to hold it very stiffly, I found these symptoms markedly increased on 5th April, 1875; and in addition there were difficulty in swallowing, laboured and snoring breathing during sleep, and an abscess the size of a walnut situated very low down on the back wall of the pharynx. It was incised the same day and a considerable quantity of pus was evacuated. The diagnosis of spinal caries was afterwards confirmed by the appearance of

[1] Bokai and Lewandowsky describe similar cases (*Klin. Wochenschr.*, 1882. No. 8).

abscesses on the back and neck, by paralysis of the arms and paresis of the lower extremities. Another case, observed in the polyclinic, had a quite similar course.

VII.—*Dentition and its Symptoms.*

Although the eruption of the teeth generally indicates the end of the period of suckling, and Nature herself thus gives us to understand that the exclusively liquid food may now be exchanged for a somewhat more solid dietary, the obligation to such a change of food is not by any means imperative. As a rule the first teeth appear between the 7th and 9th months, and yet it is customary for the mother or wet-nurse to give the breast till the end of the 9th month at least (and generally still longer), even when the children have got all their incisors. When this is done the nurse may certainly be injured by the child biting the nipple; and for the child itself, unpleasant consequences may result from this, as we may learn from a case which I observed:—a healthy child of one year being frightened by the sudden scream of the mother on being bitten, started violently, and immediately had an attack of convulsions.

Every physician knows from experience that the most diverse disorders of infants, especially of those in the first half year of life, are attributed by the relatives to "the teeth." Superstition and indolence here lend a hand, especially in practice among the poor, to produce all sorts of mischief which it is often very difficult to undo afterwards. Every attack of diarrhœa or convulsions which occurs in those children, is put down to "the teeth;" and is accordingly either neglected or even regarded as salutary. The physician's aid is often only called in when it is too late. This old-standing tradition, still in full force among the laity in spite of the improvement of education, is now most positively contradicted by a large number of medical men of the present day. Teething, they hold, is a physiological process, which cannot be the occasion of any morbid symptoms, and everything formerly regarded as such is a delusion, caused by illnesses happening to occur along with them, without having anything at all to do with it. It may however be questioned whether this positive denial is altogether warranted, and while I very fully acknowledge the service it has rendered in limiting the

"diseases of teething," I cannot help thinking that there is a want of moderation in this view. We know that dentition occurs in the following way:—the growing fang of the tooth gradually pushes on the already complete crown, and forces it out of the alveolus after it has burst through the overlying gum which has been gradually thinned by the increasing pressure. Is it, then, so very inconceivable that this gradually advancing process should exert an irritating action on the dental branches of the fifth nerve, and occasion reflex symptoms extending not only to the province of the motor, but also to that of the vaso-motor nerves? It seems to me quite conceivable, and I certainly consider it is going too far to deny utterly the possibility of convulsions being caused by the irritation of teething. I shall supply instances later on where, *e.g.*, partial contractions of the muscles of the throat and neck were undoubtedly connected with the eruption of a group of teeth. Also the indisputable fact that obstinate vomiting, diarrhœa, a spasmodic cough, or eczema of the face, which for days or weeks has defied all treatment, will all disappear as soon as one or a couple of teeth emerge from the alveolus, and this can only be explained by the reflex action from the dental branches of the fifth upon the peristalsis, the vagus or the vaso-motor nerves. We must guard against throwing overboard the views of our medical predecessors with that presumption which has become the fashion with a section of the younger school;—and also against putting forward principles without such practical experience as is necessary, and can only be the result of a long professional life and very numerous personal observations. It is a matter of fact that a large number of children are out of sorts during the cutting of each group of teeth, cry a great deal (evidently from pain), are restless during sleep, and cease to gain weight[1]; they may also have a flabby skin, a pale complexion, urine milky from the presence of urates, and even slight variations of temperature.

Although generally the first tooth appears between the 7th and 9th month, examples are not wanting of teething taking place much earlier. I have frequently seen cases in which one or two incisors had already come through by the end of the 2nd or 3rd month, or a little later. More commonly, however, the process is rather delayed, and even in children who

[1] Dehio, *Jahrb. f. Kinderheilk.*, Bd. xx., 1883, S. 64.—Staege, *ibid.*, S. 425.

are perfectly healthy and quite free from rickets, we sometimes do not see the first tooth until the 10th or 11th month. You will also have heard of another abnormality, which in certain historic personages was regarded as presaging a vigorous and masterful disposition,—I mean being already provided with teeth at birth. According to my observations we can distinguish two forms of this phenomenon. In the first form we see one or two pointed, more or less hook-shaped teeth, which, being only imbedded in a fold of the gum, are from the beginning loose and easily moved. As a rule they are the two middle incisors of the lower jaw; and in one child (five weeks old) they appeared almost normal in shape, but had serrated and grooved edges. Probably in such cases the tooth-germ had not only had a premature development, but also an abnormally superficial position, so that the crown came through before the root had reached maturity. I have always removed such teeth immediately, and without difficulty, with a pair of forceps, for they generally injure the nipple and also the under surface of the child's tongue, on which there may appear one or two ulcerations corresponding to the teeth. In one case only, where these ulcers healed under the application of a solution of sulphate of zinc—2 per cent.,—the teeth gradually became more firmly fixed in the alveolus, and I therefore let them alone. But I do not know what became of them finally. In the second form I have found real teeth firmly set in the alveolus, distinguished, however, from the normal ones which appeared later on by their rough surface and yellowish colour—due to their want of enamel. These teeth require greater force for their removal, and I advise you rather to leave them untouched until they become loose; but as soon as this happens I think they should be extracted, for in such cases I have always observed a morbid process in the alveolus which can only be cured by the removal of the tooth. The following cases may serve as examples of this form:—

Girl of 3 months, brought to the polyclinic on 2nd April, 1875. There had been a tooth in the left upper jaw at birth, extracted on the 5th day. Soon after there was swelling of the left cheek. On examination a marked thickening of the left upper jaw was found, also fistulous openings on the alveolar margin, out of which pus oozed. Discharge of pus from the left nasal cavity and from a fistula situated below the margin of the orbit. The

pus was extremely offensive. Fluctuating abscess in the region of the left zygomatic arch. On the 20th, separation of several pieces of dead bone from the alveolar margin ; later on, artificial removal of a larger sequestrum. Further course unknown.

Girl of 5 months, brought 5th October, 1877. Had developed a painful swelling on the left cheek after the forcible extraction of a tooth which was present at birth in the left upper jaw. On examination, the upper jaw was found to be thickened and tender, and there were fistulous openings on the left alveolar border and a discharge of pus from the left nostril. Did not return for treatment.

Boy of 2 months, brought 4th January, 1878. The whole left half of the lower jaw much swollen, very tender on pressure, the gum dark-red and swollen. Pressure under the jaw caused a flow of pus into the mouth—and this also occurred spontaneously. In the region of the first molar, there was a small hole in the gum out of which the pus came, and a probe passed into it came against something hard. It was stated that the first left incisor had come through at the age of six weeks—that is to say, simultaneously with the commencement of the swelling and suppuration. When the child was brought a second time, on the 15th, the first molar had completely emerged from the opening mentioned. Both teeth were pretty loose in the jaw, and had to be extracted. Unfortunately I had no further opportunity of observing this child's case.

A child of 13 days (10th January, 1884), took ill on 4th day of life, without apparent cause. The margin of the lower alveolus swollen, red, covered with pus, which oozes out on pressure as from a sponge. During the last few days both lower central incisors have appeared and been abstracted, leaving behind two suppurating cavities. The teeth consist only of a crown coming to a point below without a root.

The last two cases, in which we have to do not so much with congenital teeth as with extremely premature dentition, appear to me to throw light on the whole process, as they make it probable that periostitis of the alveolar margin, whether in the upper or lower jaw, forces the crown of the tooth out by swelling and exudation inside the alveolus. Accordingly, I consider periostitis to be the primary cause, and not the result of the violent extraction of the tooth, as I once thought. And I think the first cases may also be so regarded. How this disease of the bone, occurring at birth or soon after, was occasioned, I must leave undecided. At any rate, in all four cases hereditary syphilis could be with certainty excluded. The extraction of

such teeth might thus, under the circumstances, be not only without danger, but even necessary in order to free the alveolus from the irritating foreign body. Samelsohn[1], who observed a case of periostitis of the orbit in a child of 14 days, thought the cause of the disease (which was accompanied by an enormous protrusion of the eyeball) was to be found in the first molar tooth, which was being prematurely forced through; and when it was extracted, the whole process ended favourably. The tooth showed a well-developed crown, and the beginning of a fang. I think, however, that the cause of the process was not the tooth's being "prematurely forced through," but that it was forced out prematurely through the occurrence of periostitis of the upper jaw, due to causes unknown. From three cases published by Klementowsky[2], we see that, even in children in the first days and months of life, the alveolus may be laid bare, and the teeth may come through and drop out owing to inflammation of the gum and periosteum ending in necrosis. Unfortunately, I cannot make out clearly from the extracts quoted from this work (which is inaccessible to me in the original) whether the author's opinion, that these were cases of gangrenous stomatitis, is really justified.

The process of teething, even when it takes place at the proper time, may be accompanied by various local morbid symptoms which must undoubtedly be regarded as produced by some irritation connected with the teeth. Very often, a general redness of the mucous membrane of the mouth is observed, especially of the gum, which is covered here and there with small detached fragments of epithelium, and with a great excess of saliva. Every touch of the gum causes pain, and readily excites little hæmorrhages. In other cases the inflammatory symptoms are confined to the immediate neighbourhood of the teeth which are coming through; this appears dark red, and is ulcerated to some extent on the surface or becomes the seat of small, frequently-recurring abscesses. At times, also, a large number of those yellowish-grey plaques are developed on the tongue and other parts of the mucous membrane; these we shall afterwards become better acquainted with under the name of "stomatitis aphthosa." Among the fully developed teeth, it is

[1] *Centralzeitung f. Kinderheilk.*, i., 1876, S. 190.
[2] *Ibid.*, 1879, S. 186.

specially the two middle incisors of the lower jaw that by their sharpness injure the under surface of the tongue during sucking or even during violent coughing, and produce little ulcers upon it. Indeed, in one child of 8 months, quite healthy and without the least cough, I found that the frenulum linguæ had been almost entirely destroyed by a yellowish-grey ulcer, which bled readily and was due to the sharpness of the two middle incisors. This ulcer had arisen from the continual passing of the under surface of the tongue over the teeth in sucking, and was quite analogous to those which occur in whooping-cough. All these local symptoms are, however, rare; in the great majority of cases dentition takes place without any local trouble in the mouth, and this very circumstance, it seems to me, affords support to the view which I have put forward above—that disturbances of remote organs (which are absent in most cases) may occur under certain conditions, especially in peculiarly nervous children disposed to reflex action. Whether one adopts this view or not, it is now pretty well agreed that any attempt to facilitate the cutting of the teeth and thereby to remove the symptoms depending on "difficult" teething is absolutely useless. The practice introduced from England of scarifying the gums down to the coming tooth with a bistoury, I have made use of in former years often enough to convince myself of its uselessness. I have found that this neither caused the tooth to appear sooner, nor influenced in the slightest degree any spasmodic attacks (especially spasmus glottidis) which chanced to be present. Indeed it seems to me questionable whether this procedure (formerly so much praised) does not, by the scar which it occasions, rather increase the resistance to the cutting of the tooth. The only benefit that we need expect from this small operation is that from the bleeding, in the rare cases where the hyperæmia of the gum is unusually severe, and this, in such cases, may easily become excessive. On all these grounds I have now for many years entirely given up the scarifying; and in this I am in agreement with most medical men.

The cutting of the twenty milk-teeth which a child has, goes on in certain periods which are separated by intervals. As I have already remarked, the lower central incisors appear between the 7th and 9th months, often later than that, seldom earlier; and

several weeks (6—8) after them the central incisors of the upper row follow. Next come the upper lateral, and after some weeks the lower lateral incisors; and in normal circumstances they are usually through by the end of the first year. Variations from this order are not at all uncommon; *e.g.*, the upper incisors may be the first to appear, and the lower ones come after them. The group of four front molars generally appears between the 15th and the 18th month, but in rare cases they develop—at least in part—before the complete cutting of the lateral incisors. Between the 18th and 20th month there follows, as a rule, the cutting of the four canine or eye-teeth, which fill up the gap between the molars and incisors. After the longest pause—sometimes of several months—the four posterior molars appear between the 20th and 26th month, and complete the process. This finishes the first dentition. All this, however, applies only to healthy children. Teething is very often delayed owing to a bad constitution, especially rickets, the first incisors appearing only at the end of the first year, or even much later; and the whole process may be prolonged far into the third year of life, owing to the intervals between the different groups being also longer. One child (æt. 5 years) who was not rickety showed a very rare abnormality, the two upper outer incisors appearing for the first time in the 4th and 5th years respectively. I cannot here enter upon all the possible abnormalities of the first dentition. I may just mention however the occasional occurrence of two teeth in place of one:—for example, this happened in one of my patients in the case of the right eye-tooth, in whom instead of one there were two eye-teeth—one situated in front and almost normal, and the other placed somewhat obliquely behind, smaller and more pointed.

We have now concluded the pathology of very early infancy, and pass to the consideration of those diseases which affect childhood generally, from early infancy upwards. The variations in the clinical symptoms due to their more tender age will at the same time receive due consideration.

SECTION III.

DISEASES OF THE NERVOUS SYSTEM.

I.—*Infantile Convulsions.*

The pathology of the nervous system derives a large and most important part of its material from childhood. The liability of the nervous system in children to disease does not, however, affect all its parts equally. Whilst, of the central organs, the brain is pre-eminently subject to a very great number of diseases, the spinal cord—apart from its congenital affections (spina bifida) and infantile spinal paralysis—is much more rarely affected. Among the so-called neuroses, those of the sensory functions (neuralgia and anæsthesia) are very unimportant compared with the derangements of the motor functions, especially convulsions which constitute one of the most frequent maladies of childhood from birth to about the end of the third year. It has been attempted in various ways, even by experiments (Soltmann) to explain this extraordinary tendency of the organism in childhood to convulsive attacks. Although Soltmann proves that the strong tendency to reflex manifestations in the very early life of animals—up to the 10th day—depends on the absence of centres in the brain and spinal cord controlling reflex action,[1] still the great tendency to convulsions which is also present in older children—in the 2nd and 3rd year—cannot be explained in this way. If we keep to clinical observation we find this tendency to reflex convulsions confirmed as a matter of daily experience. If you watch a little child quietly for some time, you will see how its whole body twitches spasmodically at any sudden noise or unexpected touch; and how during violent screaming it becomes suddenly breathless from laryngeal spasm. We also see how frequently simple indigestion causes general convulsions through the reflex irritation proceeding from the stomach and

[1] *Cf.*, on the other hand, the experiments of Tarchanoff (*Centralbl. f. Kinderheilk.*, ii., 1879, S. 183), Lemoine, Marcacci and Paneth (*Biolog. Centralbl.*, 2, 1886).

intestinal canal,—which under similar circumstances in adults would certainly be a very exceptional occurrence.

The symptoms of convulsions, or eclampsia infantilis as it is usually called, vary in no way from those of an epileptic seizure. The attack commonly begins by the eyeballs turning upwards or to the side, or with a strange fixed look, while consciousness disappears. Twitchings of the facial muscles follow, sometimes unilateral—the mouth being drawn to one side; then the jaws are firmly closed by trismus, or owing to spasm of the pterygoids are moved from side to side on one another causing grinding of the teeth. Chewing movements are also sometimes observed. Tetanic rigidity of the extremities, interrupted more or less frequently by spasmodic twitchings like those excited by an electric current, almost always occurs. The fingers are generally strongly flexed and can only be extended with difficulty; the feet are dorsi-flexed or in the position of pes equinus, according as the extensors or flexors are most affected by the convulsive rigidity. The muscles of the trunk also participate; retraction or rolling about of the head, contraction of the respiratory muscles with alarming pauses in the respiration alternating with very rapid superficial breathing, rigidity of the abdominal muscles, involuntary expulsion of the urine and fæces—all these are, if not invariable, at least frequent accompaniments. After a very few seconds the distorted face becomes somewhat cyanotic round the nose and mouth, and the saliva is forced out from between the lips in the form of froth by the violent action of the muscles of the tongue and those of mastication, and by the cheeks. In older children who have teeth this froth is often mixed with blood owing to the tongue being bitten. These symptoms, which alarm the parents extremely—especially if they are inexperienced—last, as a rule, only a few minutes, the spasms then diminish gradually in intensity and frequency; the stiffened limbs are relaxed, the face becomes quieter, the colour returns, and at length only slight spasmodic contractions from time to time passing over the unconscious child, remind us of the storm that has passed—like the distant lightning and faint peals after the thunderstorm is over. But this quiet is but temporary and deceptive. Even before the child has recovered from its stupor, the attack begins again with renewed fury; and in this way the convulsions may be repeated three or four times,

one after another, while in the invervals the comatose condition and complete loss of consciousness and sensation continue. The persistence of the reflex sensibility may in such circumstances easily mislead; for touching the conjunctiva often produces contraction of the orbicularis, and sprinkling with cold water excites reflex contractions. But in many cases this symptom is absent, and I could then lay the point of my finger on the ocular conjunctiva without observing the slightest movement of the eyelids. We must not, however, at once regard this want of reflex sensibility as a fatal sign, as many do regard it; for I have seen a number of children who displayed this symptom and yet recovered completely. The duration of the paroxysm is of much greater significance. These attacks, interrupted only by short periods of coma, may go on for hours, and you can readily understand that under these circumstances the arrest of the respiration, the venous engorgement in the brain, and finally, the complete exhaustion of the child's strength may lead to death. But even then, the fatal issue is not always inevitable, and every physician will recall cases of this kind which, in spite of convulsions recurring constantly during many hours, days, and even weeks, ended nevertheless in complete recovery.

Ordinary attacks, lasting only a few minutes, are often over by the time the physician arrives. He then usually finds the child comatose, and this condition passes imperceptibly into a healthy sleep which may last several hours, or even a whole night, and from which the child wakens apparently quite well and looking as if nothing had happened. Still, we must here be always on our guard. An attack of eclampsia seldom occurs alone; sooner or later, we must expect a repetition of it, and the cases are not uncommon in which the distressing spectacle is repeated daily or perhaps twice a day. In many other cases, however, weeks and months pass before a new attack occurs.

When you are summoned to such a case and find the child still in convulsions, there is no time for finding out from the terrified bystanders details as to the origin of the malady. What is wanted from you is to stop the convulsions at once, and, fortunately, no exact anamnesis is needed to guide one in treating the attack. Causal indications must here first of all give way to vital ones, and I know of no remedy which fulfils the latter more certainly than do inhalations of chloro-

form. Do not waste time with other things, such as chloral hydrate, purgative enemata, cold compresses, the application of leeches to the head, &c.; but always use chloroform at once, when you wish to arrest an attack exceeding the average duration (*i.e.* over 5 minutes). A teaspoonful of chloroform poured on a handkerchief and held before the child's nose so as not to exclude sufficient air, is often quite enough. Even after a few inspirations the convulsive excitement is calmed, and the inhalation may be confidently continued until the convulsions have entirely ceased. The pulse and respiration of the child must be narrowly watched of course during this time, in order that the inhalation may be stopped at once if need be. I have never myself met with any unpleasant effect, although I have used chloroform in many cases of eclampsia, even in children only a few months old. In one such child who had more than 40 attacks in the course of one day, each time as soon as a new attack set in I ordered chloroform; two or three inhalations of the vapour always sufficed to allay the convulsive movements at once, and next day (after a good night's rest) the child—apart from great exhaustion—was perfectly well. I have even ventured repeatedly in these cases to teach the relatives how to administer chloroform, and instructed them to give it whenever new attacks occurred; and I have never yet had to repent this confidence. It is indeed impossible—unless the physician can sit with the child the whole day long—to have skilled aid at hand whenever it is required; and so the only resource is to venture the experiment with the relatives,—or, better still, with a good nurse. I never regard cyanotic discolouration of the face due to convulsions as contra-indicating chloroform. The discolouration always disappears as soon as the remedy begins to act. Nor yet have I refrained from using chloroform for convulsions in the course of broncho-pneumonia. The convulsions soon cease, while the lung disease pursues its course. I must not, however, conceal from you that chloroform is not an absolutely certain remedy for convulsions. Apart from the fact that it generally acts as a mere palliative and is not able to prevent the repetition of the convulsions, I have also found it practically useless in a few very violent cases:—the pauses which the inhalations produced scarcely lasted two or three minutes, and the attack finally ended in exhaustion and death. You must

also take care not to give this remedy if you find the child already collapsed with the pulse very small and rapid and the extremities beginning to become cold. Such cases, however, form but a small minority, and need not restrain one from strongly recommending the use of chloroform. On the other hand, the compression of the carotids recommended by Parry, Blaud, Trousseau, and others, which I have myself frequently tried[1] gives far too uncertain results to merit serious consideration.

As soon as the attack of eclampsia to which you have been summoned has ended either spontaneously or by the aid of chloroform inhalations,—the question arises as to the cause of the disease; for only by realising the causal indications will you be in a position to guard against the return of the attack. It is not my business here to enter fully into the pathology of epileptiform attacks generally. I would only remind you of this, that experiments have certainly indicated a threefold origin of such attacks:—anæmia of the brain owing to contraction of the smallest cerebral arteries (Kussmaul and Tenner); the division of the spinal cord or sciatic nerve on one side, followed by irritation of the corresponding side of the face (Brown-Séquard); and blows on the head resulting in slight extravasation of blood in the medulla oblongata (Westphal). In considering the pathology of infantile convulsions some value—according to my thinking—may be attached to the first and the third of those series of experiments. On the one hand a few examples are recorded of a violent fall or blow on the head causing epileptiform attacks in a child,—and those even recurring habitually, and I have myself observed two such cases. On the other hand, anæmia of the brain in exhausting diseases, owing to cardiac debility (the convulsions of inanition) or a spasmodic contraction of the small cerebral arteries with anæmia may be assumed when we have to do with a condition of reflex irritation or with a febrile attack commencing with convulsions. These explanations however cannot, I think, by any means be regarded as exhausting the pathology of eclampsia. I should only remind you of the fact that during the attack we frequently observe increased tension, prominence, and very marked pulsation of the great fontanelle—symptoms which rather point to hyperæmia than to anæmia of the brain.

[1] *Beitr. zur Kinderheilk., N.F.:* Berlin, 1878, S. 97.

We now turn to the etiological conditions of eclampsia as ascertained from clinical experience. The first question which meets you in every case is one very important for the prognosis, namely—Whether the convulsions arise from organic disease of the brain or not?—a question you cannot at once decide, more especially if you are unacquainted with the child. When the convulsions are unilateral, this has been regarded as in favour of a cerebral origin, and I grant that this is in general correct, provided that when the attacks recur, the same side of the body is always affected and the other remains free. Along with this however one must not overlook that occasionally convulsions occur on both sides when only one side of the brain is affected (*e.g.* in tubercle), and that on the other hand unilateral convulsions have been observed in cases where no real cerebral disease is present. I have repeatedly seen the first attack under these circumstances, confined to one side of the face or to one half of the body, or else the paroxysm only consisting of rotatory motions of the head with rolling of the eyes and spasmodic contractions of one arm; and the spasms only appeared later on the other side of the body also. In a child of 8 years, who died of intussusception, I observed, on the day of death, convulsions affecting the right side of the face and body exclusively. Nevertheless the unilateral character of the convulsions is always an important feature, making it incumbent on us to examine the child very thoroughly, in the intervals between the fits, for any affection of the brain, and to make very exact enquiries as to the history. At the same time you must not forget that many diseases of the brain (*e.g.* tubercle and tumours) may for a long time, even for many months, only reveal their presence by attacks of eclampsia recurring from time to time, until suddenly hemiplegia or coma makes the mistake manifest. It is often difficult for the physician to give an opinion, and I would specially point out that even in reflex convulsions (especially in little children) apparently serious symptoms may often occur in the intervals;—the child is pallid, apathetic, never smiles, starts frequently, and there are increased pulsation of the fontanelle and slight elevations of temperature. A cautious physician will always do well to delay giving a definite opinion until he has been reassured by further observation and the non-appearance of more serious cerebral symptoms.

In all cases of convulsions recurring more or less frequently, I would recommend you to direct your attention in the first place to the osseous system. According to my experience, the tendency to convulsions is favoured by nothing so strongly as by rickets; and, taught by innumerable cases, I always make a point in the case of every child who comes under my treatment for eclampsia, of immediately examining the epiphyses of the ribs and of the bones of the forearm, and also the skull. In most children between six months and the middle of the third year, I have found indications of rickets more or less well marked. Almost always in these cases there are simultaneously attacks of laryngeal spasm, which either usher in the convulsive attacks or alternate with them. Only rarely is the eclampsia unaccompanied by laryngeal spasm. Wherein this tendency of rickety children to convulsions consists, is as yet undetermined. It would be rash to make the deficient nourishment of the nerve centres responsible for it; for eclampsia occurs just as readily in rickety children who are well nourished as in those that are atrophic. But at any rate, in such children we must be prepared for the recurrence of the attacks, for which definite causes can be assigned only in a very few cases.

In my opinion rickets is a much more influential factor in this disease than dentition, which so often gets the blame for convulsions occurring about that period. We might with as much justice derive rickets itself from teething—an idea which would occur to no rational being. It is only rarely that convulsions are observed in teething children who are not rickety. Quite definite reflex causes must therefore be discoverable. With these causes, as I have already remarked (p. 155) we may certainly class cutting of the teeth under specially unfavourable circumstances; but such cases are at all events rare and hard to prove. You must not allow yourself to be diverted from the careful investigation of other causes which are much more frequently operative, by the mothers' habit of calling their children's convulsions "teething fits." Among these causes, an irritated condition of the digestive organs undoubtedly occupies the first place. Even in new-born children and infants, we see convulsive attacks not unfrequently occur reflexly during dyspepsia; and especially

unskilful artificial feeding—along with over-feeding—may be the source of most violent attacks of eclampsia.

> In a child of this kind, aged 4 months, to whom more than 3 pints of cow's milk had been given daily, and had produced extreme flatulent distension, I saw numerous convulsive attacks occur during 16 consecutive days. Not uncommonly they were repeated 50—60 times in one day, so that there were no intervals to speak of. The attacks generally began with tenesmus and flushing of the face, or with rumbling noises in the abdomen, and the motions consisted of light clayey masses, mixed with large, hard lumps of casein. After the deleterious contents of the bowel had been cleared out by castor oil and enemata, and the flatulent distension removed, it was found possible also to lower the state of enormously increased reflex excitability, and to put an end to the fits by the cautious use of chloroform inhalations (iced compresses on the head). The child was then put to nurse, and throve well, but remained permanently extremely feeble-minded.

This was one of the cases in which the convulsions occurred unilaterally from the first (movement of the head towards the right, and twitching of the right arm) and thus a suspicion of real brain disease had been aroused. The further course showed that the convulsions were reflex and originated in the intestinal canal; and in this connection I would call attention to the tenesmus, and rumbling in the abdomen which almost always preceded the convulsions, as well as to the character of the motions. It was so evident here that the eclampsia depended on the disturbance of the digestion, that even the backwardness in psychical development afterwards ascertained cannot be regarded as proving primary brain disease, but must rather be looked upon as the result of the innumerable convulsive attacks. I have seen the arrest of psychical development in another child also, who before the occurrence of the eclampsia was perfectly normal in this respect, although extremely rickety. In this case also, hundreds of convulsive attacks were observed in the space of some weeks, and it appeared a miracle that life was preserved. Neither paralysis nor any other symptom of chronic cerebral disease ever occurred; only aphasia and mental hebetude remained, and after a year's interval showed but slight improvement. We may therefore assume that convulsions occurring in unusual numbers and many times daily for weeks, may impair the psychical energies of a previously healthy brain for a long

time, or may even injure it permanently to a considerable extent. Fortunately, cases of such severity and long duration are very rare.

To the same category belong the cases of infants in whom eclampsia occurs soon after violent excitement or abuse of alcohol on the part of the mother or nurse, so far as they are to be regarded as exclusively caused by a change in the milk injurious to the child's digestive organs. At a later period of childhood, up to the second dentition, very violent convulsive attacks may be caused by overloading of the stomach and intestine by food injurious either in quality or quantity. Out of the long series of cases of this kind which I have observed, the following may serve as examples :—

Child of 3½ years. At mid-day indulged largely in cucumber-salad and plums. In the evening convulsive fits, which, with intervals of coma, lasted about 2 hours. Cold compresses to the head, enemata, an emetic when the coma had passed off.

Child of 2 years, healthy. Took ill on 3rd October with shivering. During the night, violent fever. On the 4th, about 9 and 12 o'clock, convulsive attacks. After these complete anorexia, yellow-coated tongue, nausea. Emetic; later, infusion of senna. Recovery.

A child of 2 years, on 17th March, ate a large quantity of "sauerkraut," whereupon followed marked flatulent distension and unusual sleepiness. These symptoms were still present on the morning of the 18th. Suddenly nausea and vomiting took place, and, about 11 o'clock, violent convulsive fits, which lasted with short intervals till 2 o'clock. Two enemata were given, which brought away some hard scybala. About 2·30, I found the child still completely unconscious—the eyelids firmly closed and difficult to open—the jaws clenched, the respiration accompanied by a rattling sound and irregular, from time to time still some slight spasmodic movements of the extremities, pulse 120, very full. Treatment:—Sinapism, cold compresses to the head, 4 leeches behind the ears, calomel, gr. i. every 2 hours. At 6 o'clock severe after-bleeding, return of consciousness, urine passed, and child wants to eat. Quiet sleep during the last ½ hour. No motion of the bowels. Infus. sennæ ℥ ii. On 19th, after a free evacuation the child feels quite well. The convulsions did not return.

Boy ♂ of 6 years, admitted 30th October, 1882. Epileptic attacks following an attack of diarrhœa, which during the last 24 hours are often repeated with intervals of coma. Loss of consciousness complete, pupils dilated and sluggish, pulse 124,

small and irregular; tongue thickly furred, T. 98·6° F. Irrigation of the intestine, ice-cap to the head. On 31st return of consciousness and speech; no more convulsions. Still repeated vomiting and offensive motions. Purgatives. On 2nd November, quite well.

In the last child we see the coma lasting more than 24 hours after the cessation of the convulsions, and it is just such cases that, on account of the suspicion of meningitis which they arouse, may not only be very disquieting to one beginning practice, but may even cause anxiety to an experienced physician. This happened to myself and a colleague with whom I treated the following case:—

Boy R., 5½ years old, who had frequently before been affected by headache and vomiting owing to dietetic errors. Otherwise perfectly healthy. In December, 1884, he took a violent attack of vomiting and fever after an overloading of the stomach, and next day had 3 severe epileptiform fits, followed by deep coma. This lasted uninterruptedly for almost 3 days with fever, but with regular pulse and without recurrence of the convulsions. In spite of much hesitation, there was still such strong suspicion of meningitis that we next ordered wet-cupping to the neck, an ice-bag, inunction of blue ointment also calomel and infus. sennæ with syr. rhamni. The motions, which were passed in bed, were always extremely offensive and contained numerous scybalæ. After 3 days the boy awakened, looked about him intelligently, recognised those around him, but was completely aphasic, though without paralysis of any part of the body. No more fever. After a few days he began to speak a few words with difficulty, as if memory failed him. There was also still furred tongue and small appetite (acid. hydrochlor.). After about 10 days, complete recovery.

The course was quicker and more favourable in the following case, which shows at the same time that under such circumstances the convulsions may be entirely absent, and in place of them we may find only drowsiness, aphasia, &c.

In October, 1882, I was consulted by a medical friend about a boy of 6 years, who some days before had taken a large quantity of raw fruit, cakes, &c., and had been attacked during the following night by profuse diarrhœa. Copious evacuation of partially digested matter took place involuntarily during semi-unconsciousness. Towards morning, fever, clouding of intelligence, aphasia, and a staring look. At midday increase of these symptoms to such an extent that the suspicion of brain-disease

became very strong. After calomel, several more green, slimy motions. During the evening, return of perception and of speech. Next day, after quiet sleep, complete recovery, except that the tongue was furred.

The following case, however (certainly a very unusual one), shows also that consciousness may remain quite unaffected and the speech alone be interfered with in the form of aphasia.

On 12th July, 1881, a boy 3 years was brought to the polyclinic, who—according to the account of his alarmed mother—had been quite well till an hour before, but since that time had not been able to speak a single word. It was, in fact, impossible to make the child speak. Only on being pinched he uttered the sound, "Au!" His look was unusually staring, but otherwise nothing morbid could be discovered. After half an hour violent vomiting took place suddenly, whereupon several cherries were brought up almost quite unchanged, and immediately after speech was quite restored.[1]

Cases like this of pure aphasia can hardly be explained otherwise than by reflex irritation from the stomach, while for the more complicated cases (convulsions, coma, &c.) the "auto-infection" (first suggested by Senator[2]) of the organism by the poisonous products (ptomaines) formed in the intestine, may come into consideration.[3]

From the cases I have given, you may see at the same time the kind of treatment to employ—emetics and purgatives, calomel and castor oil, infus. sennæ, &c. (Form. 6 and 7) are the remedies given to remove the irritating "materia peccans" from the stomach and intestines. When the abdomen is more extremely distended and tense, it is well—even while the cerebral symptoms continue—to give an enema of milk and honey (2 : 1), or irrigations of cold water, in order to empty the intestine as quickly as possible. Bloodletting is not generally to be recommended.[4] Although I employed it in a few of the cases given, it was either where I was afraid that the malady

[1] A quite analogous case was observed by Siegmund (*Berl. klin. Wochenschr.*, 1883, S. 335).

[2] *Klin. Wochenschr.*, 1868, No. 24: *Zeitschr. f. klin. Med.*, Bd. vii., H. 3.

[3] The idea that acetone had something to do with the occurrence of fits seems according to Baginsky's investigations (*Arch. f. Kinderheilk.*, ix., 1) to be erroneous.

[4] I have never yet seen occasion to use the stomach-pump, as recommended by Corby (*Hirsch und Virchow's Jahresbericht*, 1878, ii., S. 626) in order to let out gas and fluids and, if need be, to inject an emetic. But if the stomach was uch distended I should not hesitate to try this proceeding.

might turn out to be meningitis, or—in cases where the diagnosis was certain—because we were obliged to assume a considerable amount of venous engorgement in the brain and meninges, on account of the extremely long duration of the convulsions (*e.g.* from 11 to 2 o'clock in the third case). In order to avert, as far as possible, the evil effect of this, I ordered the application of a few leeches; and I recommend this procedure to you in similar cases, which are by no means rare.

> In a child of 1½ years, who had eaten large quantities of turnip, convulsions set in towards evening, which, with short intervals, lasted till the morning, when violent vomiting and diarrhœa occurred spontaneously. In a boy of 4 years the attacks, along with the comatose intervals, lasted 24 hours, and caused great anxiety.

A few leeches to the head, and cold compresses or an ice-bag in addition, are to be recommended under these circumstances as a prophylactic means, but only in robust children. In general, however, the application of an ice-bag is quite sufficient.

The old tradition that internal parasites (ascarides, oxyurides, and tæniæ), like dyspeptic conditions, frequently give rise to convulsions, still lingers in the belief of mothers and even of many physicians. I shall by no means deny the possibility of this connection, seeing that there are so many cases described, and especially as a few observations of this kind have been quite recently recorded; but my personal experience is quite at fault here. I have not seen a single case of eclampsia which I could trace with certainty to the irritation of worms, but I am quite ready to admit that the use of anthelminthics is advisable for children in whom worms are known to have been present previously, or at least are suspected. Just as little have I ever an opportunity of discovering foreign bodies in the ear, skin or nasal cavity causing the fits—as others have reported. But I shall give you later on an example in which the irritation of small concretions in the uropoietic organs was the cause of the convulsions. You will therefore do well in doubtful cases to have those things in mind and investigate accordingly.

A febrile condition preceding the eclamptic attack and continuing after it is especially significant for diagnosing its etiology. Even in the cases of dyspeptic convulsions of which we have just spoken, fever may be present, but under these

circumstances you must never neglect the examination of other organs, for acute diseases of these not uncommonly begin in childhood with fever and violent convulsions. In the first rank I would here name primary pneumonia, and next to it pleurisy and enteritis, and in considering these diseases I shall give you examples of this mode of onset. I will only remark here that the diagnosis of pneumonia beginning in this manner is often at first difficult and even impossible, because on physical examination of the chest at this early stage there is as yet no real abnormality to be discovered; so that we may remain in uncertainty for some days as to whether it may not be an acute inflammatory disease of the brain. As soon, however, as the symptoms of respiratory disease become more prominent, the cerebral symptoms usually become less so, and we recognise that the latter were only the prelude to the pneumonia. It is not quite clear in what manner the convulsions arise in such cases. They might just as well be ascribed to reflex irritation proceeding from the lungs, the pleura or the intestine, as to the high temperature which in children of an irritable habit is of itself sufficient to produce convulsions. In two children of 6 and 8 years, who were extremely feverish from simple tonsillitis, I witnessed repeated attacks of eclampsia take place on the first day, causing great anxiety to the friends and to myself. On the following day, however, the fits ceased along with the fever, and did not return. In one of the cases the parents stated that this had occurred two or three times before. Faure[1] publishes a similar observation from Barthez's clinique. Thus we see that even trivial local affections, if only they are preceded by intense fever, may at first be accompanied by eclampsia, and it is therefore only natural to ascribe it to the fever. If we consider that the rigor of fever is itself a convulsive phenomenon, we shall not find anything very surprising in the fact that in very irritable subjects it becomes aggravated into regular convulsive fits. The convulsions which at times occur in the initial stages of acute infectious diseases (measles, small-pox, scarlet fever) probably belong to the same febrile category. But it is conceivable that in them the infective material circulating in the blood may contribute its own share.

[1] Faure, "De l'expectation et du régime dans les maladies aigues des enfants," *Thèse:* Paris, 1866, p. 12.

In all these cases the convulsions can only be treated symptomatically by the application of an ice-bag to the head, cold baths (88°—81·5° F.) evacuant enemata and mild purgatives. One must just wait and see what will develop from these initial convulsions, and direct the further treatment accordingly.

Uræmia, to which I shall return under *Nephritis*, is another of the acute diseases which begin with violent convulsions, and intermittent fever (in children especially with tolerable frequency) is another which may suddenly commence in the same way. As a rule it is only the first attack that takes this course, and it may easily be mistaken for simple eclampsia, until the appearance of the ordinary intermittent attacks discloses the error. Far more rarely, even the first or second attack of this convulsive form of the disease, presents all the symptoms of pernicious intermittent fever, which is an extremely dangerous condition.

The following case observed by me will illustrate this to you[1]:—

> A healthy girl of 9 years complained on the Friday before Whitsunday, 1871, about 10 A.M. for the first time of double vision; soon after, of cold hands; her intelligence was also soon affected. She no longer recognised those around her, but mistook one person for another, and about 1 o'clock took a convulsive fit, which—according to the description—seemed to be completely epileptiform. This, with intervals of coma, lasted for about an hour. Then sleep came on; after which the patient—apart from slight headache—appeared quite well. As she had never before had an attack of this kind, and no epilepsy had occurred in her family, and as indigestion also could certainly be excluded, intermittent fever occurred to me, all the more readily because the family lived on the canal, where malarious diseases are not uncommon. Next day passed without any event. On Sunday, at 4 P.M., however, the attack recurred (tertian). I was myself present when the child began to wander in her speech. Suddenly she ceased to recognise those around her, mistaking one person for another. Her hands were cold, and in the free intervals which were observed she complained of giddiness and double vision. An hour afterwards another violent epileptiform attack occurred, lasting continuously till 6 o'clock. I now found the child cyanotic, pulse small and very rapid; and—as I hesitated to use chloroform under these circumstances—I gave first an injection of morph. acetat., gr. ½. Soon after, however, encouraged by the co-operation of an expe-

[1] *Berl. klin. Wochenschr.*, 1873, No. 26.

rienced colleague, I gave chloroform inhalations. The very first inhalations were sufficient to arrest the convulsions. The child became quiet, the cyanosis disappeared, and peaceful sleep set in, lasted 10 hours, and the child was quite well when she awoke.

As I was now convinced that I had to do with a case of intermittens perniciosa, I at once ordered quin. sulph., grs. ivss. every 3 hours (grs. xxiiss. on the first day), in order to prevent a third attack, if possible. On the second day she had gr. iiss. every 2 hours; on the 3rd, gr. $1\frac{3}{4}$—so that in the first week after the attack about 92 grs. of quinine were given. The result was that no further attack occurred; only on Tuesday at midday the child had headache and giddiness and commenced to shiver; but this condition did not last beyond 20 minutes. Since that time I have seen this patient frequently enough to be able to answer for her perfect health.

In addition to the causes already described, psychical causes also may produce convulsions in children with a very irritable nervous system, especially a sudden start, more rarely fear; and I would refer many cases in which convulsions have ensued after a fall on the head, more to the fright than to the injury. Under these circumstances it is not always limited to one attack; on the contrary, there may be frequent recurrences. Thus on 5th January, 1878, a child of one year whom I have already mentioned, who had been perfectly well and in whose family epilepsy was unknown, was brought to me at the polyclinic. Five months before, the child while sucking had bitten its mother's breast with its two incisors (which were prematurely developed), and, when she screamed violently, at once fell into severe convulsions, its whole body being affected in the fit. These convulsions had since then been repeated four times without any cause and without any tendency to rickets being observable. Such cases cannot but rouse anxiety lest the disease should become habitual and develop into epilepsy.[1] Experience shows that epilepsy very frequently begins in childhood. Surely, therefore, no one can pretend to determine with certainty whether convulsive attacks—especially those which reveal no cause—have only a temporary significance, or indicate

[1] Among the cases of reflex epilepsy in children, one published by Demme (*Jahresber. des Berner Kinderspitals*, 1879) is especially remarkable. The patient was a boy of 7 years whose attacks at once disappeared after the extirpation of a rectal polypus. An attempt, which was made on the day before the operation, to draw out the polypus with the point of the index finger, caused an epileptic attack lasting about 3 minutes.

the beginning of habitual epilepsy. In these cases the attacks are not invariably continuous, for the convulsions may come on in infancy, and make long pauses before they reappear in riper years. Among others I observed a boy of 12 who had suffered from epileptic attacks in his second and third year, then remained unaffected till his 11th year, and after the interval of another year was again attacked by epilepsy. In this case mental hebetude seemed to be the aura; and in this condition he was still able to go down to the street, where he fell down in convulsions. The diagnostic features of inveterate epilepsy, namely, diminution of brain-energy, loss of memory and alteration of character are not to be expected at the commencement of the malady in children (except in congenital atrophy of the brain accompanied by epileptic fits), and therefore can scarcely be of any value for distinguishing a transitory eclampsia from incipient epilepsy. Among the cases of real epilepsy which I have seen develop in childhood, the following appear to me worthy of notice:—

In a boy of 10 years, who after an attack of "inflammation of the brain" in his 2nd year, had retained hallucinations (especially the constant reappearance of a sheep). Epileptic attacks came on in the end of the 3rd year, with a sensation of giddiness as the aura.

In two other cases, the attacks commenced 5 weeks and 2 months respectively after a head-injury (a blow against a tree and bruise by a carriage-wheel). Both children complained frequently of headaches, and were somewhat backward in intelligence, and in the 2nd case the attacks were preceded by nausea as an aura.

In a child of 3 years epileptic attacks had commenced one year subsequent to a fall, when a knitting-needle had entered beneath the chin and penetrated to the floor of the mouth.

A child of 3 years took his first fit a few hours after seeing the corpse of a favourite brother.

A healthy-looking little girl of 13 years had had a convulsive attack in the first year of life, which recurred in the 3rd and 12th years. She learnt to speak first in her 5th year. Since the 7th year, attacks of a peculiar convulsive character in the throat, namely, the sensation of choking in the larynx occurring in fits; expirations rapidly following on one another; staring look, and slight mental hebetude. Every attack ended in violent palpitation of the heart, after lasting a few seconds. Sometimes 10—12 such fits occurred in one day, while on the other hand some weeks passed without any occurring. Intelligence and memory weak,

senseless laughing often came on. Frequent pain in the neck. No molimina menstrualia noticed. It is said that after violent epistaxis these attacks ceased for some time. Local blood-letting from the neck and purgatives had no effect, for instead of these attacks regular epileptic paroxysms soon set in, preceded by vomiting and spasms in the throat as aura; so that the latter, which had lasted for about 6 years only as abortive attacks now turned out to be an aura in the sphere of the vagus.

A girl of 12 years had suffered for the last 5 years from epilepsy. The aura in every fit was a noise in the ears—especially in the right ear—which wakened her out of sleep. The attacks occurred only in the night-time.

In a boy of 14, who had been an epileptic for several years, the aura consisted in fits of winking with both eyelids and nodding of the head. Before the epilepsy commenced this aura had existed as a separate disease in fits which sometimes lasted for an hour.

A child of 3 years, whose brother is imbecile, suffered for some months from epileptic fits, with the following aura—in the midst of play the child would suddenly run to a certain point, staring and apparently blind, and then fell to the ground unconscious, with convulsive movements of the muscles of the eyes and of the arms.

A girl of 11 years, in whose family mania and epilepsy are hereditary, had after a severe fright begun (9 months ago) to be delirious at night and to sing loudly. Later on there were added painful spasmodic contractions of the legs, gradually also of the arms, face, and eyes. Finally there set in regular epileptic attacks both by day and night, but never as yet occurring out-of-doors. Any mental strain or slight punishment readily produced an attack. At night she often suffered from bulimia and then greedily swallowed food without knowing what she was doing.

In a healthy girl of 12, with no hereditary tendency, 5 epileptic fits had taken place in the last six months or so. These occurred only when the eyes were shut, *e.g.* when washing herself or when asleep. Then there would set in convulsive movements of both arms, more rarely of the legs; and we were able to produce this aura in the ward by telling the patient to shut her eyes. When her eyes were opened the aura also disappeared. The aura always began with a tremor of the eyelids. Was the action of the light important to the brain? Further course unknown.

I consider it superfluous to discuss epilepsy here in detail, as it differs in childhood in no way from the same disease in adults. The cases I have given—only some of which were hereditary—illustrate especially the various kinds of aura, which in a few of them existed for years as an apparently independent disease, and only revealed their real nature later on by the development of

regular fits. I therefore advise you in all cases where nervous symptoms of this kind occur in children otherwise healthy—be it twitchings of single limbs, of the head or eyes, hallucinations or any other psychical abnormalities—not to take the matter lightly, but to bear in mind that they may be the premonitory symptoms of epilepsy. In some of my cases I have also observed delirium not only after the attacks, but in the intervals also; and more rarely, the so-called "somnambulistic" symptoms, such as getting out of bed during the night, creeping under the table, climbing on the top of the furniture, all when half asleep, and with the consciousness either entirely, or at least partly, in abeyance; likewise an irresistible impulse to jump about the room, to climb and to sing with a loud voice. Sometimes the delirium reaches such a height as to be called "ecstasy": as, for instance, in a girl of 11 years who in the intervals appeared quite stupid, and kept on repeating the word "what?" You must remember that all these were cases of real epilepsy and not of "hysterical" affections, which we shall discuss presently, and which, indeed, occurs far more frequently in this form than real epilepsy does. Such symptoms, however, are by no means peculiar to epilepsy as seen in childhood, but are observed in adults also. All that I wanted here was to make you acquainted with the difficulties which we encounter in many cases in diagnosing them from simple eclampsia.[1]

In conclusion I have to add a few therapeutic observations, since those already given (p. 171) are only concerned with cases of convulsions where there are decided causal indications. Unfortunately, however, there are many convulsions the proximate cause of which is not to be found. This is especially the case in those so common in rickety children, with or without laryngeal spasm. In these cases the treatment of the rickets is certainly, I think, the chief matter; and when the convulsive attacks only occur seldom and in a mild form, I always think it best to disregard them and to give iron, codliver oil, and lukewarm baths with salt or decoction of malt: to this I shall return in speaking of rickets. You will find cases, however, often enough in your practice in which the convulsions are so numerous

[1] As to the influence of drunkenness on the part of the parents, or the excessive use of alcohol by the child in occasioning epilepsy, *cf.* Demme, 22. *Jahresbericht des Jenner'schen Kinderspitals: Bern.*, 1885, I have not myself as yet met with a single well authenticated case of this kind.

and severe that they—at least for the moment—may be regarded as forming the chief disease, and demand to be considered before anything else. I must freely confess, that in such circumstances our art has no great results to boast of. I know of no remedy certain to prevent the return of the attacks; and you will therefore pardon me if I serve up to you once more the confused medley of inefficient drugs which have been recommended for centuries. Many physicians to this day swear by the preparations of zinc, especially the oxide, sulphate and valerianate. But from my own experience I cannot recognise these remedies as superior in any way to many others which have become obsolete; and in fact I have long ago given them up, along with asafœtida and musk. Of greater importance appear to be two remedies which have come largely into use in recent times—bromide of potash and chloral. I am very far from ascribing to them a specific action; and unfortunately I have had cases in which they did little or no good. On the other hand we cannot deny that these remedies have a quieting influence on the irritated nervous system; and they are therefore always worth a trial. I prescribe pot. brom. grs. ivss—xv. (according to age) thrice daily (Form. 8). Chloral hydrate internally gr. i.—ii., or in the form of enemata in doses of grs. iii.—viii. (Form. 9). With these doses, even in childhood, no soporific effect follows, as a rule; such effects, moreover, need not be feared in the circumstances because children with a tendency to eclampsia are usually apt to be rather sleepless or at least very restless and nervous. Therefore when the restlessness and sleeplessness are very great and the fits constantly recurring, it may be necessary to order a full dose of chloral (grs. xv.) or even to give morphia.

II. *Laryngeal Spasm.*

Among the convulsive conditions of childhood affecting a limited nervous area, but having a tendency to become general at any moment, laryngeal spasm is by far the most important. It is commoner in boys than in girls, and occurs almost exclusively between the 6th and 24th months. Beyond this age I have hardly ever observed laryngeal spasm; but often

before the 6th month, in children of 5—6 weeks or even only a few days old.

You may in fact observe, even in a healthy child, many of the features of this affection, when in the midst of violent and noisy crying there is a sudden quiet and the child lies with its head thrown back, its face dark red or somewhat cyanotic, the breathing arrested and the limbs stiffly extended. Excessive screaming, along with passionate excitement, seems in such a case to cause a spasm of certain of the muscles of respiration, which as a rule, gives place to an entirely normal condition after a few seconds. This is analogous to other spasms occasioned by over-straining of the affected muscles (writer's, shoemaker's, and milker's cramp &c.). When morbid conditions exist there is not necessarily any such cause for the convulsions, for often enough we see the attacks take place during complete rest, even on waking from sleep. But any overstraining of the respiratory organs, especially screaming, always acts in this way—such as is due to psychical influences, anger and fright. In order to demonstrate an attack to my pupils in the ward I usually make the child scream by pressing on the larynx, and this almost always succeeds.

The simplest form of the attack consists in a momentary stoppage of the breathing, in apnœa lasting only a few seconds, followed by a few crowing or whistling inspirations. Between this and the severest form there are countless gradations, which it would be impossible to describe individually. The sudden stopping of the breathing is common to all. The child generally throws itself back violently; its face is pale with a somewhat cyanotic tinge round the mouth and nostrils, the arms and legs are often stretched out, the fingers are doubled up into the palm, and the toes sometimes flexed upon the soles or else extended. The return of respiration is announced by laboured and whistling breaths, first faint, afterwards louder. With these the attack ends after lasting some seconds. The occurrence of the "crowing" indicates the abatement of the paroxysm seeing that it is caused by the air rushing through the still contracted glottis. So long as the spasm remains, there can be no breathing at all, and consequently no "crowing." Those attacks therefore are most to be dreaded in which the apnœa is protracted beyond the usual time and there is no whistling sound at all.

In such the complete stoppage of the respiration may be fatal almost instantaneously from asphyxia; and in estimating the prognosis this fact must be kept in mind from the beginning. For a child may suffer for weeks from slight transitory attacks which scarcely arouse anxiety, until all of a sudden and quite unexpectedly an attack occurs causing instant death. Be therefore always on your guard in your practice, and in every attack of laryngeal spasm that you meet with—however slight it may appear to be—forewarn the relatives of the possibility of a fatal issue.

But the fact must not be overlooked that this convulsive affection may extend further. The name "laryngeal spasm" has become naturalised, but, strictly speaking, it is far from being correct. For, although in slighter degrees the whole attack may consist solely in a more or less transitory spasm of the arytenoid muscles (*i.e.* exclusively in the sphere of the recurrent laryngeal); still, we very often see the spasm passing on to other parts of the respiratory system (muscles of thorax, diaphragm), and in this way there may be occasioned complete apnœa, or striking irregularities of breathing (*e.g.* inspirations following rapidly on one another without any noticeable expirations). In addition to this, the ocular nerves often enough participate (turning up of the eyeballs) and the spreading of the irritation to a wider area is indicated by the contractions of the muscles of the fingers and toes, so often observed in such attacks, or even of the flexors of the forearm, which I have myself seen very distinctly; *e.g.* in a boy of 5 months. Once or twice I have even observed trismus-like contractions of the masseters and temporal muscles during the attacks, and in these cases the attack only wanted loss of sensation and consciousness to stamp it as eclampsia. So far as an opinion on the matter can be attempted—considering the shortness of attack and the tender age of the patient—I really believe that in severe cases of spasm of the glottis there must be a brief period during which consciousness is lost. At any rate, cases do occur in which children after an attack lie for 10—15 minutes as if in a stupor. Therefore, it need not appear surprising that attacks of laryngeal spasm very often alternate with fits of eclampsia, and that spasmus glottidis often appears first, and general convulsions speedily follow. I have sometimes observed the above-mentioned

contracture of the fingers and toes persisting during the intervals. The combination of laryngeal spasms with eclampsia is so frequent that in an earlier work I was able to distinguish 46 cases out of 61, as cases in which both affections occurred, while only 15 presented laryngeal spasm alone. Since then, the number of my observations has increased very much, but the proportion I have given has always remained the same. In every case of laryngeal spasm, therefore, I usually forewarn the parents that general convulsions may suddenly set in.

You will remember the connection which exists between eclampsia and rachitis, whether the former occurs alone or is combined with laryngeal spasm (p. 167). This connection, in rickets especially, is so well marked that in every case I at once examine the cranial bones and the epiphyses of the ribs and extremities, and I very rarely fail to find rachitic changes in them. Even in infants of 3—4 months—in whom rickety changes are not very common—I have repeatedly found that where there was laryngeal spasm, the cranial sutures were widely open, and the bones were soft near them and yielded to pressure, the epiphyses of the ribs being already distinctly swollen. From my own experience I can boldly assert that at least two thirds of the children who suffer from laryngeal spasm are rickety, and I must therefore regard this connection as something more than a mere chance coincidence. This also explains the family tendency to spasmus glottidis occasionally met with. Only in exceptional cases have I seen rickets limited to the cranial bones, the ossification of which was then considerably retarded. For example, in a sickly little boy of 7 months who had previously been syphilitic, the large size of the head and wide sutures and fontanelles, combined with the frequent attacks of laryngeal spasms and eclampsia, suggested hydrocephalus; but this apprehension was proved groundless by the patient's complete recovery. When Elsässer wrote his book on "Cranio-tabes"—of which I shall have more to say under *Rickets*—he allowed himself to be misled by the softness and partial wearing away of the cranial bones (especially of the occiput and the parietal bones) into making laryngeal spasm ("tetanus apnoïcus" as he inappropriately calls it) dependent on this disease of the bone; and he assumed that when the children were lying, the brain was not sufficiently protected against pressure by the softened bones. I can assure you

that I have examined hundreds of cases for cranio-tabes, and have only very rarely found the condition described by Elsässer. At any rate, we must also regard it as a rachitic symptom, and only from this point of view is its connection with spasmus glottidis to be considered. The frequency of the attacks—which in general admits of great variation—may be incredibly great in rickety children. In the course of a single day, 20 or even 30 attacks not unfrequently occur. Every fright, every attempt to drink, every fit of crying occasions one, and it is in such states of extreme irritability that we have to fear the occurrence of general convulsions at any moment. If this condition lasts for weeks or months, getting alternately better and worse but without free intervals of any duration, complete exhaustion may ensue, to which the child at last succumbs.

A boy of one year, very anæmic and rickety, when I first saw him in December, 1869, had already been suffering for 2 months from attacks of spasmus glottidis, which latterly had alternated with eclampsia. During the last few weeks the latter had become very prominent, so that sometimes 15—16 attacks of convulsions occurred within the 24 hours. The child was evidently becoming collapsed. The most diverse remedies—even crucial incisions into the gums (which I allowed in deference to the physician in charge)—were quite unsuccessful. Only exceptionally did intervals of 12—18 hours occur. From the middle of December to the end of March, more than 600 fits of eclampsia were observed, alternating with laryngeal spasm. The constant current was also quite without effect; and the child died in a state of collapse in the beginning of May, after the cutting of the first incisor tooth.

In other cases death occurs suddenly, as I have already mentioned, from complete apnœa; but this mode of termination—according to my experience—is not so common as you might think. When it does occur, it is usually extremely sudden, in the midst of perfect health—just as in cases where a foreign body has found its way into the glottis. The already-mentioned (p. 143) sucking-in and turning-up of the tongue towards the hard palate have also been blamed for this, and I will not deny that the forcible inspirations, which occur especially when the spasm is becoming relaxed, render such an occurrence possible, since I have myself clearly observed it in one case.

A rickety child of one year, in my ward, suffering from spasmus glottidis, was being auscultated by me on its back on account of

bronchial catarrh; and the nurse was making it bend very much forward. Suddenly such a violent attack of apnœa set in that the child at once became very cyanotic. Sprinkling with cold water caused the respiration to return, but in spite of the whistling and laborious breathing, the condition threatened every moment to end fatally. I quickly passed my finger into the child's mouth, and found the tip of the tongue so firmly pressed against the palate that I was obliged to use considerable force in order to get past the root of the tongue. I then drew it quickly forward; and the respiration at once resumed its normal character.

Cases such as this may induce one to regard the sucking-in of the tongue as the usual cause of apnœic symptoms in laryngeal spasm. I consider this as quite unjustifiable, however, for in very many cases I found on examination of the mouth—which was almost always open—that the position of the tongue was perfectly normal. Its being sucked in is therefore assuredly only an accidental and rare complication; but still it must not be over-looked, for—as the above case shows—it may be a very important circumstance in the treatment.

There is a third class of cases, in which death finally ensues from a violent and protracted attack of eclampsia, or from its consequences. In the post-mortem, which I have performed in several cases of this kind, extreme venous congestion of the pia mater, was always found, generally also of the brain; once or twice œdema of the pia mater and serous effusion into the ventricles. But I regard these as the results simply of considerable venous engorgements occuring during the convulsions, for they were always found most strongly marked where, in addition to spasmus glottidis and eclampsia, there was a third factor favouring engorgement (viz. whooping-cough). I have observed this complication not unfrequently, and it either accompanied convulsive attacks which had already lasted a considerable time, or the whooping-cough came first and the spasmus glottidis only set in when it was abating. The complication is of course merely accidental, for whooping-cough can only occur from specific infection. But the combination of these diseases with one another favours the occurrence of general convulsions very much indeed, and in my experience justifies an unfavourable prognosis.

To the uncertain relationship between rachitis and spasmus glottidis I need not return after what has been already said

(p. 167). The fact remains, although its explanation is wanting, and all attempts at explanation—*e.g.* the most recent by Oppenheimer[1], are strained and highly contestable. Poorly nourished delicate children—and, of course, those of the poor especially—are most apt to be affected; but well-developed apparently thriving children are by no means exempt. If only the tendency is present, the spasm occurs either spontaneously or from reflex irritation, and in this respect the eruption of the teeth (p. 155) is undeniably of some importance, although it may be very much overestimated. The same may be said of derangements of digestion, of constipation, and of diarrhœa.

> E. R., 11 months old, weaned in middle of March, 1875. A few days after, dyspeptic diarrhœa and at the same time attacks of spasmus glottidis, along with almost continuous contracture of the fingers and toes—which lasted during the intervals also. Violent screaming, bad temper. Frequent attacks, even during sleep. After lukewarm baths and small doses of calomel, constipation set in so that enemata became necessary. On the 28th, tongue thickly coated, anorexia, offensive diarrhœa again, along with which the attacks of laryngeal spasm (which were already much diminished) commenced anew very violently. Hydrochloric acid caused rapid improvement. Nestlé's food given, which was well borne, and was continued from this time. After 4 weeks, recovery, only slight rachitic bone-changes being left.

Among the reflex causes, the influence of cold and of catarrh of the upper air-passages must also be mentioned as very important. This is shown by the special prevalence of the malady during the cold season of the year. I have always observed by far the greatest number of cases—in the polyclinic as well as in private practice—during the months from January to May inclusive; and I therefore urgently warn the mothers of children with a tendency to laryngeal spasm, not to expose them to cold air. A relapse of the disease may at once result, especially if there is catarrh of the larynx or trachea. In these cases the inspiratory "crowing" acquires a harsh character, which is easily explained by the catarrhal affection of the glottis.

All these causes, and perhaps also others less evident, may produce laryngeal spasm even in children who are suffering from no rickety conditions. But as far as my experience goes, these cases are infinitely less common than those complicated with rickets. The high degree of reflex irritability, already normally

[1] *Deutsches Arch. f. klin. Med.*, Bd. xxi. . H. 5 und 6.

present at that age, appears therefore to be greatly intensified by rickets. Anything else that has been written on the etiology of laryngeal spasm is either hypothetical or positively incorrect; particularly the view that the disease arises from enlargement of the thymus gland (asthma thymicum); which view still has its supporters. I could never observe any such enlargement, either at the post-mortem or by percussion during life; and it has been made almost certain by Friedleben's researches that thymus glands, which were formerly thought to be hypertrophied, were perfectly normal.

The prospects with which one approaches the treatment of spasmus glottidis are not very encouraging; as you now know there are dangers for which you must prepare the relatives from the first. On the other hand you can reassure them by telling them that the majority of cases end in complete recovery, although they may last for months owing to repeated relapses. This result will be best attained, in my opinion, by improving the general health, *i.e.* by removing the rickety tendency. I therefore usually make this my chief aim, except where the too frequent recurrence of the attacks calls for special treatment. With regard to the latter I can only repeat what I have said about eclampsia (p. 178). Neither bromide of potash nor chloral have given me reliable results. Even although the success at the beginning of the treatment is sometimes surprising, it is not sufficiently permanent; and we must always be prepared for relapses in spite of the continued use of the remedy. I have seen no good effect from zinc, and I consider the reports of its success to be entirely erroneous. In some cases musk has seemed to me to have a soothing effect, and slightly to diminish the frequency of the attacks; but in others it was absolutely worthless. I have given as a rule tincture of musk, gtt. x. every hour, or every two hours. But when it is desirable to bring to an end as quickly as possible the enormous frequency of the attacks which is exhausting the child, I have no hesitation in employing morphia (Form. 10). Whenever the child becomes quiet and drowsy you should stop the medicine in order to avoid the risk of poisonous symptoms. With proper care, however, I have never known anything of this sort to occur, and with this drug I have frequently had the gratification of quieting the symptoms for a considerable time and rescuing from imminent death

children who had been given up for lost. As to the treatment of the separate attacks, only in rare cases will you be able to practice it; for before you arrive either the fit is over or the child has been suffocated. For this reason also the recommendation of tracheotomy for the emergency can hardly be regarded as practical. It is however advisable to instruct the relatives as to what they should do on the occurrence of an attack. The sprinkling of cold water on the face and chest may at once put a stop to the threatening apnœa, and it should always be tried by the relatives as well as the drawing forward of the tongue already recommended (p. 184). Artificial respiration is more difficult; and it, as well as the faradisation of the phrenic nerve, ought only to be attempted by a medical man.

Attention to the cause of the reflex irritation is the matter which first claims our consideration in cases where the alleviation of symptoms is not an immediate necessity—protection from cold air, attention to any catarrh that may be present, purgatives when there is constipation, anti-dyspeptic remedies when there is dyspeptic diarrhœa. Scarification of the gums, when there is irritation from teething, is—as I have already said—utterly ineffectual. Above all things, however, I recommend to you the treatment of the underlying tendency by anti-rachitic remedies—pure warm air, salt and malt baths, iron and cod-liver oil. Of these I shall speak more fully under *Rickets*.

III. *Idiopathic Contractures.*

You will remember that during attacks of spasmus glottidis spastic contractures of the fingers and toes are often observed, and sometimes persist in the intervals between the attacks. Such contractures may also occur independently of spasmus glottidis and extend to wider areas of the muscular system. They occur in general under the same circumstances as eclamptic attacks; not uncommonly they alternate with these and with laryngeal spasm; and they are either only passing or else may last many hours, even days. Most frequently we find the fingers and toes flexed on the palms and soles; less commonly, extended. Sometimes, however, the joints of the hands and feet are also implicated, or the elbow-joint—so that the forearm

appears flexed upon the humerus, the hand upon the forearm, and the foot upwards or else towards the sole. The crying of the children seems to indicate that the contracture is painful, especially if you try to extend the stiffly contracted muscles. In cases where this condition lasts for many hours, days or even weeks, I have not uncommonly observed œdema or a cyanotic tinge of the backs of the hands and feet; and this is to be traced to the pressure of the contracted muscles on the intermuscular veins. Actual ecchymoses such as Bouchut describes, I have only seen in one case, which I shall give presently. At first the contractures only come on in paroxysms; but later on they generally become more or less continuous. In sleep they are usually relaxed; and, like Bouchut, I have very rarely seen cases where this did not occur. The circumstance that they are almost always bilateral, may, as in the case of convulsions (p. 166), be held to indicate their purely nervous and innocent nature. A unilateral onset, on the other hand, must always arouse suspicion of an affection of the opposite side of the brain; and I have often seen these unilateral contractures occurring as a symptom of cerebral tuberculosis, frequently combined with paralysis and tremor. The following case seems indeed to favour the view that unilateral contracture may occur as the result of reflex irritation. Still, owing to its incompleteness, it cannot be regarded as a proof.

> On 24th November, 1876, an otherwise healthy infant of 11 months, was brought to my polyclinic. Five weeks before, the first tooth had appeared, and had been rapidly followed by three others. On examination, I found contracture of the right lower extremity at the hip and knee joints. This was found both when the child was lying on its back, and when we attempted to make it stand; and the foot assumed somewhat the same position as in coxitis, but to a much greater degree. The attempt to extend the limb was difficult and caused loud screaming. According to the mother's statement, this contracture had hitherto appeared before the eruption of every tooth, and had come to an end when the tooth was fully cut. The toes also were stiffly flexed upon the sole. For 14 days there had been dyspeptic diarrhœa and colic. In the middle of December this condition was still unchanged. Unfortunately I lost sight of the child.

In two other cases I have seen contractures of the fingers and toes lasting almost continuously for a week during the eruption

of the upper lateral incisors, and vanishing at once when these had appeared.

The reflex irritation, which is here in the dental nerve, may also be situated in the course of other nerves; and dyspeptic conditions are especially to be mentioned as causes, just as in eclampsia (p. 167)—flatulent distension, hard slimy fæces or dyspeptic diarrhœa. I have myself repeatedly seen cases of this kind, and many such have been described.[1] In rare cases the uropoietic organs are the seat of the reflex irritation.[2]

> Child of 5 months, on the breast, emaciated, said to have cried loudly each time before passing urine ever since birth. On 10th October, 1861, examined for first time. A fit of eclampsia a fortnight before, repeated a week afterwards. The toes of both feet had remained persistently flexed on the sole ever since the first attack. After the second the fingers and knee joints were affected by similar contractures. Stiffness of the affected flexors; attempts to extend very difficult. The muscles of the throat and neck are also rigid, so that the head is moved with difficulty. For the last 3 weeks round fragments of the size of a pin's head recognised as uric acid concretions, have been seen on the diapers which were soaked with dark coloured urine. On various parts of the body there were purpuric spots on the skin, which were said to have appeared immediately after the convulsions. On the 17th after a warm malt bath and the passage of other 3 similar concretions, the contractures ceased, and there were repeated spasmodic contractions in the upper and lower extremities. Œdema of the lower eyelids, of the left leg and foot; fresh purpuric spots of the size of a threepenny-piece on the head and chest. I did not see the child again till 21st November, when I found no trace remaining of the former condition. Two and a half years later, when the child was brought to me again, they had not returned. The treatment consisted in malt baths and small doses of iron.

In this case we find, as I have already mentioned, little hæmorrhages and partial œdema resulting from the persistent contracture. The attack began with convulsions of an eclamptic nature, and the contractures were noticed soon after. You see therefore that the two symptoms had a like significance; and in fact the difference between them consisted solely in the conscious-

[1] *Cf. e.g.*, Koppe "Zur Lehre von der Arthrogryposis des Säuglingsalters," *Archiv f. Kinderheilk.*, Bd. ii., 140. A similar case was observed in an adult by Riegel (*Centralbl.*, 1874, No. 12), in which a cure was obtained by treatment for tapeworm.

[2] *Vide* my *Beiträge zur Kinderheilk., N.F.:* Berlin, 1868, S. 357.

ness being retained in the one and not in the other. If we consider the period of unconsciousness to be due to spasmodic contraction of the small arteries of the brain, causing necessarily arterial anæmia of it, then we should only have to dispose of this affection of the arteries, and the distinction between the eclamptic attacks and the contractures we are speaking of would be practically removed, since a tonic form of the latter also occurs not uncommonly in ordinary convulsive fits. The occasional very long duration of the contractures causes only an apparent difference; since, as we have seen, even convulsive attacks may last for days, separated from one another by short periods of coma. For these reasons I regard contractures as essentially identical with convulsions—as a kind of abortive form of them—and in regard to their etiology and treatment I can only refer you to what was said on the subject of eclampsia. This view is also strengthened by the fact that contractures, like convulsions, are especially common in rickety children. They also, like eclampsia, occasionally have an intermittent type. I have already elsewhere[1] published two cases of this kind.

> In a girl of 3 years there occurred, about 7 o'clock every evening for a fortnight, stiff contractures of all four extremities; the arms were extremely flexed at the elbow joints, the legs were drawn up upon the abdomen, and the feet assumed the form of talipes varus. These attacks were accompanied by a dark red flush on the face, and by loud screaming, and lasted 2 hours, after which the child fell asleep and was quite well till the following evening. Quinine stopped the attacks in a short time.—In another case, affecting a boy of 6 years, there had occurred several days before a stiff contracture of the right sterno-mastoid, with torticollis. This gradually increased every day about 3 P.M., and finally became quite rigid, lasting till evening when it disappeared, not returning till next afternoon. In this case also the use of quinine caused rapid recovery.[2]

Many writers class the contractures which we have been considering, along with tetany. This disease, very obscure in its nature and by no means constant in its symptoms, does occur in children, but is generally more common in adults; and I have therefore no occasion to discuss it here. In my opinion it is well to separate the contractures in children which I have just

[1] *L. c.*, S. 101.

[2] Folliet and Simon (*Revue mens. Fèvr.*, 1883), give quite similar cases of Caput obstipum intermittens.

described entirely from tetany, as they are far more nearly related to eclampsia than to it. In particular, I have never been able, in the cases of idiopathic contracture in children which have come under my notice,[1] to make out the sign of tetany described by Trousseau and confirmed by others—namely, the production of the contracture by pressure on the main artery or the nerve of the affected limb.

Permit me to take this opportunity of adding a few words on the rarest convulsive symptom in childhood, namely, tremor. While in adults and in old people this condition is often observed either as an independent disease (tremor senilis, potatorum, mercurialis, &c.), or as an accompaniment of serious central diseases (paralysis agitans, sclerosis of the spinal cord), I have found tremor in childhood only in typhus and in other serious infectious diseases, but especially affecting paralysed and contracted limbs in tuberculosis of the brain, in basilar meningitis, and other cerebral diseases.[2] I have only once seen a general tremor without any serious symptoms accompanying it; and it ended favourably.

> On 5th February, 1879, a child of 15 months was brought to the polyclinic. It was well nourished and had formerly been healthy but was said to have suffered from inflammation of the lung 4 weeks before. About 14 days before, a continuous trembling had set in in both hands and feet and also in the head (which was usually somewhat retracted, but could easily be moved forwards and from side to side). The child cried very often and for long periods, as if it felt pain; and its cry also, instead of being sustained, had a quavering character, analogous to the tremor of the extremities. Since the beginning of this condition the child had lost the power of standing, but was able to grasp and hold toys in its little trembling hands. It seemed to feel quite well and all the organic functions were normal. After admission into the children's ward, the condition at first remained unchanged. On the 18th, the tremor began to diminish, and by the 20th it had completely disappeared. The treatment consisted in the administration of chloral hydrate (gr. i.).

---

[1] The tetany in infants described by Baginsky (which, however, I can only regard as a symptom of various irritated conditions, sometimes central, sometimes reflex—and not at all as an independent disease) I have never hitherto observed with certainly (*Archiv f. Kinderheilk.*, Bd. vii.).

Demme publishes one interesting case (19. *Jahresber.*, S. 36), of general tremor in a child of 4 months, which lasted till the 11th month and was associated with backwardness of the mental development.

I cannot suggest any cause for the trembling in this extremely rare case. Considering the very good general health of the child, I thought that I must assume some reflex irritation connected with dentition as the cause. I am still of this opinion, in spite of the fact that no teeth were cut while he was under our observation. We may readily imagine that the tooth in process of growth presses upon and irritates the alveolar nerves for a considerable time, thus producing reflex symptoms; but the further advance of the tooth, even before its complete eruption, again frees the nerves from pressure. This view of the origin of tremor forces itself on me when I compare the case with others in which similar movements occur as the result of the said irritation, although these are more strongly marked and are confined to certain groups of muscles. I refer to the spasmodic condition which has recently attracted attention under the name of Spasmus nutans.

IV. *Spasmus Nutans* (Nodding Spasm).

My first observations of this condition were published in the year 1851.[1]

> The children affected were of the age of 6 and 8 months respectively. There was a continuous rocking of the head backwards and forwards, which gave the children the appearance of the well-known Chinese mandarin dolls. In one of the patients the eyes also were occasionally up-turned. During sleep the movements ceased. While awake, they only ceased for a short time if the child's attention was in any way attracted. When they were forcibly checked by holding the head, great restlessness and crying followed. The mouth was hot, the salivary secretion profuse. In both cases the treatment had no effect. The eruption of the teeth (in the first case, of the first incisor) brought immediate recovery after a duration of 3 months and several weeks respectively.

About the same time Faber and Ebert[2] described one or two quite similar cases, and when the attention of medical men was once aroused, it soon appeared that the affection was by no means very rare. From the cases of this kind which I have myself observed since then, I give the following:—

Child of 9 months has had almost continuous nodding move-

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

[2] *Annalen der Charité*, i., 1850.

ments of the head while awake, with slight rotation towards the right. Complete cessation during sleep. Along with the nodding, there was continuous nystagmus of the right eye, in which the movement was strongest towards the inner side. After a few weeks the movements of the head ceased, owing to the eruption of a tooth, while the nystagmus persisted.

Child of one year. Same appearances as in the former case; but instead of nystagmus there was convergent strabismus of the right eye. After a period of cessation following the eruption of 2 teeth, the affection returned, the child having suffered from cholera infantum and bronchial catarrh. Spontaneous recovery after 14 days.

Child of 6 months, brought to the polyclinic, 19th January, 1877. Generally healthy. The convulsive condition had lasted 3 or 4 weeks. At first it was intermittent, now almost continuous, but ceasing entirely during sleep. The movements consisted of a nodding forwards, combined with a slight rotation of the head from right to left. Ocular muscles not affected. The 2 lower central incisors shone through the gum. Further course unknown.

Child of 10 months, healthy, with two teeth. For three months there had been continuous rotating motions of the head from one side to the other, combined with a slight forward nodding movement. Cessation during sleep. If one attracted the child's attention by holding anything in front of it, or forcibly fixed its head, the head-movements ceased, but nystagmus at once commenced in both eyes. Course unknown.

Boy of 1 year, with 7 teeth, examined by me along with a colleague on the 13th February, 1878. For about 14 days frequent weak rotatory movements of the head from right to left, combined with slight nodding. There was almost continuous nystagmus of the left eye. General health good. After a few weeks spontaneous recovery—whether due to a fresh eruption of teeth or not, I could not discover.

Girl of 10 months, healthy. Spasmus nutans for 14 days, with slight rotation of the head towards the right. Movements almost continuous, only ceasing during sleep. Whenever one fixes the head the movements ceased, and a slight nystagmus of the right eye set in; not present at other times. Two incisors in the lower jaw, the upper in process of eruption. Course unknown.

Child of 9 months, rickety, formerly subject to convulsions and laryngeal spasm; now healthy, with normal dentition (2 incisors). The nodding movements in this case were not confined to the head but also affected the whole upper part of the body, occurred in fits several times daily, and were sometimes so violent that the head was bent down almost on to the knees. Spasmodic

movements of the eyes sometimes accompanied the attack. After 14 days the attacks diminished in severity and frequency. Further course unknown.

All these cases show that the movements characteristic of spasmus nutans are almost never confined to the proper muscle of nodding (sterno-mastoid), but also affect the muscles which rotate the head. Nodding and more or less distinct rotation—generally towards the same side—are almost always combined. Indeed in many cases I have found the rotating movement by far the more marked, and the nodding very slight. In almost all the children there were also spasmodic movements of the muscles of the eyes, generally nystagmus—only rarely strabismus or rolling movement; the latter usually affecting both eyes, and only very seldom confined to the eye on the side to which the head is rotated (unilateral spasm). The movements generally continue steadily; much less frequently they come on in fits, and they always cease during sleep. One may usually check the nodding and rotatory movements for the moment by holding the head or by arresting the attention; but when this is done the nystagmus becomes more marked, or appears for the first time, if it was not present before. Only in the last of my cases were the trunk-muscles also affected, so that the whole upper part of the body kept swaying forwards rhythmically after the manner of a Mandarin doll.

That the reflex irritation proceeds from dentition in certain of these children is proved by the disappearance of the spastic symptoms when the teeth come through. Also the age of the little patients—they were all between 6 and 12 months—seems to point to this. The oldest child I have treated with spasmus nutans was in its 3rd year, but its back molars had not yet appeared. On the other hand, I cannot be sure that this was the cause in some of my cases which did not return for further treatment, especially since we may well suppose that other kinds of reflex irritation are quite as likely as teething to excite spasmus nutans. From an anatomical and physiological point of view the frequent combination with nystagmus, less commonly with strabismus, is interesting. This combination has also been mentioned by other observers (Ebert and Demme), and seems to indicate that the root-nuclei of the spinal accessory and of the upper spinal nerves supplying the

affected muscles of the throat and neck, stand in very close relation to those of the ocular nerves (oculo-motorius). A few cases affecting older patients give further confirmation of this relationship.

> On 26th March, 1879, a boy of 12 years was brought to the hospital, who since the second year of his life had been deaf and dumb, as the result of an attack of cerebro-spinal meningitis. His intelligence was unaffected, and he had developed a talent for drawing in an eminent degree. This boy had almost continuous movements of the head, rotating towards the left, combined with slight nodding and with permanent nystagmus—which increased to a remarkable degree whenever one attempted to fix his head. The general health was undisturbed; and I was unable to form a conjecture as to the cause of these symptoms which externally corresponded entirely with those of spasmus nutans, the more so as the boy did not come back.—Quite similar was the case of a boy of 9 years, in whom disturbances of speech were also present, without any cause being discoverable.—Finally, I have observed in a boy of 10, otherwise healthy, an inclination of the head towards the right, with rotatory movements occurring every few minutes. These were always combined with an up-turning of the eyeballs, and had originated about 1½ years before, as the result of a fright. Prolonged application of electricity and residence in the ward produced good results, although not complete recovery.

We must distinguish from the reflex variety a much more serious form of spasmus nutans, depending unquestionably on a central disease of the brain. The very first description given by English authors (Newnham and Willshire) related to such cases, in which mental disturbances and epileptic fits were combined with nodding movements, not only of the head but of the whole upper part of the body. These swaying movements of the body either occurred in fits (at the rate of 50—100 per minute), or else they were more continuous, and in that case less severe. They invariably ended fatally; but, so far as I am aware, there are no reliable accounts of post-mortems. I have myself observed only one case of this nature, in which the morbid movements came on some days after a fall on the occiput, and death suddenly followed; a post-mortem was unfortunately refused.[1] I may also mention here the not uncommon cases in which there occurs, in weak and imbecile children, a frequent falling forward of the upper part of the body,

[1] *Cf.* Hochhalt, *Jahrb. f. Kinderheilk.*, xiii., S. 99.

with a sprawling out of arms and a squinting of the eyes. You will now see that all cases of spasmus nutans are not to be judged in the same way; and I shall have occasion, later on, to refer to a third form which I have sometimes met with as one link in a chain of spastic symptoms which one is accustomed to group together under the name "Chorea Magna."

It will be seen, from the cases given above, that the treatment of the reflex form must be purely expectant. If you wish to try the remedies usually recommended for convulsions (p. 179) you may do so; but remember that they can promise no result till the source of the reflex irritation—which is usually dentition—is removed.

I will also mention in passing that I have frequently noticed more or less continuous swaying movements in the upper part of the body in little children due to the excitement caused by masturbation. These movements are of course voluntary, and must not be confused with the real spasmus nutans. The other local spasmodic affections which occur in children, whether of the muscles of the neck, extremities, or face (the latter especially occur reflexly in connection with eye-diseases), resemble completely those in adults. In these cases also, the influence of dentition is not to be dismissed off-hand. Thus, in a child of a year and a half I observed, twice running, conjunctivitis palpebralis with very violent convulsive closure of both eyelids (the eyes were only opened in the dark); this took place each time during the eruption of a group of teeth, and lasted 2—3 weeks.

I may also be allowed to say a few words on another, very rare spasmodic affection, because I have never yet met with it in adults. I refer to a kind of convulsive laughter, of which I have seen 3 cases, in which the reflex irritation started from the intestinal canal. The 2 first of these—which I have elsewhere described[1]—affected the children of two sisters.

> Child of 4 weeks, on the breast, during the previous 8 days had moderate diarrhœa. Some days before had sudden spasmodic movements of the muscles of the face and trunk, along with which the child laughed loudly, to the mother's great alarm. The fits lasted about 5 minutes, and occurred 3 or 4 times daily. During the intervals there was frequent violent straining, the

[1] *Beiträge zur Kinderheilk., N.F.*, 1868, S. 85.

face was deeply flushed, and the child sobbed without any screaming. Small doses of ipecac. and opium cured these symptoms in 7 days.

Child of 18 days, on the breast. Constipation, extreme restlessness, crying, drawing up the legs, turning up of the eyeballs, clenching of the hands, with loud laughter. Flatulent distension of the abdomen. After warm baths, inunctions of oil, and a dose of castor-oil, copious motions took place. The attacks ceased entirely. They recurred six months after. Recovery under the same treatment.

Child of 3 months. Diarrhœa for about a week. At the same time almost every night spasmodic contractions of the eyes and hands. Also often during the day loud laughing, with "crowing" inspirations in the intervals between. When the diarrhœa was stopped, the fits of laughter ceased, while the spasmodic contractions still sometimes returned. Final recovery.

V.—*Chorea Minor—St. Vitus' Dance.*

Chorea is about the commonest of all the neuroses affecting children after the commencement of the second dentition, *i.e.* from about the 6th year to near puberty. Its occurrence earlier than this is much less common; still, I have myself seen several cases in children of 4 and 5, one even in a girl of 3, following rheumatism. Adults are only exceptionally affected (it being most common in pregnant women); but into this I need not enter here. The number of girls affected exceeds that of boys to a marked degree.

The symptoms of chorea are so peculiar that any one who has seen them once will hardly confuse it with any other convulsive condition. In very well marked cases we find the whole of the child's body in a state of constant restlessness and motion, reminding one of the sprawling movements of a jointed doll, and, indeed, looking not a little comical. As a rule, the extremities are most severely affected; the arms and hands can scarcely be kept still for a moment. They are continually performing sprawling movements and wonderful contortions. The shoulders are sometimes raised, sometimes sunk, the head drawn down to the side, and more or less rotated. The facial muscles also participate, the eyes are alternately shut and opened, the forehead is wrinkled, and quickly smoothed again. The corners of the mouth are twisted to one side or the other. The lower limbs are often still able to support and carry the body; but in many

cases, the sprawling and shambling is so great that walking is more or less difficult, and the children often stumble and fall. In severe cases, indeed, not only walking and standing, but even sitting is rendered impossible. If you ask the child to put out its tongue, it often does so with a jerk, and draws it in again in the same sudden way. But even when the children are able to keep it out for some seconds, you may still always notice distinct muscular contractions in it. The speech also becomes stammering and indistinct, owing to the muscles of the tongue becoming affected; and in severe cases it is quite abolished. In such cases, in spite of the utmost endeavours—during which the muscular movements of the face and of the rest of the body are markedly increased—the children are unable to pronounce a single word, and this is the symptom which usually makes the parents most anxious. The fear, so often expressed, that the child may remain dumb, is never justified, however; and you may with perfect confidence hold out the prospect of complete restoration of the speech. The reflexes, especially the patellar reflex, I have generally found increased—in one girl of 8 years to such a degree that even a slight tap on the tendon caused marked contractions of the quadriceps.

This description of the disease, which applies to a large number of the cases, is subject to many variations, both in degree and distribution of the convulsive movements. Very often the latter are not so severe as those I have described, remaining during the whole course of the disease comparatively weak, and only becoming more troublesome when the patients pass from a state of quiet into one of movement. Even in this slight degree of the disease all parts do not equally participate; whole groups of muscles may remain completely unaffected. From this very slight degree of the disease, up to its strongest development, there are a number of gradations in which you may observe an ever-increasing severity and duration of the spastic movements. Finally, in the most extreme form, their energy and persistence is so great that almost all the muscles, from head to foot, perform an uninterrupted series of the most grotesque movements, which scarcely leave the body at rest for a moment, and jerk it about in every direction—even against the corners of the bed-posts—causing bruises in many places. I have seen patients covered with bruises all over, and have

even known them to be pitched out of bed; and I have frequently had to have the bed padded with thick cushions, in order to prevent more serious injuries. In one case a large abscess formed over the left shoulder-blade, and had to be opened. Swallowing also may be rendered difficult, and in that case some of the food is rejected. In these very violent cases the muscles of the eye also become affected, so that the eyeballs acquire a rolling movement. On the other hand I have never been able distinctly to observe the alternate dilatation and contraction of the pupils mentioned by some authors[1] as coming on quite independently of the influence of light and combined with diminished power of vision during dilatation.

In many cases of chorea you find the movements much more marked on one side of the body than the other, or the disease may only affect one side, while the other remains entirely unaffected (hemichorea). This limitation is either seen only at the beginning, or persists till the end of the disease. Still, a short time ago I saw a boy of 14 whose extremities on the right side were very severely affected during an attack of chorea lasting 9 weeks, while those on the left side did not show a trace of the disease. The muscular bundles of the tongue, exhibit distinct movements on both sides, even in cases of hemichorea, and this is to be explained by the numerous crossings of these bundles. The apprehensions usually aroused by the occurrence of convulsions, or contractures on one side only (p. 166) need not—as far as my experience goes—be entertained in hemichorea, to which in general I can attach no more serious importance than to that occurring on both sides.

The movements of chorea, even in the most violent cases, are hardly ever equally sustained, for they vary in intensity from day to day, and from hour to hour. Often when we think there is a distinct improvement, the disease suddenly takes another bad turn. Under all circumstances, any purposive movement tends to make the chorea worse; any attempt to write, to take hold of small articles, to raise the arms above the head, &c., causes the choreic movements to increase considerably. Even looking fixedly at anything may have the same effect, as I have observed in one case, which was distinguished by the fact

[1] Cadet de Gassicourt, *Traité clinique des maladies de l'enfance*, T. ii., p. 215: Paris, 1882.

that the ocular muscles were affected. These children are, therefore, quite unfit for all occupations requiring the use of their fingers—writing, sewing, playing the piano, &c.; when they attempt to write, the pen jerks about, spluttering the ink over the paper. In severe cases every attempt to speak or to sit up causes most violent aggravation of the symptoms, and even passive movements—such as the attempt to raise the children out of the horizontal position, &c.—may occasion very severe convulsive movements. Many are unable to eat without assistance, and have to be fed because they are unable to hold the spoon firmly, or can only bring it to the mouth in a round-about way, spilling the contents in the process. Every "contageous" excitement—*e.g.* laughing—at once produces a lively grimace (at any rate in the most severe forms of the disease), and also an increase of all the movements. Embarrassment produces the same effect, and also the consciousness of being observed; although a few patients under those very circumstances have more control of the muscular restlessness than others. In almost every case, however, quiet sleep produces a complete cessation of the movements; even when very violent they cease then, and do not begin again until the child awakens. Only in a very few cases have I known them to persist during sleep, and then they were but slight; the children threw themselves restlessly hither and thither with slight sprawling movements. The conditions of these exceptional cases, however, are unknown to me, and they cannot be held to interfere with the general rule. It is most important that the sleep be quiet and deep, for should it be disturbed the movements may certainly continue throughout, and even with great severity. In this we see a definite indication for treatment, namely, to procure quiet nights for the child, so as to arrest the movements at least for a number of hours. Fits of terror with a feeling of oppression, and a somewhat irregular action of the heart, which came on in a girl of 11 years during the first sleep (although the heart appeared normal) lasted half an hour, and aggravated the choreic movements, but did not delay complete recovery. I have always been struck by the fact that in spite of the violent movements going on all day long, there was no appearance of the child's being fatigued. If you just try to imagine such violent movements being carried on for so long voluntarily, you will at once see

that the thing, if not altogether impossible, would certainly produce the most extreme exhaustion. In a few severe cases where we were able to take temperature-observations (there was always a risk of the thermometer being broken), we were unable to make out any increase of temperature, in spite of the prolonged violence of the attack.

These are the main features in the clinical picture of chorea. All other symptoms which have been described, I regard as neither characteristic nor well-established. Among these we have *e.g.* the tenderness of the spinous processes of certain cervical vertebræ, especially of the upper ones; or again, the possibility of increasing the morbid movements by pressure on certain nerves (the brachial plexus or the crural nerve). Apart from the choreic movements, the general health of the children is excellent, their functions are in good order, and although some of them appear pale and delicate, this is by no means either a constant or necessary feature. Sensory disturbances almost never occur. Two cases (girls of 11 and 12) in which along with hemichorea I observed anæsthesia and analgesia respectively of one side of the body and alteration of the psychical condition, had such an appearance of hysteria that I was inclined to regard them not as ordinary chorea, but rather the form described by Trousseau as "chorée hystérique"—which only resembles chorea[1] in some of its symptoms. The children sometimes complain of weakness of one of the arms, but I have never seen complete paralysis. They were almost always able to make any movements I wished, at least to a certain degree. In a very few cases there was more marked paresis, especially of one arm, and this in one case was so considerable that for days the child was obliged to lift the paretic limb with the other hand. In another child the right arm could for weeks only be raised to the horizontal position with difficulty, and the pressure of the right hand was feeble, although the movements on the right side of the body were less marked than those on the left. The cause of this paresis is still unknown, and its occurrence is very rare; and although mothers often describe the child's illness as "paralysis," this is only because they are

[1] For the first of my cases, *v. Klin. Wochenschr.*, 1883, S. 802; also Oppenheim and Thomsen (*Archiv f. Psychiatrie*, Bd. xv., H. 3) have seen chorea come on in a boy after violent mental excitement, with complete hemianæsthesia.

impressed by the inability to move the hands and arms in the ordinary way. On the other hand, I have occasionally found the mental disposition altered; the children were irritable, peevish and passionate, but only very rarely does it reach the point of complete derangement of the intellect, and when it does, this usually takes the form of ecstatic delirium. I remember only one well-marked case of this kind, in a girl of 10 years, who had been suffering for many weeks from a moderately severe attack of chorea, and who had periods of ecstasy during which she assumed the *rôle* of a "princess," demanded all sorts of services from those around her, and harangued them on the subject. This abnormal mental condition disappeared along with the chorea. A girl of 8 years who took chorea in the course of an attack of acute rheumatism, became delirious also, lost her senses, cried and screamed. Still, these symptoms occur—as is well known—in exceptional cases of rheumatism even when there is no chorea. Loss of memory, mental hebetude, and local anæsthesia, of which some authors speak, I have never myself observed.

The course of the disease is almost always very protracted, lasting for many weeks, often for several months. As a rule, its first development is described as being very gradual and scarcely noticeable. Unsteady movements of one hand, or distortions of the facial muscles are the first things that attract attention; and, not unfrequently school children are punished by ignorant teachers for these, and for blotting their copy-books. One poor girl came to my ward with streaks of blood on her hands, caused by blows with a ruler. The severity and extent of the movements gradually increases, until after 4 or 5 weeks they reach their highest point and then they gradually diminish; so that some months pass before recovery is complete. In rare cases the disease lasts for 9 months or longer (either primary or as a relapse), getting now better, now worse, but presenting no complication whatever. As a rule, cases which develop slowly and are of moderate severity have a stronger tendency to become chronic, while I have occasionally seen some which commenced with violent symptoms and presented the most extreme form of the disease, end favourably within 6 weeks. Cases lasting "for years" always rouse suspicion that there is more the matter with them than ordinary chorea minor.

I have met with two cases of this kind; one (December, 1880) was that of a boy of 7, who was said to have become affected in the beginning of his 2nd year; the other boy was 8 years old, and the disease was said to have lasted four years, and to have arisen after an attack of typhoid. The disease differs from ordinary chorea in its commencing at such a very early age, and especially in the circumstance that attempts at co-ordination (which in ordinary chorea aggravate the muscular restlessness), in both of these cases arrested the movements. The same thing happened in the cases of hysterical chorea which I alluded to (p. 201).

Chorea ends fatally only in the very rare cases which have an extremely violent course; and death usually takes place in a state of coma, which sets in along with delirium and most violent convulsive movements. I have only seen this termination in three cases out of all the chorea patients who have come under my care, and in one of these the disease was complicated with incompetence of the mitral valve.

An anæmic girl of 10 years, about whom I was consulted on 6th May, 1876, had suffered for 10 weeks from an attack of chorea which was increasing in severity. It had got worse during the previous 4 weeks especially. Violent continuous movements, necessitating constant confinement to bed. Complete cessation during sleep. For about 3 weeks, mental dulness, great apathy, inability to sit upright; when attempting to do so, the upper part of the body swayed hither and thither. Failure of sight and hearing, paralytic dysphagia, so that she had to be fed with an œsophageal tube. Choreic movements during the last few days still persisting to a moderate extent. Pulse extremely small—50-60 in the minute; nothing abnormal discovered in the heart. Constipation, extreme emaciation. All treatment unsuccessful. Death a week after, in a state of collapse. Post-mortem refused.

The post-mortem was also unfortunately refused in both the other cases. Still I may remark that the changes found in the central organs in the cases of chorea which have ended fatally, have been in no respect characteristic. The microscopic changes recently described in the great ganglion cells, in the spinal cord, and peripheral nerves, stand in need of confirmation.[1]

I have never yet met with cases of incurable chorea—in

[1] There is an interesting case described by Nauwerck ("Ueber Chorea": Jena, 1866), in which microscopic inflammatory areas (in the form of perivascular accumulations of small round cells) were found in the medulla oblongata and in the white matter of the cerebrum, likewise small hæmorrhages and degeneration of some of the nerve fibres in the spinal cord.

childhood, that is—except the above-mentioned fatal case and those just given, which cannot be regarded as examples of ordinary chorea. The notion that cases of chorea sometimes remain uncured, is, according to my experience, largely due to the fact of this disease having been confounded with other conditions; and to this I shall shortly return. In particular, I consider that all cases must be excluded which have existed since birth or from the first years of life, and those in which the chorea lasts for years, though varying in degree (ceasing for not more than a few months at a time, and then recommencing), and where it is combined with hemiparesis, local contractures, or mental weakness. The disease, however, is certainly characterised by an unusual tendency to relapses; and I therefore advise you to warn the parents in every case that sooner or later these may occur, and that they may set in just as violently, and last quite as long, as the earlier attacks; but usually they have a less severe and shorter course. The interval between the first attack and its recurrence has varied, in the cases I have observed, from 3 months to 2 years. I have had many cases in which several relapses occurred.

Girl of 9 years. Chorea in summer, 1844. Recurred in February and November, 1846; in November, 1847 and 1848; in September, 1849; finally in December, 1850, *i.e.* in all, 6 relapses in 6 years. In January, 1848, acute rheumatism, after which—in November, during the 5th relapse—incompetency of the mitral valve was discovered.

Girl of 13, brought to me on 10th February, 1874. Four years previously had chorea for first time. A violent relapse one year after. In beginning of February, 1874, had this 3rd attack.

Girl of 13, brought to me on 13th May, 1874. First attack three years ago, with yearly relapses since; duration 3—5 months.

Girl of 10, brought to me 31st May, 1875. Chorea 2 years ago. First relapse November, 1874—February, 1875. Second relapse a few days previously.

Girl of 10. Ever since the end of her 6th year had suffered from violent chorea, which was caused by a fright. It lasted about 3 months at a time, then ceased for some months and again recurred. During the last 4 years, therefore, the patient has suffered from chorea almost half the time.

I may here remark in passing that the relation between chorea and rheumatism has not always to do with the occurrence of the relapses. Such an influence could only be assumed in the

first of the above cases, while in the three others a rheumatic affection was quite out of the question. The cause of this tendency to relapses—which also belongs to other nervous diseases, especially those of a convulsive type—is unknown, and will remain so, until we can obtain further insight into the real nature and seat of the disease.

So far this insight has been denied us, although there is no lack of hypotheses or of explanations suggested by experiments. At the very first glance you will be struck by the difference between choreic movements and those of other convulsive diseases, *e.g.* eclampsia and tetanus. For while the latter present either rigid contractures, or spasmodic contractions occurring in fits as if caused by an electric shock, in chorea you observe only such movements as are performed under normal conditions—flexion and extension, adduction and abduction, pronation and supination—only, all these movements are uncontrolled and precipitate. The muscular actions, as Romberg pointed out, are therefore always combined or co-ordinated, increasing in severity whenever the patient wishes to put any special group of muscles into action for a definite purpose; and this very inability to prevent a series of accessory movements forms an important feature in chorea. It has not, however, actually been proved that the co-ordinating centre is really the seat of the disease. Indeed, so far, writers have been unable to agree as to whether the chorea originates in the brain or spinal cord. Former experiments show, at any rate, that decapitated animals are able to perform combined movements,[1] and also the experiments of Chauveau, Legros, and Onimus[2] seem to be in favour of the seat of the disease being in the spinal cord, namely, in the nerve-cells of the posterior horns, or in the fibres connecting these with the motor cells. On the other hand, the implication of the facial muscles, the combination of psychical conditions (delirium, ecstacy, &c.), the unilateral form of hemichorea, and the unmistakable influence of psychical causes, are also in favour of its being an affection of the brain. In my teaching I have for years maintained that chorea, like epilepsy, is not a disease of itself, but only a symptom, and that it is therefore well to

[1] Romberg, *Lehrb. d. Nervenkrankh.*, i., S. 509.
[2] *Journal de l'anatomie et de physiologie*, No. 1, 1870.

confine the name "chorea" to the definite neurosis which, with few exceptions, is peculiar to childhood; and in other cases—where similar movements may arise under various circumstances—to speak of such as "chorea-like." Among these undoubtedly are certain affections of the central organs, especially of the brain. In a few cases of cerebral tuberculosis I have observed along with hemiplegia, or with partial paralysis of one arm, almost continuous automatic movements very like those of chorea in the affected limb. To the same category belongs the "post-paralytic" hemichorea in hemiplegic and anæsthetic limbs, the seat of which is placed by Charcot in the corona radiata (?); likewise the condition of "athetosis," which is allied to choreic movements. Occasionally also cases have been observed, in adults (much less commonly in children), of chronic chorea extending over many years; in which after death various changes were found in the brain and spinal cord, *e.g.* sclerosis in the cervical cord in a girl of 14 years who had suffered from congenital chorea (Eisenlohr[1]).

In the vast majority of cases of chorea in children we may exclude any material affection of the central organs, as indeed might be expected from the almost invariably favourable termination of the disease. One must, with Nauwerck (see p. 203, *note*), assume that the inflammatory changes in the central nervous system which he found may be recovered from, without any traces worth mentioning being left behind. Without denying the possibility of this assumption, I still prefer to regard chorea as always a "neurosis," probably arising from an irritated condition of the co-ordination-centres. The causes of this condition of irritation certainly elude our observation in many cases. Very often, in spite of the most careful search, you will not be able to find any cause. A few cases in which the father and the aunt have suffered repeatedly from chorea do not seem to me conclusive. As a rule the children are healthy in other respects, and the composition of the blood seems normal. In many cases, however, we find anæmia, with pallor of the skin and mucous membranes, venous bruits in the neck, and general weakness. Fright or fear (psychical impressions) have frequently been the occasion of chorea developing.

[1] *Centralbl. f. Nervenheilk.*, 1880.

A girl of 12 years had been so terrified by a dog leaping upon her that for two or three days she was almost speechless. Chorea set in immediately after.—In a girl of 11 years it occurred after a fright caused by a strange man coming into the house.—A girl of 12 years took chorea after bathing in the sea for the first time, when she had been much frightened and had struggled much.—A girl of 10 years, who, while sitting in a closet was exceedingly frightened by a boy flinging open the door, showed the first signs of chorea next morning.—Another girl, suffering from spinal caries, was very much terrified by being suspended in the process of applying a Sayre's jacket, and took chorea that evening.—In a girl of 5 years the first movements appeared on the day after the Sedan holiday, during which she had been very much frightened by the loud reports of the cannon. I have frequently seen the disease come on after a blow or a fall, and in these cases attribute much more importance to the fright than to the trifling injury. Mental over-exertion at school I have never been able to ascertain with certainty as a cause. It is much more likely to be due to dread of the teacher or fear of threatened punishment.

Most certainly, however, rheumatism in one of its various forms must be regarded as one of the commonest causes of chorea. The observation on this subject published by French and English writers (Bouteille, Sée, Hughes, Bright and others) did not at first receive from us the attention they deserved, and we have only gradually learned to estimate their value. In 1846, 1851 and 1868,[1] I published a number of cases of this rheumatic chorea myself, and since that time I have had abundant opportunity, both in hospital and private practice, of observing its frequent occurrence. Roger's[2] work especially contains abundant material. It is most frequently during the subsidence of, or convalescence from, acute rheumatism that chorea developes; and I should advise you always to be prepared for the possibility of its onset. Very rarely, indeed, I have observed chorea during the most acute stage of the polyarthritis; and then the most violent pain is caused by the continuous movements of the affected joints. The children cry and scream and get into a state of the utmost excitement. Occasionally we observe an alternation of the two affections, as in a case of Roger's where six attacks of acute rheumatism and five of chorea were counted.

[1] Romberg und Henoch, *Klinische Ergebnisse.*, S. 20.—Also their *Klinische Wahrnehmungen und Beobachtungen*, S. 66.—Henoch, *Beiträge zur Kinderheilk.*, *N. F.*, S. 105.

[2] *Arch. gén. de Méd.*, 1866, Dec., et seq.

But also apparently slight rheumatic conditions may be followed by chorea, wandering pains with slight swelling of single joints lasting only a few days and scarcely accompanied by any fever, or even by simple pains in the back, calves, or various joints without either swelling or fever. I have frequently noticed that the reappearance of such rheumatic conditions during the course of chorea has aggravated the movements although they were in course of subsiding. Cases even occur in which a quite limited rheumatic affection, *e.g.* torticollis, is followed by chorea. In one boy of 14, with right hemichorea, the preceding pains and swelling had been entirely confined to the joints of the hands and feet on the right side; but this is by no means always the case. Less commonly, chorea appears first, and rheumatism only sets in later; as in one of the cases mentioned to illustrate relapses (p. 204), where an attack of acute rheumatism with endocarditis came on for the first time after the fourth attack of chorea. I have observed the same thing in the following cases:—

A girl of 12, who took chorea when in perfect health, had repeated painful swellings of the joints of the feet and hands and vague pains in the limbs throughout its course, which lasted 3 months.

A girl of 12, admitted into the ward with chorea, 18th December, 1872, was discharged cured in the beginning of February. There was no trace of rheumatism whatever, and the heart was found to be perfectly normal. In November, 1875, she had an attack of acute rheumatism, followed in December by a violent relapse of the chorea. When examined in the polyclinic, incompetence of the mitral valve was discovered.

In a boy of 10 years, admitted with chorea in October, 1885, who had never formerly suffered from rheumatism, there occurred, at the beginning of his 3rd week in the hospital, painful swellings of the joints of both feet, accompanied by moderate fever (103·1° F.), to which was soon added a systolic mitral murmur.

You will see from these cases that the chorea may be the first expression of the rheumatic affection, and that it readily recurs if such an affection developes in a child who has previously had chorea. That in these circumstances we may often find valvular affection of the heart, often of the mitral, less frequently of the aortic valves, is easily explained by the well-known connection between rheumatism and the endocardium. I therefore consider it

my duty in every case of chorea to examine the heart carefully; and I have often enough found valvular disease and its results, which had not been revealed by any subjective symptom—in no case by palpitation. I would especially draw attention to the fact that we must guard against mistaking anæmic for organic cardiac murmurs, since in such cases we very often have to do with anæmic children. When, however, it is maintained that in such cases chorea is always the result of the heart-affection—arising either reflexly or from embolic processes occurring in the great cerebral ganglia—I hold this to be quite erroneous; because I have frequently known cases of chorea rheumatica in which the heart was absolutely unaffected. A short time ago I saw a girl of 14 who within five years had had two attacks of acute rheumatism followed by chorea, but whose heart showed not the slightest abnormality. Experience shows, moreover, that where there is valvular disease, and the relatives declare that there has been no rheumatic affection, it has often really been overlooked owing to its trifling nature. It is also conceivable that chorea may run its course as the expression of rheumatism, with endocarditis, and without the occurrence of pains or joint-swellings (Botrel, Sée, Roger). In this way we may explain the undeniable cases in which a murmur has been found in chorea without a history of past rheumatism—apart from the fact that valvular disease may be quite accidental, or even congenital. The embolic processes in the corpus striatum and its neighbourhood would certainly have quite other results, and would hardly end so quickly in complete recovery as is seen in almost all such cases of chorea—as every physician who has many children under his treatment will acknowledge. Therefore, in my opinion, the heart-affection has nothing to do with chorea, but both are due to the same cause—rheumatism—which appears to act in an unexplained way on the co-ordinating centres.

Chorea also occurs much more rarely as a sequela of acute infectious diseases. Thus I have seen it in two girls a few weeks after measles, in an anæmic boy of 8 about a fortnight after recovery from diphtheria, and in four other children in connection with scarlet fever. As a general rule, however, I hold (in opposition to Bouchut) that the occurrence of chorea as a sequela of scarlet fever is rare, and I have never been able

to confirm his statement that under these circumstances chorea occasionally only lasts 6—8 hours. Two of my cases accompanied scarlet fever, and were therefore not sequelæ. In a girl of 7 who had already had chorea once, it recurred during the eruptive stage of scarlet fever; while in the other case there were important complications.

> On 2nd February, 1876, I was consulted about a boy of 3 years, who had suffered during the first weeks of scarlet fever from painful swellings of the joints of the feet, knees, and many of the fingers. A few days afterwards severe chorea developed, which had lasted for 1½ weeks when I saw the child. On examination we found a loud systolic murmur at the apex, very violent action of the heart, and high temperature, rising towards evening. At end of 3rd week, hæmorrhagic nephritis, ending fatally from œdema of the lungs. The boy had never before suffered from chorea, and his heart had been quite normal.

This case might support the view of those who regard endocarditis as itself a cause of chorea, were it not that chorea, as we have just seen, occurs also during and after scarlet fever without any heart-disease. The synovitis which has also been observed in other similar cases[1] is certainly significant whether we regard it as a condition of reflex irritation, or as one of a series of pyæmic symptoms which may result in "chorea-like" movements (p. 205).

I have never yet met with any case of chorea in a child which was caused by reflex irritation, and which could therefore be placed alongside of chorea gravidarum. Irritation from worms, or in connection with the genital organs, is much more frequently assumed as a cause than the facts warrant; at least, I have never yet succeeded in curing chorea by anthelminthics, even although worms were expelled by their use; nor by an operation for phimosis.

How do intercurrent diseases affect chorea? This question has been answered in various ways, and the following cases prove that nothing definite can really be said on the subject.

> Boy of 9, admitted 27th January, 1873, with chorea of unknown duration. Heart normal, but pulse irregular and intermittent. On 6th February an attack of indigestion, with colic, high fever

[1] Litten, "Beiträge zur Aetiologie der Chorea:" Charité-Annalen, *Jahrg.*, xi., S. 14.

(105·8° F.). An emetic. Next day, T. 100° F., but chorea very severe. During the next few days acute pleurisy developed on the left side. On 10th February considerable decrease in the choreic movements. Pulse always slow and irregular. On 2nd March chorea almost quite gone. A relapse dating from 17th May. Pulse always 68, somewhat irregular. Pleuritic exudation almost entirely re-absorbed. Recovery after a fortnight.

Boy of 13 (10th February, 1873). Right-sided chorea for last 8 days without known cause. No rheumatism, heart normal; altogether quite healthy. In spite of the most approved remedies, the disease lasted till the end of May, when it began to subside. On 1st June, dislocation of the shoulder-joint from a fall on the elbow. Immediately after its reduction—which was very painful—the chorea was found to have completely and permanently disappeared. In the ensuing autumn, acute rheumatism, with endocarditis, but without any recurrence of the chorea.

Boy of 7 years treated for chorea in the hospital. Systolic murmur in the mitral area. An acute tonsillitis with temp. 104° F. had no influence whatever on the chorea.

Girl of 10 treated for chorea in the hospital (also tubercular). Neither an inter-current diptheritic sore throat (temp. 102·9°—104° F.) nor an attack of measles ensuing had any influence whatever on the chorea.—I have observed the same in a girl who took scarlet fever and broncho-pneumonia during an attack of chorea (T. 104°); indeed, in one girl of 11 the movements increased markedly in severity when the scarlet-fever rash appeared.

You see, then, that febrile affections certainly do not always cut short the disease as some maintain. The rapid disappearance after the dislocation is very remarkable, but you must remember that the chorea had already lasted four months and was in process of subsiding, so that in all probability it would have disappeared about this time spontaneously. This natural cure of the disease after an average duration of three months is another source of difficulty in judging of the treatment employed, and renders desirable the strictest criticism of the numerous remedies that have been recommended. At a certain stage all these appear to be of use, because the disease is in process of spontaneous cure. You will therefore consider it excusable if I do not discuss further remedies to which I attach absolutely no value; and unfortunately I must answer entirely in the negative the question whether there is a remedy which is certain to cut short the course of chorea. I should certainly give the first place to arsenic, which—on Romberg's recommendation—I have used ever since I began practice. But even this remedy has no con-

stant action. In spite of its prolonged use, I have frequently seen the disease persist for months, although in the majority of cases a moderating influence on the violence of the movements was soon noticeable; and many cases treated with this drug ended favourably in a comparatively short time (5—6 weeks). According to my experience, arsenic suits all cases of chorea—rheumatic as well as any other—unless it is contraindicated by derangement of the stomach or bowels. It has appeared to me to be especially useful in anæmic patients. I always order 2 or 3 drops of Fowler's solution (Form. 11) thrice daily—about an hour after breakfast, dinner and supper—and when it was given thus, I have very seldom been called upon to discontinue the medicine owing to nausea and diarrhœa. Most children can bear arsenic very well for many weeks; and I therefore look upon all the apprehensions and warnings which have been expressed, as old wives' stories invented by the ignorant. Neither have I ever seen zoster from the use of this drug.[1] I have no experience of the hypodermic administration of arsenic which some recommend.[2] I should use it however in cases where the stomach was unable to bear the drug. The addition of opium (about ♏xx of the tincture to the mixture recommended above) has appeared to me to increase the action of the arsenic in some very violent cases. In cases where Fowler's solution has no effect I have repeatedly seen an unexpectedly rapid action from the use of arsenious acid (gr. $\frac{1}{130}$ to gr. $\frac{1}{60}$ daily) in the form of pills (Form. 11a).

Should the violent movements continue through the night owing to sleeplessness, I recommend you to give chloral hydrate (grs. viiss.—grs. xxiii). To some children suffering from a very violent form of the disease, we even gave this dose two or three times a day, and this soon induced sleep and consequent diminution of the movements. In a girl of 10 years, however, an eruption very like that of scarlet fever took place over a large part of the body, as the result of the chloral.

[1] Bokaï, *Jahrb. f. Kinderheilk.*, Bd. xxi., S. 411.—Guaita, *ibid.* xxiii., S. 216.

[2] Garin, *Archiv f. Kinderheilk.*, i., S. 335. Injection of 4—5 drops of Fowler's solution every 3 or 4 days or even daily. Recent cases are said to have been cured after about 18 injections.—Frühwald, *Jahrb. f. Kinderheilk.*, xxiv., S. 42, recommends sol. Fowl., aq. dest., equal parts—1 division of syringe to be injected, to be increased daily by one division up to 8 or 10 divisions; then to be diminished in the same way.

Chloral is only suitable under these circumstances as a first step in the treatment, and must give place to arsenic after a certain amount of quietness and sleep have been obtained.

The purgative treatment, so popular formerly, as well as the use of tartar emetic have all fallen more or less into disuse, which is certainly justified, considering the delicate anæmic constitution of many choreic children. Where, however, this contra-indication is not present, and the patients lack neither blood nor fat, the use of purgatives has seemed to me advantageous. In such cases I begin with two or three days purging, and also later on I stop the arsenic every week for 24 hours, and give instead 1 or 1½ spoonfuls of ol. ricini or infus. sennæ. From the many other remedies recommended you need not, as far as my experience shows, expect any good results; nor yet from large doses of bromide of potash or strychnia, which (following Trousseau's example) I have repeatedly tried both internally and hypodermically (gr. $\frac{1}{33}$—$\frac{1}{22}$).

Although the action of the latter in one case surprised me, I was soon convinced that its success was only apparent and accidental. I can say just as little in favour of ether spray to the spinal column, sulphur baths (℥iss—℥iiiss to the bath) or the constant current, of which others speak in very high terms.[1]

Mental exertion is to be avoided if possible while the chorea lasts; school attendance must therefore be discontinued, the more so because their schoolfellows are very apt to mock choreic children. I have never known of the disease being transmitted to other children, but some have asserted that this may occur—especially in schools and institutions—owing to mimicry. In severe cases you must keep the patient in his bed, and have it padded with cushions to avoid bruises. The more speedy recovery in hospital as compared with private practice is perhaps due to the children being kept constantly in bed for a longer time.

A few new remedies I have had no occasion to try—sod. salicyl., eserine, hyoscyamine and antipyrin. A trial of eserine (physostigmine) which we made in a girl of 12, caused the most unpleasant symptoms of poisoning (collapse, vomiting, profuse sweating, imperceptible pulse) within 15 minutes, although the dose was small (gr. $\frac{1}{30}$ hypodermically) and the child only slowly recovered from this condition under the use of stimulants. A second trial, in a younger boy, had a similar although not quite so severe result.—Antipyrin (up to grs. xlvi. in the day) has very recently been strongly praised by Legroux and Dupré (*Revue mens.*, Mars, 1888).

We may further recommend nourishing diet, pure air, cold frictions in the morning (as long as the children do not resent it; if they do, warm baths are better), suitable gymnastics and, finally, a preparation of iron (Form. 12).

This seems the most suitable place to mention a condition which is often confounded with chorea by medical men and described as such, but which—apart from its much greater rarity—is essentially separated from it by its symptoms. I have already elsewhere described it as "chorea electrica,"[1] and Hennig[2] also has described similar cases under this name.[3] In this form you never find those hurried co-ordinated movements, aggravated by purposive muscular action, which form the very essence of chorea. The patients remain perfectly quiet; but from time to time "lightning" spasms occur—especially in the muscles of neck and shoulders, but also in other parts—resembling very closely those caused by a weak faradic current. As a rule they are only slight, and are gone so rapidly that sometimes very careful observation is necessary to see them at all. In addition to the four cases given in the above-mentioned work, I have since met with several others quite similar. Altogether I have had more than a dozen such cases, both in boys and girls, all between 9 and 15. When the patients are stripped, one can readily see and feel the rapid spasmodic contractions of the individual muscles; and when the tongue is put out, it shows in some cases vermicular movements similar to those in ordinary chorea. Each individual contraction lasts but a moment, but the intervals vary greatly. Occasionally the next contraction follows in a few seconds; in other cases after several minutes, especially if the patient's attention is otherwise taken up. The speech is unaffected, and the ability to write, sew, &c., is unimpaired, except when these actions are momentarily interrupted by the spasmodic movements of the arm. One half of the body is occasionally more severely affected than the other. In one girl of 15 years, the contractions were limited to the right side of the body and face, and they were so often repeated that they considerably interfered with her writing or doing work with her

[1] *Beiträge zur Kinderheilk., N.F.*, S. 113.

[2] *Lehrb. d. Krankh. des Kindes.*, 3 Aufl., 1864, S. 343.

[3] Trousseau (*Clinique* &c., ii.) appears to have indicated analogous cases in his description of "Tic non douloureux."

right hand. Moreover, the power of movement remained perfectly normal, and there were no other morbid appearances whatever. Only in one of the cases did the movements persist during sleep, but they were weaker and less frequent. In all the other cases they completely ceased as in chorea. In a boy of 11 there were spasmodic contractions of the head, by which the face was turned upward and to the left. These were sometimes accompanied by winking of both eyes and twitching of the left ear. It turned out that this boy had suffered for some weeks, a year previously, from spasmodic winking of both eyes. In one case the occipito-frontalis muscle was also affected by "lightning" contractions. In a boy of 10 years, whose whole body was convulsed by these spasmodic contractions while his head was almost quite unaffected, there occurred along with each shock a spasmodic inspiration accompanied by a gurgling noise, indicating implication of the diaphragm, perhaps also of the glottis. Generally the speech was unimpaired, or at most only interrupted at the moment of the shock.

According to the history in one case, it appeared that the child had had epileptic convulsions until two years before, and the spasmodic twitchings had set in when these disappeared. In two other cases, tearing pains in the limbs and acute articular rheumatism had preceded; and in one girl of 14, violent screaming fits. A girl of 11 became affected after a fall on the head when she was walking on stilts. In two cases the cause was said to have been fright, from the violent slamming of a door, and from seeing a person hanged. Frequently, however, I was unable to find any noteworthy points in the history, and therefore was at first tempted to regard it as altogether the result of a habit. I believe that these contractions also—like chorea and epilepsy—are merely different manifestations of the various kinds of direct or reflex irritation in the nerve centres. Thus it may happen that in rare cases very surprising combinations of convulsive symptoms appear, generally accompanied by blepharospasm (continuous winking movements).

On 6th March, 1879, a boy of 10 was brought to my polyclinic, who had suffered from the following symptoms since he was 3 years old—that is, for 7 years. The left side of the body, especially the arm, showed almost incessant chorea-like movements; but the arm also from time to time took fits of spasmodic con-

traction just as occurs in an epileptic attack. The left side of the face had also previously been affected; but this was no longer the case; and the lower extremity had also become quieter. Complete cessation during sleep. He could take hold of nothing with the fingers of the left hand, but he could retain anything with them. Intelligence and general health perfectly normal. Electricity was said to have formerly had a good effect. Did not return for treatment.

Thus we find here a combination of real choreic movements with clonic convulsions, the pathogenesis of which is utterly obscure. Unfortunately this was the rule in the cases of chorea electrica that I have observed. The treatment could therefore only be empirical and not very hopeful. Only in one case have I seen a markedly good effect from bromide of potash; it also succeeded in a relapse which took place as the result of a feverish gastric attack. In none of the other cases did I get any good result from this or from any other remedy. Arsenic, atropine, injections of strychnia, and extract of physostigma, all remained unsuccessful. I should most strongly recommend the continuous use of the galvanic current, as in a few cases this certainly had a favourable effect. In two cases it even brought about complete recovery; but for the permanence of the cure I cannot answer.[1]

VI.—*The Hysterical Affections of Children.*

You will have learned from the study of disease in adults that the remarkable nervous symptoms which we are wont to group together under the name "hysteria," do not always deserve this designation, which implies a connection with the female genital system. You know that in many hysterical women even the most careful examination of the genital organs reveals no abnormality, and that quite similar symptoms are observed, though much less commonly, in the male sex. You will now see that even childhood is by no means exempt from them.[2] I

[1] Cadet de Gassicourt (*l. c.*, p. 256) praises the action of electricity (induced current), while Bergeron says that he has seen good results from the use of antim. tart. (gr. ½ given at once), *i.e.*, an emetic. Berland, *Thèse:* Paris, 1880. —Tordeus, *Journal méd. de Bruxelles*, 1880.—Remak (*Berl. klin. Wochenschr.*, 1881, No. 21—23) cured a case that I observed by galvanic treatment continued for 9 months.

[2] *Cf.* Smidt, "Ueber das Vorkommen von Hysterie bei Kindern," *Jahrb. f.*

do not know what more suitable name could be applied to the somewhat remarkable phenomena we have here to deal with, and my only excuse is that we also know next to nothing about the real hysteria in adults, that all theories advanced on the subject break down, and that one must be content to include under the one name a combination of the most varied neurotic symptoms —motor, sensory, psychical, and even trophic—which may form continually changing combinations, and alternate with one another. The starting point of these symptoms, and the nature of their essential connection with one another, remains entirely unknown to us, and the favourite supposition—of increased reflex excitability of a "nervous" disposition—is not well calculated to fill up the gaps in our knowledge.

We find quite the same in children also, boys as well as girls—although the latter are somewhat more frequently affected. I am convinced that the following description will be attacked on many sides, because it includes in one group a number of morbid conditions which are usually treated as differing from one another—such as chorea magna, catalepsy, voice-spasm and many others. I grant that I may be wrong in this, but I consider that my view is favoured by the circumstance that in practice we not uncommonly find transitions from one form to another and combinations of them, so that one may readily be at a loss what name to apply to any given case. The experienced practitioner will understand this and appreciate it, and set more value upon this fact than on any objections advanced by mere theorists. Considering the great diversity of the symptoms and their numerous variations, even in one and the same patient, it seems to me quite impossible to sketch a general comprehensive picture of the "hysterical" conditions of children. I can, therefore, only endeavour to give you in outline certain classes of cases from my own practice, which illustrate as far as possible the different forms.

The first class comprises those cases in which the psychical symptoms predominate—complete cessation of consciousness, hallucinations and delirium, pavor nocturnus and diurnus. To this category belong almost all the conditions described

---

Kinderheilk., xv., 1880, 1.—Peugniez "De l'hystérie chez les enfants," *Thèse:* Paris, 1885—a work from the school of Charcot, very rich in clinical material. —Riesenfeld, "Ueber Hysterie bei Kindern," *Diss.:* Kiel, 1887.

under the name catalepsy or eclipsis. Consciousness is suddenly lost, or at least considerably weakened, and the children remain sitting or standing with a staring look, or with up-turned eyeballs, and gradually sink on the floor if not caught hold of. More rarely they are still able to go about in a semi-conscious condition as in a dream, sometimes talking to themselves unintelligibly. A little patient of this kind fell on the street and went right through the window of a cellar. In other cases the eyes are closed, the expression of the face unaltered and the colour pale; the normal condition of the pulse, however, and of the heart's impulse, and the unaltered temperature, distinguish the condition from syncope. After a few seconds—or at most, a few minutes—it is over, and the child feels perfectly well. Many are quite unaware that they have had such an attack, others remember the beginning of it, and others again only partially lose consciousness, so that although unable to speak, they see and hear everything that takes place around them as if half asleep. After the attack they generally go on with the employment in which they have been interrupted, as if nothing had happened. Only exceptionally have I found during the attack that exaggeration of the muscular tone, which in the catalepsy of adults is known as "flexibilitas cerea" of the limbs—in which they remain in whatever position they are placed. The attacks almost always occur very irregularly, sometimes five, six, or even more times a day, at other times only every few days or weeks, without any definite cause being discoverable. The troublesome thing for the physician is that he can never be absolutely sure that these will not degenerate into epileptic attacks; but this does not take place as a rule. Although in the hospital and polyclinic we cannot always observe the final results of these cases—and indeed I have many times failed to do so—still, in private practice I have often had the opportunity of ascertaining that they did recover in the end, after many months of alternate improvement and relapse. I am, therefore, always in the habit of giving a good prognosis, unless there happens to be hereditary predisposition to epilepsy, or real epileptic fits have already occurred.

This was the case, for instance, in a girl of 10, who had had several epileptic fits six years previously. Three months before, another of these had occurred; and since then attacks had

occurred every 2 or 3 weeks, which were announced by a sensation of itching in the hands and feet, and only consisted of a mental change, wandering about in a state of unconsciousness, delirium, and hallucinations. Although in this case no convulsions at all were observed one cannot doubt the epileptic nature of the condition, which might at any moment have given place to a regular fit.[1]

Even the occurrence of convulsive symptoms should not at once make us anxious. In a few cases in which repeated attacks of the kind described occurred in the day time—loss of consciousness with unintelligible speech and vacant staring—delirium was often observed during the night, with slight twitchings of different parts of the body, during which many of the children sat up in bed without knowing what they were doing. A few of the following cases show that such a complication may occur during the daytime.

Girl of 12 years, admitted 1st November, 1881. Had always been healthy, with the exception of an attack of pneumonia in her 6th year. Since August attacks of palpitation and pains in the region of the heart. Almost immediately after a violent fright caused by a boy who was going to hit her, these symptoms were replaced by maniacal attacks—she screamed passionately, clenched her fists and stamped her feet and stared wildly round. Any fright, even the voice of the boy or that of his relatives, induced these attacks. Intervals of perfect sanity. About a fortnight before admission into the ward these attacks had suddenly disappeared, and the third phase of the disease now began, characterised by attacks of extreme apathy and a kind of dream-life. She wandered about unconsciously, seeing and hearing nothing, staring into space, sinking helplessly on to the floor, and from time to time took violent fits of laughing and weeping, winking of the eyelids, and quivering of the right arm. Several of these fits in the day, with free intervals. Sleep and general health undisturbed. Treatment: rest in bed for some weeks, and daily warm baths lasting half an hour. Improvement and, in the end, recovery. No return of the disease by March, 1882. Menstruation had not yet set in.

A boy of 9 years, of perfectly healthy family, was suddenly affected by giddiness in August, 1865, while taking salt-baths. In the end of January, 1866, the first of the attacks, about to be described, took place and they recurred in April and August. Suddenly, without cause and without premonitory symptoms he complained of giddiness, which was occasionally so violent that he fell down. His look became staring, his head very hot and

[1] *Cf.* a similar doubtful case which I have published in the *Charité-Annalen*, Bd. ix., S. 616.

delirium ensued, which seemed to be always occasioned by the same hallucinations. The boy saw on all sides great "cupboards" and armed men pressing upon him; and at the same time his hands could be seen to twitch slightly. An attack of this kind lasted 2 or 3 days, not indeed continuously, but interrupted by intervals of quiet—in which, however, consciousness was never perfectly clear. The attack ended suddenly, and the boy at once said that it was all over. With the exception of headache, he was perfectly well in the free intervals; all his organic functions were normal. I ordered him to take bromide of potash for a few months. In the night of the 23rd December, *i.e.* after an interval of 4 months, he again had a fit, ushering in an eruption of measles on the 26th. Since that time no fit has been observed. The headaches, also, have long since disappeared, and the boy has grown up perfectly healthy, and is now an officer.

Boy of 13, convalescent from perityphlitis. Admitted in October, 1883. Three weeks ago repeated general convulsions with semi-unconsciousness. Since then only slight partial convulsions, rolling of the eyes, headaches, sudden fits of somnambulism with hallucinations, clapping his hands. Otherwise quite well. Gradual improvement. After 2 months was discharged cured.

Girl of 8 (admitted 27th November, 1879). Attacks of globus hystericus during the past year, which commenced with an aura rising from the umbilicus to the neck. Rolling of the eyes, semi-unconsciousness, falling to the ground, and hallucinations of various kinds occurring several times in the day. At the same time great restlessness, rapid articulation, change of colour and an erotic tendency, which was shown by a certain coquetry and by her frequently asking the house physician to kiss her and to press hard on her abdomen.

Girl of 12 years, had twice had chorea. During the last three months (sometimes every day, and sometimes with 6—8 days' interval, especially after any mental disturbance) she had had attacks of frontal headache, followed soon after by religious fancies and hallucinations. She then spoke of God, saw an angel come floating down, called her mother Eve, sang hymns interspersed with secular songs, did not recognise her relations, and stared vacantly before her. She had no recollection of these fits, which lasted 15—20 minutes. Free intervals. After a few weeks these fits disappeared and were replaced by convulsive contractions of the face and upper extremities, without loss of consciousness, but with loss of speech. These were ushered in and accompanied by vertical headache. The threat of sending the child from her parents to her grandmother in the country had a speedy effect. The fits diminished quickly, and after some days entirely ceased.

Girl of 9 years, admitted 8th July, 1881. Had always been

very subject to frights. Nine weeks before the school-teacher had punished her by striking her on the hands till they became swollen and painful. Soon after, "nervous fever" (?). Ever since, her wits had been confused, she answered slowly and indistinctly, occasionally also had cataleptic attacks with rigidity and aphasia. Great weakness and pallor. Organs and functions normal. Speech slow. Before speaking she first opened her mouth wide and then articulated laboriously and indistinctly. Memory good. Movements weak; can neither stand nor walk. Skin hyperæsthetic in many places. Enuresis nocturna, sometimes also diurna. Application of the faradic current to the spinal column. On the 11th she could walk with a little support. On 12th, occasional delirium. She says she sees snakes creeping in at the windows. Cold affusions. On 17th, everything normal; on 30th, discharged cured.

The second class includes those cases in which the convulsive symptoms predominate. These are sometimes confined to a certain nervous area; *e.g.* in an anæmic girl of 8 years they occurred in the form of violent fits of hiccough, which lasted from one to two weeks, and only ceased during sleep. More frequently they affected the vocal organs or included in their action all the muscles of the body to a greater or less degree. The essential character of these general attacks (which are usually called "hystero-epilepsy" to distinguish them from real epilepsy) seems to me to consist chiefly in the fact that consciousness, and the functions of the senses are retained, or at least not completely lost, and that the attack is often complicated by fits of crying and screaming.

Anna H., 9 years old, brought 31st December, 1878. Of healthy family. Had only suffered from three convulsive fits in her 4th year. During the last three weeks she had at irregular intervals suddenly uttered a cry—about every 5—15 minutes—which could only be compared to the roar of a wild beast. Complete cessation during sleep. The attempt to cough only ended in a bellowing noise. Health otherwise perfectly good. Arsenic, chloral, and pot. brom. were of no use. Recovery, from the application of the galvanic current after a few sittings.—Also in a boy of 8 years, who for some weeks had had such violent attacks of convulsive screaming, that on the street one could hear the screaming from his house on the second flat. The use of electricity produced an unexpectedly rapid recovery.

An anæmic girl of 12 complained of constant dryness in her throat, so that she had to keep drinking. Urine normal. If her thirst was not at once satisfied, fits of crying and screaming

soon ensued and lasted some minutes.—In a healthy girl of 12, I observed attacks of violent dyspnœa with loud screaming, which after some minutes gave place to striking-out with the hands and feet. In other cases (girls of 10, 12, and 13 years) violent spasmodic attacks of coughing took place with whistling inspiration, lasting for hours and audible through several rooms, without any accompanying symptoms except a feeling of oppression over the stomach and larynx.

While in these and similar cases there was nothing but voice-spasm, in other children this was only the precursor or accompaniment of the spasmodic contractions which I have described as "chorea electrica" (p. 214). In other cases the voice-spasm was accompanied by convulsions or paralytic symptoms, and we learn from the following observations that the same sudden changes of nervous symptoms from one extreme to another, which we see so often in hysterical women, may occur in childhood also.

Girl of 9 years. For about 5 months, several attacks daily, both in the daytime and at night. They began with loud groaning or grunting, then rotation of her head took place to right or left, so that she looked over her shoulder; and she anxiously complained that some one was standing behind her. Consciousness confused. When she was firmly taken hold of she at once came to herself. General health perfectly good. Further course unknown.

Boy of 10 years, brought to my consulting room, 15th March, 1879. Since his 3rd year, and without any recognisable cause he had had short but violent convulsive spasms of the whole upper part of the body, in which his head was thrown forward and shaken from side to side. These attacks, which sometimes occurred every few minutes, and often at longer intervals, were also invariably combined with a cooing and clucking sound (voice-spasm). Any embarrassment increased the frequency and severity of these attacks, while movement in the open air and play almost entirely removed them. Complete cessation during sleep. During the last 7 years these convulsions had never completely ceased, but had sometimes been milder and less frequent. Otherwise quite normal; no neurotic tendency in the family. The continued application of the galvanic current during a year by M. Meyer on my recommendation, caused in the end an unlooked for amount of improvement. Only the slightest trace of the attacks was left, and the voice-spasm especially had completely disappeared. There was still, however, a great tendency to recurrence.[1]

M. Meyer, *Die Elekricität in ihrer Anwendungen auf prakt. Medicin.*, 4 Aufl., 1883, S. 386.

Marie S., 11 years old, had suffered from dyspepsia since New Year, 1878. In the end of February, 1879, attacks of eructation came on, which recurred very frequently for three weeks and sometimes lasted the whole day. They suddenly disappeared in the middle of March, and were replaced by the condition which led to my being consulted. The child—delicate, pale and wasted—lay in the corner of a sofa with a peevish, pained expression of face, and at every expiration uttered a half whimpering, half squeaking sound without, however, ever shedding a single tear. This voice-spasm—for such I considered it to be—on rare occasions disappeared after a few eructations; and during this short interval the features at once assumed a quieter and more cheerful expression—from which we might infer a combination of the voice-spasm with a similar condition of the facial muscles. The application of the galvanic current had no more effect than chloral or the Ems-water which was prescribed for the dyspepsia. On 26th March the latter disappeared quite suddenly, the tongue became clean, the appetite excellent; the other conditions remained as they were. Inhalations o chloroform rapidly produced complete cessation of the voice-spasm—even when the narcosis was slight, but after 8—10 minutes it occurred again in the old way. Only during sleep did the spasm completely disappear, and the harassed parents could then recover themselves from the depressing impressions of the whole day. In the beginning of April the sound suddenly changed its character. It became a hollow groan; and at the same time the face lost its peevish expression, the features became natural, and the child was at last able to smile again. The speech, however, was interfered with, and it was only with difficulty that we could draw a few words from the child. The application of a cold sponge to the neck (which we ordered several times a day for 15—20 minutes), had no effect whatever; and the voice-spasm, which was somewhat altered, persisted almost unchanged (except during the night). To this was now added a paralytic weakness of all the muscles, which made it impossible to hold up the head unsupported or to walk one step alone. On every attempt to do so, the legs tottered as if ataxic. It was striking to see the perseverance with which the child lay the whole day on the sofa, and cut out paper dolls. Injections of strychnia into the neck (gr. $\frac{1}{2}\frac{1}{2}$ daily) and chalybeate water had no real effect. She was soon able, indeed, to walk somewhat better; but otherwise her condition was unchanged. Speech was almost entirely gone, and every attempt to speak caused convulsive movements of the face, as in a case of violent stammering. An intercurrent febrile catarrh was also without effect; but the cough soon assumed a metallic and spasmodic character. On 18th April, the child became suddenly able to speak, although only in whispers, to walk a little without support, and to hold her head up. The voice-spasm became

weaker daily and by the 1st May it had quite disappeared. The speech now loud and distinct, walking much better, and the appearance greatly improved. But the spasmodic cough continued—every few minutes a panting inspiration occurring which was followed by a single croaking or rather whistling cough; during sleep this also disappeared. Under the continued use of strychnia injections (gr. $\frac{1}{12}$ daily), the cough was also improved by the 29th, and recovery might be said to be complete with the exception of occasional eructations. A slight relapse which took place some months later, had the same favourable termination.

Boy M., 9 years old. During the winter and spring of 1883 suffered much from migraine, sometimes several days at a time. Pale, but otherwise healthy. In May, 1883, he slept in one morning, and in spite of all exertions could not be wakened. He was at once roused by making him inhale ammonia. After a few days the attacks recurred frequently, always during sleep. His whole body became drawn together as in emprosthotonus; he kept grunting like a pig, and was continually shoving himself upwards in the bed so that he had to be brought down. Inhalations of ammonia promptly arrested the attack, but it recurred immediately and lasted 1—2 hours. Also spontaneous sneezing or coughing would at once stop the attack. Warm baths with a cold douche and cold-water treatment in Elgersburg produced a permanent cure, after the fits had recurred during several weeks with ever diminishing frequency and intensity.

Not only the laryngeal muscles, but also other muscles of respiration may be the seat of spasmodic contractions, which come on in the form of asthmatic attacks, with rapid and shallow, or else deep breathing, in which the accessory muscles of respiration participate. Such attacks also occur during the night, and are often combined with palpitation and hyperæsthesia of the præcordial region, so that they may seem to be due to cardiac disease. In these cases also we may have mental disturbances (delirium and hallucinations) and spasmodic contractions of the muscles of the face, eyes, and extremities, with intervals which are quite free from all morbid symptoms, during which nothing can be made out—on physical examination at least.

Girl of 10 years, with a "nervous" father. First attack on 31st December, 1883, and by 30th January, 1884, there had been 4 attacks, always on Sundays. They consist in a constriction of the pharynx with dyspnœa, quick shallow breathing and inability to speak. She could only give utterance to inarticulate sounds. Perception and consciousness were normal, but she had occipital or temporal headache, which often occurred during the intervals

also. Duration one or two hours. Occasional nausea and vomiting after the fit. All organs normal on examination. Gradual improvement. Complete recovery after some months.

Girl of 12 years, normally developed but not yet menstruated, perfectly healthy. Had complained of headaches for some weeks. On the morning of 8th February, 1882, after a good night repeated attacks of convulsive contractions of the upper extremities without loss of consciousness. In the afternoon she also began to scream violently and furiously, to spring up in bed, to be delirious (talking of going up a wooded hill) and did not recognise her relatives. After 8 o'clock, sudden cessation of symptoms and a quiet night. Next morning a few lesser attacks of the same kind. Then an undisturbed interval of 10 days, after which there suddenly took place (while she was taking a walk and without any apparent cause) inspiratory spasm—laboured rapid breathing, with harsh almost croupy inspiration, and distorted features. This ceased during the night. During the following days there were frequent repetitions, and then it passed off, giving place to paresis of the lower extremities (inability to walk). On 4th March, she suddenly became able to walk, and has remained perfectly well since.

The cases of the third class are the most surprising, and are accordingly apt to be regarded as simulation. In them the attacks take the form of co-ordinated movements (jumping, running, climbing, &c.), occurring spasmodically, either at quite uncertain intervals or after a definite type. As a rule we also observe during these attacks certain psychical changes—great excitement, screaming, hallucinations, and delirium—while during the intervals there is usually only an altered disposition, great irritability, unusual cheerfulness, or more frequently a tendency to cry. There may, however, be no symptoms in the intervals, and the child is then perfectly well, except during the seizures. These cases are usually spoken of as chorea magna (greater St. Vitus' Dance), and in fact they deserve this name much more than the ordinary chorea. The name "St. Vitus' Dance," was first applied to an epidemic which occurred in Schwabia in the end of the 14th century, which was characterised by a rage for dancing, combined with ecstatic symptoms. The remedy recommended was a pilgrimage to St. Vitus' Chapel in the neighbourhood of Ulm. Sydenham afterwards transferred the name "St. Vitus' Dance" to ordinary chorea, for which Bouteille introduced the latter name in 1810.

The most extreme case of chorea magna which I have met

with, I described many years ago.[1] The most unusual thing about this case was its long duration; from the occurrence of the first attack to complete recovery it lasted 5 years. I have never since then observed such an obstinate case of the disease, or one with such varied symptoms. The most diverse manifestation of altered nerve functions were here combined in one clinical picture—mental irritability, hallucinations, and delirium, fits of jumping and running, opisthotonus, choreic movements, partial hyperæsthesia of the scalp, and a kind of "clairvoyance" which enabled the patient to determine exactly beforehand the number and order of the involuntary movements (a fact which I have frequently observed in such cases).

As regards the duration of the disease and the diversity of the symptoms, this remarkable case is most nearly approached by the following one:—

> Boy of 9¼ years. Intermittent fever one year before; later spasm of the orbicularis palpebrarum muscle. On 28th August, 1882, he suddenly fell down on his way to school and had to be carried home. The attack recurred on 4th, 15th, 19th, and 22nd September. He doubled himself up, sat or lay cowering, with intelligence unaffected, but unable to make any movement of the head or limbs on account of severe pain. No contractures. Duration about 20 minutes, after which he jumped up and went on playing as if nothing had happened. Healthy in the intervals, but could not sit still, fidgeted about in his chair, made chorea-like movements and had hyperæsthesia of the back towards the right side, where in the beginning of October a patch of herpes appeared about the size of a florin. In October the fits became more frequent, occurred without cause at varying times of the day, and changed their character. After a short preliminary stage, during which the boy sat still and stared, he doubled himself up as formerly, but continued unable to walk after the attack had subsided, having to support himself by tables, chairs, &c., and dragging his legs after him. Duration ½—1 hour, occasionally accompanied by aphasia and by spasmodic attacks of hoarse coughing. The paresis of the legs usually disappeared rapidly after a few shrill inspirations quite like spasmus glottidis, and during the intervals the power of movement was normal in every respect. In November all these conditions passed off, giving place to a state of somnambulism; he slept a great deal during the day, and made violent muscular movements as if

[1] Romberg und Henoch, *Klinische Wahrnehmungen and Beobachtungen*: Berlin, 1851, S. 77; and the 1st and 2nd editions of the present work which contain a full history of the case, p. 199.

swimming, threw about everything he got hold of, and afterwards hid them away in his bed, &c., without knowing what he was about. In the intervals he was quite well, good-humoured, and went for walks which lasted hours. In December all morbid symptoms had disappeared; apparent recovery till 8th January, 1883, when he suddenly after a motion of the bowels fell down pale and speechless in the closet and could not walk till noon on the following day. A fright from swallowing a pin was given as the cause. Nothing followed, the boy was quite well and spent some months in the Harz. After September, however, he complained of frequent attacks of headache with slight convulsive movements and lost his good temper. In January, 1884, more serious symptoms again appeared. He had attacks resembling syncope, and doubled himself up after every motion of his bowels, even after micturition. He also had painful sensations passing down from his knees to his feet; with spasmodic rigidity of the fingers when he tried to take hold of anything, and his sleep was disturbed. His general health was perfectly good. These attacks also disappeared after a short time, and since then—as far as I have been able to learn—the boy has remained healthy. In this case, then, during a year and a half there occurred varying symptoms connected with every part of the nervous system, with long intervals of almost perfect health.

It is but natural that under these circumstances—especially in the first-mentioned case—simulation should occur to one; but careful and continued observations put this suspicion entirely out of the question. It is also absolutely impossible that the child's strength should have sufficed for this sort of simulation. This enormous capacity of the muscles for work, which is quite abnormal, I consider an essential characteristic of this remarkable affection, and I have been astonished to find it in other cases also.

In a boy of 8 years, who had been perfectly healthy till 3 months before, the trouble began with a state of nervous restlessness, lasting for about 6 weeks, which gradually passed into attacks of chorea magna. These at first only occurred by night—later on during the day also. After an aura, consisting of a sensation of painful pressure over the right eye, the boy began to run, spring, and stamp about continuously, uttering from time to time a piercing scream. During the attack consciousness was confused, but not lost. After a few minutes this ended with a violent trembling and shaking of the whole body, whereupon the boy awoke as if out of a deep dream. Involuntary micturition also not uncommonly occurred during the attack. Causes and further course unknown.

An anæmic girl of 13 years, whom I treated along with Romberg, had no morbid symptoms whatever during the forenoon. Between 3 and 6 P.M., however, attacks occurred every day, in which spasmus nutans (p. 192) was the principal symptom, while the mental condition was entirely unaffected. There were nodding and swaying movements of the head and whole upper part of the body, fully 40—50 in the minute, with short pauses at intervals of an hour, and they lasted so continuously that the possibility of such muscular exertion was almost inconceivable. The attack ended about 6 o'clock. Duration of the disease at least 4 weeks, after which all sorts of other hysterical symptoms remained—extreme weakness, globus, tenderness of the scalp, and so on. The appearance of menstruation finally brought about complete recovery. I have since seen the patient again as a healthy wife and mother.

A girl of 9 years, healthy, with the exception of repeated sore-throats, was brought to me on 22nd November, 1878. A year before she had had 4 "fits" with drawing of the mouth to one side, but without loss of consciousness. In the beginning of October, half an hour after having her tonsils cauterised with nitrate of silver, she took a "fit," in which she repeatedly sprang up into an upright position, with extremely quick dyspnœic breathing, accompanied by a stenotic sound; this lasted only a few seconds. Thousands of such attacks were said to have occurred since that time, during the day only. Pot. brom. and quinine had had no effect.

Emil S., 10 years old, presenting over 100 exostoses on all his bones, which had developed since he was 9 months old, had suffered during some years past from occasional attacks of migraine, with vomiting. He was violent and irritable, but diligent and ambitious at school. On 4th May, 1869, an attack of headache, lasting from morning till midday. About 2 o'clock this suddenly became worse again and there was redness of the face, convulsions of the whole body, biting movements of the jaws, rolling of the eyes, and slight mental derangement (mistaking one person for another). All movements strikingly hurried and forcible. Duration of the attack 1½ hours, after which the child became quite quiet and the appetite returned. From 5 to 7.30 a second and more violent attack. Great tenderness to pressure in the upper cervical region. Quiet night, sleep without any spasmodic contractions. Next day, between 6 A.M. and 3 P.M., four similar attacks, in which the patient threw himself with great energy out of his own bed into that next him. This was followed by a complete cessation of symptoms, and the child seemed quite well till next morning, when, at 7 A.M., a trifling and very transient attack took place. Since that time the disease has not returned, and this boy, as I have had repeated opportunities of ascertaining, has grown up a healthy young man.

In a healthy boy of 12 years (November, 1870), the trouble began with extreme hyperæsthesia of the whole front wall of the chest. The region bounded by the clavicle and the lower margin of the thorax, and laterally by the axillary line, was so tender that he could scarcely bear even a slight touch. After about 4 weeks this hyperæsthesia suddenly disappeared, and was replaced by violent attacks of spasmodic coughing, resembling those of whooping-cough, in which the prolonged inspirations were accompanied by a whistling noise (spasmus glottidis). During these attacks, which occurred several times a day at irregular intervals, and seemed to threaten suffocation, and of which I was frequently a witness, the boy sprung up with such energy that it was with difficulty that he could be held down. Injections of morphia were the only thing that relieved him. He seemed well in the intervals apart from a certain irritability of disposition. After 6 weeks all morbid symptoms suddenly disappeared; they recurred once later on for a short time, and then disappeared for good. Course of treatment in Bad Landeck.

This case is peculiar in this, that the beginning of the disease was announced by a sensory neurosis, which I have never observed in this form except here. It is especially worthy of note that the hyperæsthesia was bilateral, and not confined to the area of distribution of one or more particular nerves, but affected the front and whole side of the thorax.

To this series we must also allocate the rare cases mentioned on p. 201, in which choreic movements are combined with unilateral anæsthesia, which again disappears with surprising rapidity, or else makes its appearance on the other side of the body (transferred). I cannot deny that partial anæsthesia or analgesia, also limitations of the field of vision (hemianopsia, &c.), may occur in the most diverse hysterical conditions of children more frequently than I have hitherto thought, either because I have not examined many cases in this particular, or because this examination is extremely difficult, and readily admits of error.[1] Only in very few cases was I able to convince myself of a bilateral anæsthesia, *e.g.* in the case of a girl of 12, whose left nasal cartilage we could pierce with a needle without her feeling it.

[1] *Cf.* Barlow's article (*Brit. Med. Journal*, Dec. 3, 1881) "On Hysterical Analgesia in Children." Barlow recommends the galvanic current for the examination. Peugniez's *Thesis*, mentioned on p. 217, *note*, contains a series of cases in which anæsthesia of the skin and organs of sense were observed in children of 10—15 years in just the same way as in adults.

The fourth class includes the cases—rare in my experience—in which neuralgic or trophic disturbances are the most prominent symptoms.

Gotthelf K., 6½ years, examined May 2nd, 1878. A fresh-looking, healthy boy. Measles 4 weeks ago with normal course. A fortnight ago, while wrestling, another boy fell on the top of him. A week after, fits of pain began in his abdomen, which have gone on getting worse. They affected the whole abdomen, even its lateral regions, and were so severe that the child screamed aloud and rolled about violently in bed. Gradually the screaming and rolling about became so marked that the pains ceased to be the most prominent symptom. The frequency of the attacks increased daily, and they were only interrupted by very short free intervals. Temp. 100·4°—101·3° F. Pulse somewhat rapid, coated tongue, fœtor oris; urine abundant, dark, normal. Bowels regular, anorexia.. Nothing abnormal in the abdomen. On the other hand, extreme hyperæsthesia of the skin over it and of the whole front of the thorax, so that violent pain was caused if one raised up a fold of the skin. Treatment:—Warm bran-baths, acid. hydrochlor.; in the evening, morphia. Next day (3rd May) the attacks diminished in frequency and severity. For 24 hours almost no urine passed, except when the bowels were moved. Hyperæsthesia unchanged, and was now found also in the face in the area of distribution of the first branch of the fifth nerve on both sides. After the 4th, rapid diminution of the hyperæsthesia and of the attacks of pain; abundant discharge of urine and fæces; appetite; no fever. On the 8th, complete recovery.

In a girl of 12 years, who had lately begun to menstruate (23rd April, 1879) violent attacks of cardialgia had occurred daily for the last fortnight, which lasted for several hours, and were accompanied by uninterrupted crying and screaming, which put the whole household in a state of excitement. Otherwise healthy, but of peevish disposition and extreme nervous irritability. Morphia, here also, rapidly had a soothing effect.

Girl of 11 years, unusually early developed, but has not yet menstruated. Her mother died of phthisis. In September, 1878, I was consulted on account of frequent attacks of headache, which were often accompanied towards evening by an inclination to vomit. In February, 1879, I saw her again. Ten days before, she had violent retching, with hæmatemesis and general uneasiness, during which about half a cupful of blackish-red blood was brought up, mixed with much mucus. This had recurred every second evening about 8.30. The attack lasted about a ½ hour and never occurred during the day. Owing to sensations in the region of the right mamma, I frequently examined the lungs along

with the physician in charge, but never found anything to excite suspicion. During the last 4 days the hæmatemesis had occurred every evening at the same time—about 8.30. The motions never contained blood. Food was well borne, never giving rise to pain in the stomach. Neither quinine in large doses (grs. xv.) nor remedies given for the gastric condition, an ice-bag, opium, milk diet, liq. ferri perchlor., nor ergotin, had the slightest effect.

The peculiar character of the girl, her premature development, her inclination to stay in bed, and the fact—which her father himself acknowledged—that she had been extremely spoilt from childhood, at once led me to suspect that we had either to do with simulation or hysteria. There was no reason for the former, and examination of the teeth, throat, tongue, &c., revealed nothing which could be regarded as the source of the vomited blood. Also the physician in charge had himself witnessed an evening attack, and was convinced that there was no simulation. We could therefore only think of hysteria, and I was further strengthened in this supposition by the fact that the hæmatemesis occurred by day for the first time on the 12th, about 2 P.M., subsequently to mental excitement. We therefore ordered the patient to leave her bed, to take a drive every day, and recommended that all medicine should be given up and all anxiety dismissed. In the middle of May I met father and daughter taking a walk, and the former told me that since my last visit there had been no other attack, and that the girl was perfectly well. Her good health continued the whole summer, while she was in the country. Only extremely rarely—and always after mental excitement—did slight hæmatemesis occur. After her return home the same series of symptoms, occurring in the evening, again appeared, but not so regularly as before. Ergotin injections, which the doctor ordered, had evidently a psychical effect, for the mere threat of repeating them later on (*e.g.* in August, 1880) when traces of hæmatemesis again appeared, was sufficient to cause immediate recovery.

This is the only case in which I have seen hæmatemesis accompanying a hysterical affection, although such cases have occasionally been reported by other authors.[1] As I have seen hæmoptysis without lung disease in a hysterical patient, I regard the occurrence of hæmatemesis under similar circumstances as equally possible. The process is indeed difficult to explain, and may always remain a matter of hypothesis. But when I remember the sudden blushing which may take place from mental emotion, and recall the case of one epileptic child

[1] *Cf.* Rathery, "Contributions à l'étude des hémorrhagies survenant dans le cours de l'Hystérie:" *Union Méd.*, 1880, No. 32, 35.—Lancereaux, "Hémorrhagies neuropathiques": *Ibid.*, No. 56.

whose attacks always began with extreme flushing of the whole skin as aura, I think I may assume that it is possible for hyperæmia and hæmorrhages to take place into the lungs or stomach from an irritation affecting the vaso-motor nerves of these organs. The periodic occurrence of hæmatemesis in our case is not surprising, seeing that in some of the cases of chorea magna formerly published the convulsions took place in the most typically periodic way. I may mention here also the case of a boy of 9 years who had his "hysterical" convulsions regularly about noon and at 5 P.M., and in whom there could be no suspicion of simulation.

The cases I have given will suffice to place before you a clinical picture of this remarkable condition in its various forms. These cases do not, indeed, exhaust all the modifications, and I might have given you from my own practice examples of many other variations and combinations of symptoms—cases of aphonia, aphasia, globus, hiccough, and dysphagia. Thus we find published accounts of neuralgia in the joints, ovarian pain, and localised hyperæsthesia and anæsthesia, in no way differing from those in hysterical adults.[1] Their strange and inexplicable character always, of course, excites a suspicion of simulation; and indeed we cannot be sufficiently cautious in this particular, even in the case of children.[2] I have myself met with a few such cases; among others that of a girl of 12 (25th Feb., 1879), who had suffered for two years from frequent cataleptic attacks, and had latterly taken them three or four times in the day, but from the moment she was admitted into the children's ward to the time of her discharge (that is, for at least 2 weeks) had not a single fit. Apart, however, from the fact that cases of this kind are not, in my opinion, to be regarded off-hand as cases of intentional malingering, but rather as an expression of the "hysterical" nervous derangement, I can assure you that in all the cases given above, the suspicion of simulation could be absolutely excluded; and it was just the same in many analogous cases recorded by other writers. I cannot, therefore, entirely agree with Roger when he says, "pour les practiciens experts en pathologie infantile, toute neurose dite par imitation est une

[1] Rosenstein (*Berl. klin. Wochenschr.*, 1882, S. 522) describes a remarkable case in which there was vomiting of scybalous fæces during the attack.

[2] S. Abelin, *Centralzeitung f. Kinderheilk.*, 1878, S. 257.

neurose par simulation." The complete cessation of the fits in the child just mentioned during her residence in the hospital cannot be taken as a proof of malingering, as we know for certain that radical changes in the surrounding conditions not uncommonly produce a temporary or even lasting improvement of this "nervous" state.

Occasionally the resemblance to the hysteria of adults is even more striking, as in the following case:—

> On 5th November, 1876, a girl of 11 years appeared at the polyclinic, who had been quite blind since her 2nd year as the result of bilateral keratitis and atrophia bulbi. Being healthy till 2½ years ago, she was sent to school, where she showed the utmost application and overworked herself. Soon after, she took attacks of headache, with vomiting, so that she had to leave the school. She took to music with all the more energy; she had a marked talent for it, and now played the piano for more than 3 hours daily—of course, only by ear. For some months she had complained of sudden shooting pains in the forehead, and giddiness (so that she fell) alternating with violent colicky pains round the umbilicus and attacks of rapid dyspnœic breathing. All these symptoms occurred every day repeatedly, and at once whenever you spoke to the child about them. At the same time her mental character did not at all correspond to her age, for she was precocious, extremely talkative, and very circumstantial in describing her symptoms. Particularly striking and amusing was the fact that she always repeated exactly the last word of anything her mother said. At the same time she slept 12 hours continuously without being troubled by a trace of nervous symptoms. General health perfectly good. No sign observable of the development of puberty. Further course unknown.

I have also several times had occasion to observe cases of hysterical paralysis of the lower extremities in children, especially in girls of 11—13, even more marked than in the cases given on p. 223 and p. 225. Sometimes violent fits of crying, lasting for weeks, or other hysterical conditions had preceded the paralysis; and it came on after they disappeared, just as in adults. In lying and sitting the limbs could be moved almost as well as in the normal condition, and the sensibility as well as the function of the sphincters was intact. The children, however, obstinately maintained that they could not stand or walk, and when they tried to do so, their strength failed and they sank to the ground unless supported. The suspicion of

spine disease, which causes anxiety to the parents in such cases, could be at once discarded, and in fact these paralyses disappeared after a few weeks, either spontaneously or as the result of psychical impressions. But sometimes they were replaced by other nervous symptoms.[1]

Quite as obscure as the pathology of all these outwardly dissimilar but essentially identical conditions is their etiology. In hardly any case have I been able to find quite definite causes. The influence of emotional conditions—particularly fright—in causing relapses, must be acknowledged. One of these girls suddenly took a violent hysterical convulsive attack (the first for weeks) during my lecture on her case, at which she was present. In general it is commoner in females and at the time of puberty, and accordingly all these affections, especially chorea magna, have been closely identified with the latter. Since, however, even boys and young children between the ages of 9 and 11 years are by no means exempt, it is evident that there may be other forms besides those due to development, arising from other causes. One naturally turns first to irritation connected with the genital system, and thus we hear masturbation spoken of by many as the principal cause of these nervous disturbances.[2] I do not by any means deny that, with a strongly-marked "nervous predisposition," this vice if persisted in may assume importance as a cause; but, considering how common it is, we should certainly meet with cases such as we are speaking of far oftener than we do, if that view were correct. We are at any rate always justified in keeping this cause distinctly in mind. You will scarcely believe that many children in the second year of life, or even earlier, practice masturbation, either with the hand or by rubbing the thighs together, so as to cause distinct erection of the penis. It is often also produced by the already mentioned rhythmical swaying of the upper part of the body while sitting (p. 196). At this age the evil can still very easily be cured by sharp supervision, but it is much more difficult in older children, who in some cases will avail themselves of every unwatched moment to indulge in the vice. I remember

[1] *Cf.* Riegel (*Zeitschr. f. klin. Med., Bd*, vi., H. 5), who gives five cases of this paralysis with contractures, &c.

[2] Jacobi, "On masturbation and hysteria in young children": *American Journ. of Obstetrics*, &c., viii. 4; ix. 3, 1876.—Hirschsprung, *Jahrb. f. Kinderheilk.*, xxiii., 460.

one girl of 8 years, who although she did not use her hands, yet by rubbing the genital organs on the edge of the chair on which she sat, worked herself into a state of great excitement, which was manifested by her flushed cheeks, sparkling eyes, and rapid breathing. The diagnosis, however, is not always so easy, and the most careful observation is necessary, especially when they are going to sleep, in order to surprise them *in flagrante*. The discovery of a few spots on the linen is by no means sufficient for a positive diagnosis. I have tried in all cases of hysteria and chorea magna to investigate this point, but in not a single case have I been perfectly sure that the cause was to be found in masturbation. We must always be content with the possibility or probability which already play too large a part in etiology. Nevertheless, you will do well always to keep masturbation in mind, and, whenever it is found to be present, to put a stop to it if possible. For even although it may not constitute the real cause of the disease, still by the over-excitement of the nervous system which it occasions, it may prepare the way for its development and retard recovery. How serious such an over-excitement may become, we see from the following case :—

> Carl A., 7 years old, admitted into the children's ward on 8th January, 1873, had practiced masturbation since his fifth year. The habit had been induced by sleeping for a long time with a female relative, who had taught it him. Gradually increasing debility, enuresis nocturna, sleeplessness, and—during the last fortnight—inability to walk. He could neither sit, stand, nor walk unless supported. Even when supported he soon began to sway about, complained of giddiness, and his gait was distinctly ataxic, like that in tabes dorsalis. On shutting his eyes, the symptoms were markedly increased. In bed, all movements of the legs were free, although less energetic than in normal health. Sensibility intact. The plantar reflex movements, however, were weaker and slower than usual. Urine and fæces retained with difficulty, and sometimes passed involuntarily. Anæmia and moderate emaciation. Treatment:—A luke-warm bath for 10 minutes daily, with cold shower over the head and back, the strictest supervision of the patient, and the prevention of every attempt at masturbation. By 23rd marked improvement in walking, cessation of enuresis. On 31st scarcely the slightest unsteadiness in the gait noticeable. Complete recovery by middle of February.

The extremely rapid and favourable progress of this case, which

at the beginning showed symptoms of advanced tabes dorsalis such as I had never before met with in a child, proves that no degeneration but only a functional disturbance existed. We see, then, that constant irritation of the genital nerves in children may cause paresis of the lower extremities with ataxic symptoms, diminished muscular sense, and diminished energy of the centres analogous to the hysterical paralyses in women which are caused by morbid conditions of the sexual organs, or even in the absence of such by depressing general influence on the nervous system, and which under favourable circumstances have a similarly favourable course. To the same class also belong the paresis and ataxia of the lower limbs which is occasionally observed in children with extreme phimosis, and the consequent genital irritation which this excites, and which is cured by an operation.[1]

Most of the children who presented one or other form of the hysterical conditions we have been speaking about, were of delicate constitution, thin, and more or less anæmic. Only the minority were well nourished. We could almost always find some fault in the bringing-up which had prepared a favourable soil for the later neurosis. Children who are brought up with unusual care and indulgence—round whom, so to speak, the whole household turns—who are surrounded by extremely indulgent persons ready to give in to all their humours, and whose slightest complaint was taken up with exaggerated solicitude and made much of, are especially liable to these extraordinary diseases. Under these circumstances a sort of hypochondriasis occasionally sets in. I witnessed this especially in one very spoilt, delicate boy, of 8 years. He attended to his own health with the most anxious solicitude—examined his tongue, every spot that appeared on his body, &c. In a disposition of this sort, or where there is a hereditary tendency, or at least a neurotic predisposition in the family, all irritation acting powerfully on the nervous system, every kind of emotion, excessive mental strain, ambition at school, ill-treatment from parents, and finally also the instinct of imitation may bring the disease to its full development.

From the cases I have given, you will have seen that under these circumstances medicinal treatment cannot promise any

[1] *Oesterr. Jahrb. f. Pädiatrik*, vii., 1876, 2. Heft, Annal. S. 128.—*Arch. f. Kinderheilk.*, viii., S. 460.

real result. I know of no medicine which has done me real service except chloral (in doses of grs. viiss—xv) and morphia (by the mouth and by subcutaneous injection, gr. $\frac{1}{13}$—$\frac{1}{7}$). I have found these occasionally of some use in palliating the violent spastic symptoms. The inhalations of chloroform which I have tried in attacks of screaming and other voice-spasms had never more than a passing effect. In many cases—for example, in those of spasmodic running and jumping—even these remedies can only be used with difficulty—if at all—during the paroxysms; or they may fail to act. Under such circumstances we must just let the attack run its course, only taking care that the patients get no injury from the nature and severity of their movements. Sometimes by a sudden violent impression—*e.g.* by splashing the face with cold water, or by speaking loudly and roughly—we may put a stop to the fit. Still, this by no means always occurs. We have just as little power to shorten the course of the disease by any remedies. Even when the periodicity of the attacks was most distinctly marked, I have seen no action whatever either from quinine or arsenic. Considering the frequency of an underlying anæmic condition in such cases, it is always well to treat the children with small doses of iron, or to give arsenic as in chorea; for this medicine in small doses continued for a long time exerts a distinctly beneficial influence on the anæmic constitution. Soothing baths of lukewarm water, with soap or "bolus alba" (1½—4 oz. to each bath) continued as long as possible (half an hour), nourishing food and fresh air are to be strongly recommended, but unfortunately cannot always be obtained. In affections of the voice the galvanic current should be tried. It occasionally produces rapid recovery, but sometimes has no effect, or may even aggravate the disease. Not uncommonly all manipulations of this kind—the application of electricity, the introduction of an œsophageal tube, a subcutaneous injection, even a laryngoscopic examination and, above all, the threat of repeating these measures—act with wonderful rapidity; their influence being, of course, only psychical. One must not however expect too much from this rapid improvement; for it may be followed by sudden aggravation of the symptoms. Fortunately we are in a position to reassure the relatives from the beginning as to the result, and indeed I am of opinion that the more extraordinary and incompre-

hensible the symptoms are, and the more thoroughly they change, the more certainly can a favourable prognosis be given. You may therefore always give a most favourable opinion of cases of so-called chorea magna, of voice-spasm and hysterical paralyses. But the cataleptic form (our first class) is always a cause for anxiety, because of the possibility of its turning into epilepsy (p. 218). At any rate I advise you to prepare the relatives for quite unexpected symptoms. Where there is now paralysis there may in a few days be a convulsive affection, a sensory neurosis, or a psychical change; and this sometimes takes place even during an attack.

After recovery, you will do well to continue the tonic treatment, and, where circumstances allow it, to order chalybeate baths, or else simple warm baths in fresh mountain or forest air. As to the latter, I recommend especially the warm baths of Schlangenbad in Taunus, Landeck in Silesia, and Johannisbad in Bohemia. For chalybeate baths, which are indicated when anæmia is a prominent symptom, I would advise Schwalbach, Pyrmont, Driburg, Flinsberg; and, in Switzerland, the high springs of Tarasp and St. Moritz.

I have no doubt that by this treatment with baths and change of air, the recurrence of the conditions we are speaking of may be prevented, and their course so far shortened. I believe that under favourable circumstances a course lasting over a number of years, as for example in our case on p. 226, will hardly ever occur. When the disease is extremely obstinate, however, nothing remains but to remove the patient from his accustomed surroundings at home, into others which are quite new to him, either in a hospital or in a strange family. The mere change of abode is not in itself sufficient, unless the companionship of the mother, or accustomed nurse is also denied. School attendance is, of course, to be forbidden while the disease lasts; and, even after recovery, every mental strain is to be carefully avoided. In girls about the age of puberty, the appearance of menstruation calls for special rest and care. We learn from the case on p. 226 that when puberty is fully established, even unusually chronic conditions of this kind may end favourably.

VII. *Pavor Nocturnus* (Night Terrors).

This is the name given to a condition which, owing to the alarm which it causes the patients, often disturbs the well-earned rest of the physician. In the middle of deep sleep—oftenest in the first hours of the night—the children suddenly start up and cry violently and continuously, and catch at the air with their hands, or else sit in bed staring in front of them with an anxious expression, and uttering words that are hard to make out, or altogether unintelligible. Many tremble in all their limbs, throw themselves in terror into the arms of the frightened mother or nurse, cling to them without distinctly recognising them and call out for light, and it is only with difficulty that they can be quieted. After a short pause the scene is repeated, not uncommonly several times in succession, so that half an hour or longer may pass before complete rest ensues, and the exhausted child falls sound asleep again. As a rule, the remainder of the night is passed in quiet sleep, and when the child awakes it knows nothing of what occurred in the night, and does not remember the physician who sat by his bedside during the attack. These attacks are now repeated at irregular intervals, sometimes every night, sometimes only twice or thrice a week, or still seldomer. It is exceptional to have two attacks in the same night. During the day, the children show no symptoms that one can connect with the nightly paroxysms. I have only once had the opportunity of observing a case of this kind—between 11 and 12 in the forenoon—in a child who had fallen asleep on a sofa. The duration of this disease, which so violently disturbs the child's relatives, is quite indefinite. While in some cases the thing is all over in a few attacks, in others the attacks are repeated during many weeks or even months; but they finally disappear without leaving any bad results. In an anæmic girl of 7 years who was otherwise quite healthy, the attacks had lasted two years, with maximum intervals of 8 days, but had increased in frequency since she began attending school.

Although I have placed this affection here, immediately after the "hysterical" conditions, it is not at all because I consider them to be nearly related to one another. I have indeed seen

pavor nocturnus come on, in a few cases, in children who had been spoilt, and had been rendered hypersensitive by a bringing-up which predisposed them to hysterical derangements, and who suffered at the same time from headaches, palpitation, fainting fits, &c. But this however was just as rare as it was to find night-terrors due to real epilepsy; which I found to be the case in a girl of 10. In this case several epileptic fits had taken place three years before, at intervals of 8—10 days. They then ceased till January, 1882, when suddenly several fits again occurred, which in February were accompanied by hallucinations and screaming. In March they disappeared spontaneously, and were replaced by attacks of pavor nocturnus, occasionally occurring twice in one night. I have never yet met with pavor preceding and accompanying regular psychoses, which is perhaps due to the small number of cases of mental affections which I have met with in children.

In general we meet with pavor nocturnus almost exclusively in young children, in whom we find it occurring till near the time of the second dentition; while "hysterical" conditions usually begin after this period. In this condition also there is none of that mental change which is so important an element in hysteria. The whole trouble consists in the nocturnal attacks described, and to me at least it has always appeared as if a terrifying bad dream had frightened the children out of their sleep, and still haunted them when half awake. It is evident that visions and hallucinations are factors, as the children often describe them quite definitely. I have heard them call out to take away the chains, to drive away the wild beasts, that they would be run over, &c. Sometimes, again, they try to jump out of bed to escape from the cause of their terror. A boy of four years who was violently frightened by a bee had an attack of night terror on the night after, during which he fancied that a fish was continually threatening him. This was repeated several nights consecutively, and finally the child would not enter the bedroom, and always wanted to be out of doors. The more active the child's fancy is, and the more it is excited by the favourite thrilling tales of nurses, the more readily will the pavor come on; and this fact is one which should be laid to heart by those who have charge of children.

One of the rare cases of pavor diurnus which I have seen affected the son of an actor (7 years old), a nervous, anæmic, delicate child. For some months as many as 10 or 20 attacks took place daily, but never during the night. The child would shut his eyes and stop his ears, crying, "I'm afraid!" and clinging to his mother. Duration only a few seconds. Otherwise healthy, and, in particular, free from other hysterical symptoms. In a "nervous" child of 6, who had suffered from pavor nocturnus for 7 months, with intervals of about a fortnight, attacks occurred occasionally by day with hallucinations. Unfortunately, both these cases were lost sight of.

I cannot share West's opinion, that disturbances of digestion are generally the cause of night-terrors. I have but rarely been able to assure myself that the cure of such dyspeptic conditions as might happen to be present caused a rapid disappearance of the pavor; *e.g.* in a boy of 8, who during an attack of gastric catarrh had night-terrors five nights running. On the other hand, most of the cases presented no disturbance whatever of the digestive organs. Nor could I discover any abnormal condition of the respiratory and circulatory organs.[1] In many cases there is an undeniable family predisposition; children of nervous parents are more likely to be affected. As I was unable to ascertain the causes in most of the cases, I confined myself to forbidding every excitement of the child's fancy by evening stories, and ordering a dose of bromide of potash (grs. viiss—xv.) at bedtime; and this seemed to me to exert a soothing influence. I have not yet tried morphia or chloral, but I would have no hesitation in using these remedies in severe cases.

VIII. *Peripheral Paralyses.*

In children, as in adults, the facial nerve is that most frequently affected by peripheral paralysis. It not uncommonly appears in the earliest childhood, immediately after birth. The mouth is drawn to the unaffected side in crying, and the eye of the paralysed side often remains open. The exact symptoms depend on whether the cause of the paralysis affects the labial and palpebral branches of the facial nerve, or leaves the latter unaffected. This cause is the pressure of forceps

[1] Silbermann, *Jahrb. f. Kinderheilk.*, Bd. xx., S. 266.

at birth, which in such cases sometimes leaves behind a small ecchymosis in the parotid region. The twisting of the mouth generally causes the utmost alarm to nurses and parents, as it is regarded as a sign of apoplexy. You may, however, calm the fears of the relatives by the assurance that the paralysis 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 compression. I say "probably," for you cannot foretell a favourable termination with absolute certainty. In a few cases the pressure of the forceps appears to have been so severe and lasting in its effects that degenerative processes (fatty degeneration of the nerve fibres) take place in the facial nerve; and these are not always recovered from, but cause paralysis lasting for a whole life-time. I have myself observed one such case, in a girl of 13 years, and Parrot and Troisier[1] have furnished anatomical proof of the fact.

Much more rarely we find congenital paralysis of the facial nerve, with which the pressure of the forceps has nothing to do. I have seen this only once, in a boy of 10 years, who was born without artificial aid, and exhibited paralysis of the left facial nerve immediately after birth. All its branches were paralysed, also the left half of the soft palate and the hearing was lost in the left ear, although no disease of it had ever been found. A prolonged treatment by galvanism was entirely unsuccessful. Similar congenital cases have also been published, but their pathology is not sufficiently explained.

Unilateral paralysis occurring in later childhood has a general correspondence with the cases with which you are familiar in adults, and I shall not discuss them further. I would point out to you, however, that in order to observe these symptoms it is necessary (in children almost more than in adults) to make the features move in the expression of some sudden emotion. While the child's face is at rest you observe no striking change; but when it cries, screams, or laughs, the asymmetry of the two sides becomes apparent. The inspection of the soft palate is often particularly difficult in children, and we have sometimes to be content with a rapid glance. The causes, as well as the general symptoms, agree entirely with

[1] "Note sur l'anatomie pathologique de la paralysie faciale des nouveau-nés," *Arch. de Tocologie, Août*, 1876.

those of facial paralysis in adults. Rheumatism as a cause is here also more frequently taken for granted than proved. Still cases are not uncommon in which the action of a cold draught of air—especially when the skin is perspiring—is evidently the cause. More frequently, I have seen the scars of abscesses, or enlarged glands, behind and under the ear in the region of the stylo-mastoid foramen, cause paralysis by their pressure on the branch of the facial which issues from it.

Child of 2 years, with complete paralysis of all the branches of the left facial supplying the face. In the neighbourhood of the stylo-mastoid foramen, a deep sinuous abscess issuing from a lymphatic gland. After it was opened there remained a considerable swelling and infiltration of the connective tissue. From 25th February, 1861, this was painted with tincture of iodine. On the 7th March there was considerable diminution of the swelling; but the paralysis was unchanged. Continuation of the painting, and also, internally, iodi gr. $\frac{2}{3}$, pot. iodid. grs. xv., syrupi simpl. ʒ viss., aq. destill. ad ℥ iii., a dessert-spoonful 4 times a day. In the beginning of April, complete recovery.

Such cases occasionally occur even in very young children. Thus I have seen paralysis of the right facial nerve in two children of 5 and 11 months respectively. In the latter, enlargement of the glands, with diffuse swelling of the connective tissue, could be made out in front of, behind, and under the ear, while in the first case very careful examination was needed in order to make out the deep-seated induration under the mastoid process.—In a boy of 4 years, paralysis of the labial and nasal branches of the left facial resulted from the pressure of a large abscess in front of the ear, which developed during convalescence from typhoid fever. The paralysis disappeared almost suddenly when the abscess burst into the external auditory canal and discharged its pus into it.

We must, however, regard caries of the petrous bone destroying the nerve-trunk in the Fallopian canal, as the commonest cause of facial paralysis in childhood. The numerous cases of this kind which I have seen all agree in this —that in every one of them all the facial branches of the nerve were paralysed, while unilateral paralysis of the soft palate was not always present; for in a number of these cases the uvula was quite straight, and the movement of the palate equal on the two sides. We must notice in these cases not only the oblique position of the uvula, but also the movement of one half of the velum on breathing and phonating, whereby the

soft palate is twisted to one side. Where this symptom is absent, we may conclude that the destruction of the Fallopian canal has not taken place till after the greater petrosal nerve has left it. Deafness in the affected ear is very difficult, if not impossible, to make out in little children. The otorrhœa, which is always present, sometimes combined with bleeding, is all the more important, and along with the matter there are often discharged from the auditory meatus little or pretty large pieces of bone, or even auditory ossicles, clean as if dissected. The presence of a deeply destructive process is also indicated by a tender swelling of the temporal bone behind the ear, also by redness and fistulous openings. This cause of paralysis sometimes occurs at a very early age. I have seen it begin even in the third and fifth months, and either rapidly prove fatal with symptoms of general tuberculosis, or else continue for years, till at last death was caused by complications, especially tuberculosis of the brain or other organs, meningitis, or sinus-thrombosis. The longer the paralysis continues the more atrophic do the facial muscles become, and in one child thus affected I found them shrivelled to thin brownish-yellow bands. At the post-mortem of the cases I have met with, there has always been extensive caries or cario-necrotic destruction of the petrous bone, which sometimes reached to the dura mater. But even in the cases where there was a carious cavity close under it, this membrane itself was intact, or at most somewhat dark in colour, so that a perforation of the caries into the cranial cavity had certainly not occurred. On the other hand I have repeatedly found pachymeningitis and localised purulent arachnitis. A long sequestrum could sometimes be extracted from the external auditory meatus at the post-mortem, and then when the auricle was removed we could see into a considerable cavity occupying the larger part of the petrous bone. In a few cases we could extract pieces of dead bone even during life, either from the meatus or from a fistulous opening in the mastoid portion of the temporal bone. The abscesses and fistulæ behind the auricle always communicated with the interior of the carious bone. In one extremely cachectic and anæmic boy of 3 years the external ear was almost completely separated from the head by a semi-lunar gangrenous fissure, and from this we were able to remove a sequestrum $\frac{4}{5}$ in. long and $\frac{2}{5}$ in. broad.

Almost all the children in whom I observed this paralysis were also tubercular, and died sooner or later. In one of these cases there were numerous nodules (ranging in size from that of a millet to that of a hemp-seed) on the dura mater of the middle cranial fossa. Less commonly the caries arose from the neglect of a simple otitis media, especially when this was a sequela of scarlet fever; and I therefore recommend you when children are recovering from scarlet fever always to pay special attention to any otorrhœa that may remain. Some of the cases which I have had to do with showed that the destructive process, which begins in the middle ear and spreads to the bones, may have a surprisingly short course, and may lead to caries of the petrous bone with facial paralysis even in a few weeks after recovery from scarlet fever.

The peripheral paralysis of other cranial nerves is much less common in children, and presents in them even less that is characteristic than facial paralysis does. This is also true of the paralysis of the spinal nerves due to local causes. Among these there is only one that arises at birth, and which on account of this causation calls for remark here. Not only on the facial nerve but also on the brachial plexus, the forceps may exert so strong a pressure that paralysis of one or more groups of muscles in the affected arm may take place. Roger[1] describes one such case in which immediately after birth the facial nerve and one arm were both paralysed. The impress of the forceps over the clavicles was still visible, and after death—which soon followed—effusions of blood were found both in the neighbourhood of the stylo-mastoid foramen, and in that of the brachial plexus. Other obstetrical processes may however also have the same effect as the pressure of the forceps, especially difficult extractions or violent dragging of the arm, along with which dislocation or fracture of the humerus has been occasionally observed. The hæmatoma of the sterno-mastoid formerly mentioned (p. 39) may also occur under these circumstances. This "congenital" (or really "artificial") paralysis of the upper extremity may, like that of the facial nerve, either pass off rapidly or—should degenerated processes have been set up in the nerves of the arm by the cause of the paralysis—continue many years or even during the whole lifetime. It may also be combined with sensory

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

disturbances. Thus I have observed, in a child of five, anæsthesia occurring with the paralysis on the ulnar side of the forearm. The position of the arm, which is due to the contraction of the antagonistic muscles, varies according to the muscles affected. Most frequently there is rotation inwards with marked pronation of the hand, owing to the action of the pectorals, subscapularis and latissimus dorsi being stronger than that of the paralysed infraspinatus. The faradic irritability of the paralysed muscles rapidly disappears and atrophy of the affected limb soon sets in, in which—as I have frequently seen—even the bones may participate, so that finally the scapula and the bones of the arm and hand are considerably shortened as compared with those of the healthy side and the whole limb appears stunted. Nothing can be expected from treatment, except in the earliest stages of the disease. The continuous application of electricity, especially the galvanic current, may still be of use so long as the nerves are not fattily degenerated and the muscles are still capable of reacting. At a later stage we can expect nothing either from this or any other remedy whatever.

An excessive stretching of the brachial plexus may in later childhood, as in adults, occasion paralysis or at least paresis of the upper extremity sometimes lasting for weeks or months. I have observed, for example, paresis of this kind in the left arm in a little girl whose arm had been violently wrenched backwards and outwards while her jacket was being put on. The movement of the limbs, especially upwards and outwards was extremely limited, and it was only after several weeks of the continuous application of stimulating friction and finally of electricity, that the function of the deltoid was completely restored. Such cases, if the cause is obscure, may occasion great anxiety; since not only the parents but even the conscientious physician may not be able to exclude a cerebral origin of the paralysis until the improvement decidedly begins. The same may be said of the paresis or paralysis of an upper or lower extremity which children occasionally have for some days after violent convulsive attacks. It is not possible in these cases to determine at once whether we have to do with a passing disturbance of motion or with a cerebral disease, since, as we shall see presently, very serious cerebral diseases—especially tubercle—are not uncommonly announced by the sudden oc-

currence of convulsions, which leave paralysis behind when they go off. It disappears again after some time, then returns quite unexpectedly; or the true nature of the disease may be revealed by the onset of tubercular meningitis. I therefore advise you in the diagnosis of all localised paralyses, when their peripheral origin is not beyond doubt, to be very guarded and to keep in mind the possibility of a central disease even although no further symptoms of such should be present.

One must of course also, under these circumstances, always remember the possibility of an injury of the affected joints, of a dislocation or subluxation of the joints of the shoulder and forearm, even of fractures of the bones, and examine carefully for these conditions. I should not have mentioned this had I not several times found in the polyclinic that these traumatic affections had been called paresis by careless practitioners. The contrary sometimes occurs in the lower limbs, where a dragging of the leg or a slight limp is falsely ascribed to commencing coxitis, when it is only the result of the bruising of the muscles by a fall, and disappears in a short time if the child is made to rest.

IX. *Spinal Infantile Paralysis.*

This disease—which, before its pathology was known, was described by the name of "essential paralysis"—derives its particular interest from its comparative commonness, and from the severe effects which it has during the whole lifetime of the patient. Most of the cases you meet with affect children between one-and-a-half and four. The parents state that the child some weeks or months before lost the power of an arm or leg, or even of several limbs. On examination, we find in a certain proportion of the cases that the affected limb is really quite motionless. The child does not make the slightest attempt to grasp anything or to stand on his feet. The whole limb is as flaccid as that of a doll, so that you can throw it about in all directions without resistance. The sensibility, on the other hand, is almost always unimpaired. In other cases the paralysis is already beginning to diminish. Certain movements of the limb can be performed, others are quite impossible. Thus, for

example, the forearm can be pretty well flexed and extended at the elbow joint and the hand at the wrist, while movements of the upper arm outwards and upwards, and the pronation and supination of the hand are either quite impossible or can only be effected to a very limited extent.[1] All this time the child is usually quite well; all its functions are in good order, and its appearance generally very good. The sphincters of the bladder and bowel are only exceptionally affected. The commencement of the malady is almost always described by the relatives in the same way as in the following cases, which I give as examples.

On the 20th July, 1874, a girl of 4 years was brought to my consulting room. Formerly healthy, she had taken ill suddenly in September, 1873—that is, about 10 months before—with violent fever, the temperature rising to 105·8° F. The child complained at the same time of headache and was drowsy. There were no other local symptoms. After 2 days, the temperature fell. When she tried to stand up, we noticed paralysis of both lower extremities and of the right arm. In the course of 3 or 4 days power returned to the legs; she could then walk, but the arm remained paralysed, and on examination it presented the characteristic symptoms which we are about to describe.

Child of 1½ years, brought to the polyclinic on 15th October, 1881. Three weeks previously, fever lasting for several days. This was succeeded by paralysis of all four extremities. When brought to me, the movements of the arms had already almost returned to the normal state, but the paraplegia remained unchanged. One week later the left leg was also tolerably well moved, while the right was completely paralysed. Sensibility perfectly normal.

This is the usual course. In the midst of perfect health the children become feverish (occasionally the temperature is very high), they complain of headache if they are old enough, and are somewhat drowsy. More rarely they lie in a regularly comatose, half-conscious state, out of which they can only with difficulty be roused by shaking; or they may even show convulsive movements and contractures. Still more rarely the disease begins with convulsive fits, and in one of my cases these were repeated 7 or 8 times in one night. After a few days—or a week at most—this condition passes off, and the parents are alarmed by

[1] For particulars on the localisation of paralysis in certain groups of muscles, and their relation to corresponding patches in the spinal cord, *vide* E. Remak, *Archiv f. Psychiatrie und Nervenkrankh.*, Bd. ix., Heft 3.

finding that one or more limbs cannot now be moved. In a less numerous class of cases the preliminary febrile stage seems not to occur at all, and the paralysis comes on almost suddenly, without any premonitory symptoms, in the morning after a good night's sleep. Without wishing to deny that this form of onset occurs, I still think that the relatives—especially in the lower classes—often overlook slight preliminary disturbances. Now, as to the seat of the paralysis, either both legs and one arm, or a leg and an arm on different sides, rarely an arm and leg on the same side may be affected (in a hemiplegic form); or still more rarely it may be both arms, and more frequently both lower limbs, and sometimes even all four extremities. The paralysis is also often confined from the first to one limb only. The characteristic point however is, that the paralysis almost always reaches its worst at the very beginning; all the harm that is done, is done at once (as in the apoplectic paralysis of adults), or at least in the first 24—48 hours. After that there is a distinct tendency towards improvement. Only quite exceptionally have I been told that the paralysis continued to increase after the first week, or passed after some days from one of the lower extremities to the other, which Duchenne also observed. The power of motion is in many cases very rapidly recovered, as in those just given. Even after a few days, or after a week, one or other limb is once more able to exercise its functions; or some groups of muscles in a limb may be capable of motion, while others remain absolutely paralysed, so that we have an incomplete paralysis of the affected limb. In the upper extremity, the muscles of the shoulder and upper arm are especially affected, less commonly those of the forearm, so that the hand and fingers can generally be moved; while in the lower extremity, the muscles of the leg, supplied by the peroneal nerve, and in the thigh, the quadriceps muscle, are especially apt to be paralysed. After some weeks the paralysis is oftener still confined to a single group of muscles in one arm or one leg, but in these they usually remain with a sad persistence. After many months, and even years, the condition may be unaltered, and it not unfrequently remains so for the whole lifetime. In other cases, however, the paralytic symptoms, after remaining for months, improve in a most surprising way, as *e.g.* in the following case:—

Child of 2 years, brought to the polyclinic on 17th March, 1882. Seven months before, fever lasting some days, and general malaise. This was succeeded by paralysis of the muscles of the neck and of all four extremities. After some weeks the head could again be held up, but the paralysis of the upper and lower extremities persisted almost unchanged for three months, so that the child could not grasp anything, and was unable to leave its bed. After this time the paralysis of the right arm and left leg disappeared under electrical treatment; finally also that of the right lower limb and of the left forearm, so that when he was shown in the hospital there was nothing to be made out but paralysis and atrophy of the upper arm, especially of the deltoid.

When the paralysis has existed for some weeks, or even months, a number of additional symptoms appear which must be regarded as quite characteristic, and which at once place the diagnosis of the disease beyond a doubt. These symptoms are: increasing atrophy of the paralysed extremity, diminution of its temperature and of its electro-muscular excitability. The paralysed limb diminishes steadily in circumference owing to wasting of the muscles. The region of the deltoid and the shoulder muscles, especially, wastes in a very marked manner, so that a space may be felt between the acromion and the head of the humerus, and the shoulder seen from behind looks much flatter than the healthy one. The upper arm and forearm also become wasted as a whole, all the muscles are shrivelled and thin and the ligaments strikingly loose, so that the affected limb may appear a little longer than the healthy one. In very fat children the atrophy of the muscles may appear less than it really is, owing to the amount of adipose tissue. When the hand is applied we feel distinctly the lowered temperature of the paralysed limb compared with that of the healthy one; and we have been able by a suitably-constructed thermometer, to measure this diminution, which may amount to 1·8° F. The behaviour of the muscles to the electric current is also very characteristic. I have no experience of the increased faradic and galvanic reaction which some (Benedikt) have observed during the initial stage of the disease. When the paralysis is present however the reaction disappears almost as completely as in peripheral paralysis—that to faradic electricity especially early, while the galvanic current still acts, and may even cause an exaggerated reaction (reaction of degeneration). Occasionally even on the

fifth day after the onset of the paralysis (and more frequently after one week) some of the muscles contract but feebly, others not at all to the faradic current. This is always a bad sign, for when the muscles cease to react some weeks after the onset of the disease, they usually remain incapable of reaction during the whole life. The further the degeneration of the muscles proceeds, the weaker does the reaction to the galvanic current become, until finally it also entirely disappears.[1] The plantar reflex (on tickling the soles) is usually absent, as also the patellar reflex (knee-phenomenon). Still we must remember that even in healthy children the latter is more difficult to obtain, on account of their struggling, and especially stretching out their legs, and therefore it more frequently fails us than in adults.[2]

In addition to the atrophy of the muscles, an arrest of growth in the bones is also observed, so that the limb appears shorter than the healthy one. This arrest of development of the bones, as Duchenne and Volkmann have pointed out, does not always proceed *pari passu* with the degree and extent of the paralysis and of the muscular atrophy. The latter may be very well marked, and yet the limbs scarcely appear shortened; while in some cases, where paralysis and atrophy are only very limited, the growth of the bone may be arrested to a considerable extent. This fact, according to Charcot, is in favour of the direct influence of the central disease on the nutrition of the osseous system.

If the paralysis is not recovered from within ten or twelve months from its commencement, there is generally but little hope of any recovery taking place at all. About this time a new series of symptoms usually develops. As the paralysis and atrophy do not affect all the muscles of a limb equally, but are

[1] *Cf.* on this subject, Seeligmüller, Gerhardt's *Handb. d. Kinderkrankh.*, Bd. v., Abth. 1, 2. Hälfte, S. 68.

[2] Eulenburg (*Deutsche Zeitschrift f. prakt. Med.*, 1878, No. 31; and *Neurol. Centralbl.*, No. 8, 1882), in 124 children between 1 and 5 years of age found the knee-phenomenon absent on both sides in 5·65 per cent., and on one side in 2·42 per cent. *Vide* also Haase, *Beitr. zur Statistik der Reflexe bei Kindern, Diss.:* Greifswald, 1882.—Bloch, (*Arch. f. Psychiatrie u. Nervenkrankh.*, xii., 1882) and Faragó, *Arch. f. Kinderheilk.*, viii., S. 385). Pelizaeus (*Archiv f. Psychiatrie*, xiv., H. 2) found only 1 out of 2,403 children in whom he was never able to obtain a patellar reflex, while Zeising (*Ueber das Kniephänomen u.s.w., Diss.:* Halle, 1887) failed entirely to find it in only 1·4 per cent. of his cases, although it was often indistinct or much diminished (altogether in about 11 per cent.). We are evidently not yet in a position to speak dogmatically on this matter.

almost always confined to single muscles or groups of muscles, deformities are produced by contraction of the opposing muscles which have not lost their tone and contractility. In the great majority this takes the form of pes equinus, but we may also have pes varus, club-hand, and other abnormal postures of the upper and lower extremities. This explanation of the deformities as due to the contraction of the antagonistic muscles, was generally accepted until very recently, and still has many supporters. Hüter and Volkmann were the first to try to replace it by a mechanical explanation, according to which the deformities are supposed to be due to the position and weight of the limbs; while others (Hitzig) take into account in their explanation the contraction of the connective tissue of the muscles whose nutrition has been interfered with. However this may be, when the deformities commence the disease may be regarded as having reached its last stage; and we have then only to do with a crippling which the patient will have to carry with him to the very end of his life.

The anatomical researches (to which the first impulse was given by Cornil, Laborde, and Charcot in the Salpetrière in Paris in 1863-4) prove that the former views of the nature of the disease—that it was an "essential" affection, or a disease of the peripheral nerves or of the muscles—were incorrect. They have entirely confirmed the supposition of those physicians (Heine) who regarded the spinal cord as the real seat of origin. Almost all the anatomical observations, indeed, were made on the later stages of the disease, generally even on adults and old people, who had carried the infantile paralysis into old age. All the observations, however, prove this fact beyond a doubt, that we have to do with an inflammatory process of the grey substance of the anterior horns of the spinal cord, which may extend into the antero-lateral column. Slight changes in the posterior horns have also been found in exceptional cases. We find patches of myelitis either in the upper or lower part of the cord, according to the position of the paralysis, especially in the cervical and lumbar enlargements. In comparatively recent cases—as in those described by Roger and Damaschino[1]—in which the paralysis had existed for two and six months respectively, these patches had a length of about

[1] *Gaz. méd.*, 1871.

1—1½ ctm., and a breadth, at their widest part, of 1—2 mm. They were of a soft consistence and reddish colour, and under the microscope showed an increase of the capillaries, a thickening of the walls of the blood vessels with a profuse formation of nuclei in them, and very numerous granular cells. The multipolar ganglion cells of the anterior horns, and the motor root-fibres passing from them were atrophic; and slight sclerosis of the white anterior and lateral columns was to be found. Roth's case,[1] which had lasted several months, was a quite similar one; but in it the patch implicated on the right side not only the antero-lateral column, but also the posterior column. A case recently published by Archambault and Damaschino[2] is of especial importance, because the post-mortem took place on the 26th day after the commencement of the disease.

> Paralysis of the left leg. Sensibility normal; all reflexes absent. Paresis of the right arm; paralysis of the neck; faradic reaction entirely absent. Death from measles and broncho-pneumonia. *P.-M.*—in the grey anterior horns of the cervical and lumbar regions there were several very small patches of softening; vessels over-distended with blood; numerous granular cells; the ganglion cells very atrophic. In the anterior nerve-roots and at their point of origin in the grey anterior horns and white anterior columns, the medullary sheath and the axis-cylinder were wanting. The nerve sheathes were partly empty and partly contained medullary substance which stained black with osmic acid, exactly as in nerves which have been divided.

The older the trouble is, the more prominent is the appearance on which Charcot laid especial weight, namely, the atrophy of the multipolar ganglion cells, combined with sclerosis of the grey anterior horns and atrophy of the motor root-fibres passing out of them. In old cases, especially when the post-mortem is not made till an advanced age, we may have a diffuse atrophy of the anterior horns and of the white substance of the antero-lateral columns, with disappearance of the large ganglion cells and development of numerous corpora amylacea (Charcot, Leyden[3]), even an arrest of development and atrophy

[1] Virchow's *Archiv*, 1873, Bd. 58, S. 263. *Vide* also F. Schultze, *Neurol. Centralbl.*, i., No. 19.

[2] *Revue mens. des maladies de l'enfance:* Févr., 1883.

[3] *Klinik der Rückenmarkskrankh.:* Berlin, 1875.

of the motor area of the cortex on the side of the brain opposite to the paralysis.[1]

As regards the muscular atrophy which plays so important a part in this disease—a large part of the primitive bundles seem simply to atrophy in the earlier stages without undergoing fatty degeneration (Damaschino, Volkmann and Steudener). The accumulation of fat in the sheaths of sarcolemma begins at a later period, filling the place of the primitive bundles which have disappeared; and at the same time also, in the interstices between them—sometimes to such an extent that the atrophy of the muscles is concealed by it, and their volumn appears normal or even increased (Laborde, Charcot). This formation of fat is, however, by no means invariable; it may be present in some muscles and almost completely absent in others, in which case the interstitial connective tissue appears more or less hypertrophied. The appearance of the muscles to the naked eye varies according to these differences. They are either thin and pale-reddish or yellowish; or else bulky, and in that case they seem to be almost entirely converted into fat. When there is general emaciation, moreover, this fat also disappears and the atrophy of the muscles is then all the more distinct. The nerve-roots and nerve-trunks, also, have not uncommonly been found atrophied in the paralysed parts, and they then appeared attenuated and grey; while in other cases the thickening of sheaths and the increase of interstitial connective tissue and fat concealed the atrophy.[2]

The appearances being such as I have described, there can no longer be any doubt that spinal infantile paralysis is to be attributed to a myelitic process occurring in patches, which is most apt to affect the grey substance of the cervical horns, especially the cervical and lumbar enlargement.[3] In course of time the process may, as already remarked, spread to the antero-lateral columns, and may indeed occur in a diffuse form both above and below; and in a few cases an affection of the grey substance of the posterior horn has even been observed—which explains the

[1] Rumpf, *Arch. f. Pychiatrie*, Bd. xvi., Heft 2.—Sander, *Œuvres compl. de Charcot:* T. iv., Paris, 1887, p. 38.

[2] *Cf.* on the changes in the muscles and nerves, Eisenlohr, *Deutsches Archiv f. klin. Med.*, Bd. xxvi., S. 543.

[3] Kussmaul therefore proposed to name the disease "Poliomyelitis acuta anterior."

fact that occasionally disturbances of the sensory functions (anæsthesia, pains) have been observed—but this is always an exceptional occurrence. I have myself met with one case of this kind, in which the greater part of the paralysed leg showed loss of sensibility; while in another child of two years (18 July 1879) the disease had begun three weeks before, with four days of fever and severe pain in the left arm. The arm on the fifth day was quite paralysed, but still retained sensibility. These sensory derangements—especially as occurring in the first stage of the disease—have been already mentioned by Duchenne, Kennedy, Vulpian and others; but very little attention has been paid to them, owing to the fact that they are very difficult to make out, especially in children who are too young to speak. The implication of the sphincters of the bladder and bowel has only been observed in exceptional cases. I have also repeatedly seen the muscles of the neck affected. Thus in a child of three, after a febrile initial stage lasting two days, there suddenly appeared paralysis of the right upper extremity, and of the cervical muscles on the right side. The head could no longer be held upright, but rolled about in all directions, and when the child was lying down he could only move it to the left side. This paralysis disappeared after a week, while that of the arm continued and was soon accompanied by atrophy of the deltoid and shoulder muscles and by diminution of temperature.

All authors agree in saying that the brain is not affected. Leyden[2] expressly says that the facial and hypoglossal nerves and the eye-muscles have never been found implicated, and that he has only in one case found a small sclerotic patch in the medulla oblongata, which had caused no symptoms during life. These facts seem to me to make the following case all the more important.

> Bertha M., 2½ years old, brought to my polyclinic on 1st May, 1876. Three weeks before, she had sudden fever with vomiting and persistent drowsiness. These symptoms continued 2 days. On the second day weakness of the right hand was already noticeable, and on the third paralysis of the whole right arm. Drowsiness continued for 3 days after this. The child then seemed well,

---

[1] Laurent, "Symptômes prémonitoires de la paralysie spinale aiguë," *Thèse de Paris*, 1887.

[2] *Loc. cit.*, ii., S. 555.

but there was paralysis of the right arm and of a portion of the left facial nerve. The latter had not quite disappeared when I examined the child. The left eye still remained half-open when she screamed or cried, and the mouth was somewhat drawn to the right side. The right arm hung down flaccid, the upper arm was quite immovable, the forearm movable at the elbow joint; the adduction of the thumb was the only movement possible in the hand. The muscles on the left side of the face gave the normal reaction to the faradic current, while in the right upper extremity only the flexor and adductor pollicis and some fingers were moved. All the other muscles gave a very weak reaction or none at all. The galvanic current was not tried for want of the apparatus. Sensibility, bulk and temperature normal. From May to the end of October the faradic current was applied almost daily, and finally brought about a marked improvement. The flexion of the elbow and wrist joints, the movement of the thumb and of the 4th and 5th fingers almost normal. On the other hand the arm could not be moved outwards or backwards. The deltoid and muscles of the shoulder much wasted, and the whole right extremity colder than the left. The 2nd and 3rd fingers stiffly flexed and could be voluntarily extended. The facial nerve had recovered its functional activity completely by the middle of May, without electric treatment. I did not see the child again till 28th April, 1879. At that time she had been treated with electricity for nearly a year, and had made considerable progress, so that the arm could now be moved backwards and outwards. The atrophy was still unchanged, and the right hand markedly smaller than the left.

The characters of spinal infantile paralysis are in this case very well-marked, and the implication of the facial nerve forms, therefore, an exceptional feature not hitherto described. I must assume that in this case at first, simultaneously with the patch of myelitis which appeared in the right anterior horn of the cervical enlargement, a very limited patch of encephalitis had developed in the neighbourhood of the nucleus of the left facial nerve. The latter after a few weeks underwent complete resolution, while the myelitic process persisted longer and led to partial atrophy of the ganglion cells. When one remembers that other spinal affections—for example, multiple sclerosis—are not at all uncommonly combined with analogous changes in the brain, one cannot really see why the same should not occur in infantile spinal paralysis. The occurrence of coma and convulsions in many cases with a febrile initial stage, is in fact in favour of the view that the brain may be

more often affected in this disease than we are wont to suppose.[1]

The symptoms of spinal infantile paralysis are so well-marked and characteristic, that it is scarcely impossible, if one exercises any care at all, to confound it with any other form of cerebral or spinal paralysis. The febrile initial stage, the sudden onset of the paralysis (which is almost never progressive, but always retrogressive, and from being widely extended at first rapidly diminishes till it is confined to a more limited area), the almost invariable immunity of the sensory functions and of the sphincters, the rapid disappearance of the reaction of the muscles to the faradic current, the early atrophy and fall of temperature, and, finally, the deformity—all these are found thus combined in no other disease. I therefore consider it superfluous to discuss here, one by one, the diseases which might possibly be mistaken for it. The question, however, arises—whether all the cases which present the clinical characters of spinal infantile paralysis are really caused by these disseminated patches of myelitis as they have formerly been described to be. In fact, it cannot be denied that peripheral paralysis of single limbs—of one arm, or of one lower extremity—may resemble perfectly in its clinical characters the central affection which we are considering. From the effect of injuries, especially from over-stretching or compression of a nerve-trunk (p. 245), and dislocation of the shoulder-joint, paralysis may arise and be accompanied after a short time by atrophy of the muscles and loss of their reaction to faradic electricity, just as in certain cases of peripheral paralysis of the facial nerve. Duchenne has already pointed out this congenital dislocation of the humerus as an affection similar in its symptoms to infantile paralysis. One thing however is wanting in all these cases of paralysis, namely, the febrile premonitory stage, sometimes accompanied by cerebral symptoms. Many years ago Kennedy described cases of paralysis which arose quite suddenly without any warning in perfectly healthy children. In some of the cases the children went to bed well and wakened in the morning with paralysis of a lower and upper

[1] Seeligmüller gives a case not unlike my own (*Jahrb. f. Kinderheilk.* xii., 1878, S. 348). Eisenlohr gives another (*Arch. f. Psychiatrie und Nervenkrankh.*, Bd. ix. and x), which was not really a case of spinal but of "bulbar" paralysis; and in it atrophy of the ganglion cells of the left anterior facial nucleus was discovered.

extremity, which as a rule again disappeared after a varying period (the so-called temporary paralysis), but might also take the same course as spinal infantile paralysis. In such cases, one looks for local causes, without, however, always finding them; and in that case we either assume that the head has pressed on the nerves of the arm during sleep, or that there has been a chill, or reflex irritation from teething—though the assumption has generally not much to go upon. The teething, at any rate, which is blamed by English writers, I have not been able in one single case to make sure of as the cause of such paralysis. At any rate these cases of paralysis which Kennedy has described are very various in their origin, and a small proportion of them seems really to belong to the class of spinal infantile paralysis.

Uncertainty in the diagnosis can only arise when we have a paralysis of one limb along with atrophy of the muscles and loss of their reaction to electricity. For when the paralysis is extensive there can be no doubt that it is due to myelitis.

The only disease which can possibly be mistaken for this is the "atrophic cerebral paralysis" which I shall describe presently. But in the latter we are generally guided in the diagnosis by the implication of cranial nerves, mental derangement, and the condition as regards electrical reaction.

I think I ought to mention that cases of simple atrophy of one or other extremity, occasionally occur with somewhat lowered temperature, at the first glance reminding one of spinal infantile paralysis, but in which the muscular strength is little if at all impaired, and the electric reaction is normal—where, therefore, there is no paralysis whatever. Such cases of atrophy may depend on a defect of primary formation; as, for example, in a girl of 7 years, always healthy, but left-handed, whose right hand, left thigh and leg had always been to a certain extent atrophied, without the strength having suffered, and without nervous symptoms ever having been observed at any time. In such cases all the tissues—bones, muscles, and fat—in the affected extremities show a weaker development than the corresponding normal limb. In another case—that of a child of 7 months—the atrophy of the left leg and foot was the result of the umbilical cord having been twisted round it in a spiral manner. Here also neither the motility nor the electro-muscular contractility had in any way suffered. In some cases of this

kind the mothers had not noticed the atrophy at all, and it was first discovered accidentally in the hospital.

We know practically nothing about the causes of spinal infantile paralysis. The disease sets in as a rule quite suddenly, and in the midst of perfect health, and even in spite of the most careful investigation we hardly ever succeed in finding anything which could have occasioned it. In one of my cases a fall into water was given as the cause. Occasionally we observe the symptoms of spinal paralysis after infectious diseases, for example, after scarlet fever, measles, smallpox, typhoid, or pneumonia. In most of these cases recovery takes place; still, atrophy may appear during the further progress of the case, and it must for the present remain undecided whether the pathology of these cases is quite the same as that of infantile spinal paralysis. I may simply mention in passing that the latter, though much rarer, yet may occur in adults and present all its usual symptoms.

In most cases the physician is not called in until the disease has already lasted some weeks. If you are summoned in the acute premonitory stage, you never know, of course, whether spinal paralysis is about to develope, because you find nothing but more or less high fever, with or without cerebral symptoms. If the latter are present we should apply an ice-bag to the head, in very severe cases a few leeches behind the ears, or to the temples, and order purgatives (calomel, gr. $\frac{1}{2}$—$\frac{3}{4}$ every three hours, or mist. sennae co. &c.). When, however, the paralysis has declared itself, I no longer expect any result from internal treatment. Experience teaches that nothing can favour recovery from the paralysis and prevent atrophy, except electrical treatment begun as early as possible. Although some, *e.g.* Heine and Volkmann, maintain that electricity is not of very much use, or that all hope is to be given up if it produces no result within a year, this view conflicts with the great success which Duchenne and others have had, who have succeeded, even after the expiry of a year, in obtaining results by persistent treatment; and the case given above (p. 255) is another proof of the same fact. We can, therefore, only give the advice to persevere; but this is just the very point where many parents fail, and even many physicians also. We may begin the electrical treatment a very few weeks after the onset of the disease. The

galvanic current is recommended, very properly, for this early stage, because the faradic is too irritating and painful for children, and, besides, the reaction to it may already be much diminished, or even altogether wanting, while the galvanic current has still a distinct action. According to the rich experience of Duchenne—who, however, only used the faradic current—the treatment at the commencement must be very cautious. It must begin with a weak current, be applied only thrice a week, and continued each time for not longer than five, or at most ten, minutes. In the later stage the faradic suits as well, perhaps even better, than the constant current; for it is then our object to excite the muscular fibres which have not yet degenerated by a powerful stimulus, and to favour their nutrition. I repeat, that the treatment in obstinate cases must be persevered in for years before the case is given up for hopeless. Along with electricity, massage and gymnastics are to be recommended; and these, when properly used, by occasioning regular exercise of the muscles which are not yet completely incapable of contraction, have the power of strengthening their function, as well as of favouring their nutrition. During the later stages, we have to avail ourselves of orthopædic surgery, in the form of apparatus and operations (tenotomy). We may thereby endeavour on the one hand to prevent deformities and support the atrophied muscles, and on the other to remove the contractures of the opposing muscles. It is the old cases of infantile paralysis that furnish a large proportion of the material in the orthopædic institutes, and Heine's celebrated work[1] which has done so much to introduce sound views on the subject of infantile spinal paralysis, is itself the outcome of his orthopædic observations. The manufacture of such apparatus, as well as the form of gymnastics to be employed, must be suited to each individual case, and in most cases the physician should get advice and assistance from an experienced orthopædic surgeon and a clever instrument maker. Among the lower classes I have on several occasions found intelligent fathers, who of their own accord had constructed apparatus which in spite of its simplicity and cheapness answered the purpose pretty well.

Although the recovery of the electrical reaction is always an

[1] *Spinale Kinderlähmung, Monographie.*, 2 Aufl. . Stuttgart, 1860.

extremely good sign, still, experience shows that this reaction (to both kinds of current) may still be absent when the first traces of voluntary movement begin to make their appearance, and we must then continue the application of electricity all the more steadily. Other methods of treatment I cannot recommend to you. I have no faith in the use of iodide of potash, either at the beginning or later on, and the injections of strychnia (gr. $\frac{1}{32}$—$\frac{1}{21}$ gr. daily) which are occasionally recommended, have so far—in my hands at least—had no effect. What can, however, be recommended—where circumstances allow of it—is to send such children during the finest part of the year into the fresh mountain or forest air, and to order brine or chalybeate baths, which by the large amount of carbonic acid which they contain, have a stimulating influence on the cutaneous sensory nerves, and in this way act reflexly on the motor functions, if there should be any normal muscular tissue left. But neither Rehme and Nauheim, nor Schwalbach, Pyrmont, and Driburg, nor, finally, the equally famous indifferent thermal waters (Gastein, Wildbad, Ragaz, and others) will do any good whatever, apart from their action on the general health, after the case is old, the ganglion cells already atrophied, and the muscles in a state of contracture and fatty degeneration. Under these circumstances nothing is any longer of use, and the patients spend the rest of their lives as cripples.

Spinal infantile paralysis is the only disease of the spinal cord which is especially liable to affect children, and in doing so presents certain characteristic symptoms. The only other spinal disease which plays an important part on account of its frequency in childhood is the paraplegia resulting from disease of the vertebræ. But it differs in no way from the same condition in adults. There is the less need for discussing it here, as the vertebral disease which occasions it is fully considered in all surgical works, and also because the treatment almost entirely devolves upon the surgeon. It is certain that in childhood many other diseases of the spinal cord do occur which occasion paralysis, such as inflammatory processes, hæmorrhages, tubercle, even tumours of different kinds; although they are much less common than in adults. These conditions do not present anything peculiar or characteristic in children. Their symptoms are the same, and their special diagnosis is in most cases just as

difficult—in fact as impossible—as in later life. There are two diseases in particular, the occurrence of which in childhood has within recent times excited considerable interest—sclerosis and the so-called "spastic spinal paralysis." The former has been verified post-mortem in children, although but rarely; and we are indebted especially to Friedreich for our knowledge of a condition of sclerosis of the posterior columns throughout their entire length, with the occasional implication of the lateral and anterior columns. This condition developes hereditarily, especially about the time of puberty, is distinguished clinically by ataxic movements of the lower extremities to begin with, later also by interference with speech, paralyses of the eye-muscles, nystagmus and loss of the reflexes, and has an extremely protracted course, lasting as long as 30 years. Spastic spinal paralysis, as is well-known in adults also, is little more than a group of symptoms corresponding to no quite definite pathological change. Such cases—which are characterised by a chronic paresis of both lower limbs (rarely of the upper), existing even from the first year of life, and especially by contracture of individual groups of muscles—I have frequently met with in children. In these cases especially on trying to stand or walk, the attempt to plant the foot on the ground at once produced trembling and a rigid contracture of the calf-muscles, with the feet in the posture of pes equinus, and from the stiffness of its legs the child could only walk with much labour on the fore part of its feet, which were somewhat inverted—and even then only if supported or led. In many cases, moreover, there was such a contraction of the adductors of the thighs that they were almost crossed over one another, so that all locomotion was rendered impossible. This contracture also persisted when the child was at rest, and prevented active as well as passive separation of the thighs. The patellar tendon-reflex was generally exaggerated, the electro-muscular contractility, the sensibility, and the power of the sphincters not lessened, and no atrophy was noticeable.[1] Unfortunately all these cases passed from under my observation, and remained anatomically uncompleted. The numerous cases of

[1] Seeligmüller (Gerhardt's *Handb. d. Kinderkrankh.*, v., Abth. 1. 2. Hälfte, S. 167) has observed 5 cases combined with atrophy of the muscles and symptoms of bulbar paralysis ("amyotrophic spinal paralysis"), but in all of them pathological confirmation of the diagnosis was wanting.

this kind published by Seeligmüller,[1] Förster,[2] Maydl,[3] and d'Heilly,[4] have succeeded just as little in throwing light on this obscure subject. These writers, and also I myself have observed (though by no means constantly) a complication of the paralysis with deficient mental development—even idiocy,—stuttering or stammering, and spasmodic distortion of the face; and this leads to the conclusion that the brain may participate, or that it may even be the point of origin of such a series of symptoms. In fact I shall presently have occasion to give you an example in which very considerable alterations of structure were found in the cerebral cortex at the post-mortem. I need scarcely remind you that under these circumstances a secondary degeneration of the fibres which arise in the diseased portion of the brain may spread to the spinal cord, and can be demonstrated microscopically. It is, moreover, possible in a certain proportion of these obscure cases by means of tenotomy and orthopædic surgery, to bring about a certain degree of improvement in the walking, although not recovery.

X. *Pseudo-hypertrophic Muscular Paralysis.*

This disease, first mentioned by Duchenne,[5] but first described accurately from an anatomical point of view by Griesinger,[6] invariably originates during childhood, but may be prolonged into youth or adult age. When the disease is well developed, the symptoms are very characteristic. The muscles of the calves, buttocks and thighs—especially the first—are of unusual bulk, and frequently also of a remarkably hard consistence. Those of the chest, arms and shoulders are wasted and flabby, but not throughout their whole extent; for on closer examination we also find nodular thickenings here and

[1] *Deutsche med. Wochenschr.*, 1876, Nos. 16 and 17.—*Jahrb. f. Kinderheilk.*, xii., 1878.

[2] *Jahrb. f. Kinderheilk.*, xv., S. 261.

[3] Rupprecht, "Ueber angeborene Gliederstarre und spastische Contractur," Volkmann's *Sammlung klin. Vorträge*, 198.—Maydl, *Einige Fälle von spastischer cerebrospinaler Paralyse bei Kindern:* Wien, 1882.

[4] d'Heilly, "Revue mens. des maladies de l'enfance," Déc., 1884.—Naef, *Die spast. Spinalparalyse im Kindesalter.*; Zürich, 1885.

[5] *Electrisation localisée*, 2. édit., p. 353, and *Arch. gén.*, Janv.—Mai, 1868.

[6] *Arch. d. Heilkunde.*, 1865, vi., S. 1.

there in the deltoid, biceps, and triceps brachii. The recti abdominis and the lumbar and dorsal muscles, also, are often thickened, though not to the same degree as those of the lower limbs. In a few cases—*e.g.* in one observed by Bergeron—all the muscles with the exception of the pectorals and sterno-mastoids were hypertrophied, so that the child looked like an athlete. The patients' gait is very peculiar. They walk with their legs apart, waddling, and only touch the ground with the fore part of the foot, which is in the posture of pes equinus. At the same time the natural lordosis of the lumbar vertebræ is much exaggerated (forming a concavity like a saddle) owing to the weakness of the erectores spinæ.

If you make the patient lie down on the ground and get up again, you notice that he "climbs up his own legs," as the phrase goes; that is to say, he first brings himself into a position which enables him to use his hands as a lever to raise himself with, and finally manages to do this by placing his hands firmly on the ground, then supporting them on the thighs, in this way raising up the upper part of his body. In the latest stage in which the weakness of the upper extremities reaches an extreme degree, this mode of raising himself becomes, on that account, no longer possible. I have hitherto had the opportunity of observing this rare disease only in six cases, and in every one of them there was this peculiar method of rising up. All the patient's movements are in general clumsy, awkward and laborious, and they become weaker as the disease progresses. The electro-muscular excitability increases steadily with the progress of the disease. At the same time the adipose tissue, especially in the lower limbs, may be well preserved, but when marasmus finally sets in it disappears. The atrophied muscles in the upper part of the body often present fibrillary twitchings similar to those in progressive muscular atrophy in adults. The skin of the lower extremities not uncommonly presents a marbled appearance, owing to venous engorgement, and a lowered temperature, but an increased secretion of sweat. Many of these patients are mentally weak and their speech is slow, and in rare cases an increase in the bulk of the tongue is said to have been observed.

The development of this disease always dates, as I have already remarked, from the middle period of childhood, and it has been expressly stated by some that they have noticed the

slowness of the children's movements when they were even younger. We see most of the patients for the first time in the more advanced stage when they are 7—10 years old, and often much older. The diagnosis does not become certain until the bulk of the calf muscles has become distinctly increased. In the earlier stage, when this is still absent and we notice nothing but the peculiar gait and the above-mentioned characteristic method of rising up from the ground, we can only suspect the presence of the disease. Still, in very recent times, the diagnosis has been established even at this early stage by the examination of a fragment of muscle.[1] The general health may remain unimpaired. The case observed by Demme of a boy of 10 years with a slow pulse (44—60), and a considerable amount of sugar in the urine (which however was not always present) stands alone as yet.[2] If the general health remains unimpaired, the disease may last 10—20 years, in the course of which time it often becomes arrested, but no real process of recovery takes place. If the patients do not die from a chance complication, they generally succumb in the end to the increasing atrophy and weakness of the respiratory muscles, or to marasmus.

The pathological process in the muscles is very similar to that with which we are acquainted in spinal infantile paralysis, and in progressive muscular atrophy. We have essentially a diminution in bulk of the muscular fibres, which in the apparently hypertrophied parts (calves and thighs) is replaced by a deposit of interstitial fat, and by connective tissue (atrophia musculorum adiposa). This compensation may also occur locally in the atrophied muscles in the upper part of the body (deltoid, &c.) in the form of isolated nodules; and there are also a few hypertrophied primitive bundles between them. In what manner this atrophy is caused—whether by the primary formation of connective tissue between the bundles, as Charcot and Duchenne consider probable (paralysie myosclérosique), or in other ways—cannot as yet be determined. Also the changes in the spinal cord occasionally described (the presence of a copious finely-granular substance and many corpora amylacea, especially in the lateral columns and disappearance of a large number of the

[1] Bourdel, "Revue mens. des malad. de l'enfance," Févr., 1885, p. 54.
[2] 15. *Jahresber. d. Berner Kinderspitals*, 1877.

large ganglion cells in the anterior horns) are by no means to be regarded as constant or essential. We discover nothing else morbid on examining the peripheral nerves and the sympathetic, although even here neuritic changes have occasionally been observed. It is only owing to the interference with movement that I have decided to place this affection along with the nervous diseases; for from a purely anatomical point of view it is to be regarded as a primary affection of the muscles.[1] I agree with those writers (Seidel, Erb[2]) who regard this disease as really an infantile or juvenile progressive muscular atrophy, which differs from the form observed in adults in that it does not as in them begin first in the interosseous muscles of the hand and in the muscles of the thumb, but in those of the back and lower extremities, sometimes even in those of the face.[3]

The progressive atrophy of the muscular fibres which finally renders many of the sarcolemma-sheaths quite empty, corresponds to the diminution of the electric reaction, which is equally noticeable in the wasted and in the thickened muscles. On the other hand the skin reflexes and sensibility remain the same. Indeed Steidel and Wagner made out a prolongation of the sensation of touch as compared with the normal condition.

It is worthy of note that with few exceptions (*e.g.* the cases of two young women between 20 and 30 described by Lutz[4]) all the patients have been boys. Occasionally there have been several children in one family. Apart from this inexplicable (hereditary?) predisposition, all the other causes which have been suggested (unfavourable circumstances, scrofulous or rachitic cachexia) are open to doubt. I have unfortunately nothing favourable to tell you about the results of treatment. The administration of medicine has just as little effect as the compression of the calves by bandages recommended by Griesinger, which may at most interfere with the compensatory formation of fat, but can scarcely be supposed to have any effect on the muscular atrophy. Electricity, especially galvanism,

[1] *Cf.* Krieger, *Deutsches Archiv f. klin. Med.*, Bd. xxii., Heft 2.

[2] Erb, *Deutsches Archiv f. klin. Med.*, Bd. xxxiv., H. 5 und 6.—Buss, *Klin. Wochensch.*, 1887, No. 4.

[3] O. Heubner, "Ein paradoxer Fall von infantiler progressiver Muskelatrophie": Leipsic, 1887.

[4] Hirsch-Virchow *Jahresbericht*, 1866, ii., S. 261; 1867, ii., S. 293.

is always worth a trial. In one case which presented all the symptoms of the commencing disease, I saw these disappear under this treatment in 5—6 months. Bourdel also reports a case of this kind.

XI. *Apoplectic Conditions.*

Cases of paralysis proceeding from the brain are observed in children far more frequently than those arising from the spinal cord; and their general characters—the hemiplegic form and the long persistence of the electric reaction in the paralysed muscle—are just the same as in adults. Atrophy of the muscles may also accompany the paralysis; but this developes very slowly, seldom attains to the high degree in which it is found in spinal infantile paralysis, and appears to proceed more from inactivity and long disuse of the muscles than from any interference with the trophic influence of the nerves. It is frequently accompanied by contractures due to excessive action of the non-paralysed muscles, or oftener to direct central irritation, and also by tremor and automatic movements.

The onset of hemiplegia takes place in many cases quite suddenly in the midst of apparently undisturbed health, and we are then disposed to look for its cause—as in adults—in a hæmorrhage into the brain or in an embolic process. Both these processes, however, are comparatively rare in childhood, and hemiplegia occurring suddenly is—in spite of its apoplectic appearance—much more frequently the expression of long-standing brain disease, especially of tuberculosis cerebri.

Let us first consider cerebral hæmorrhage as a cause of sudden hemiplegia. The rarity of its occurrence in childhood is principally to be traced to the fact that the most frequent cause of the condition in adults—namely, the fatty degeneration of the arteries of the brain and the formation of small aneurisms in them—is extremely rare in children. Some of the cases described as "hæmorrhage" seem to me rather to be cases of encephalic deposits with a considerable admixture of blood. In this manner, I believe, we must explain the following case:—

Oscar Z., 3 years old, unwell (?) for some days, admitted into the ward 20th February, 1882. Since the previous night almost constant convulsions, trismus, tonic and clonic spasms of the extremities, opisthotonos, cervical rigidity, convergent strabismus, complete unconsciousness and coma. P. 144; T. 101·3° F. The fits took place every 10—15 minutes, accompanied by very rapid respiration and copious sweating. Wet-and-dry cupping, vinegar enemata, an icebag to the head, and chloroform, had no effect. On 21st, the symptoms still continued; T. 101·3° F., ev. 103·1° F. P. small and irregular, 164. Death during the night.

P.-M.—Dura mater much distended, reddened within and without. All the sinuses very full. Pia mater congested. Convolutions flattened. On the parietal surface on both sides, hæmorrhagic infiltrations of various sizes in the grey substance, in the form of bluish-red and dark-red streaks and patches. The pia mater not implicated. On section the cortex was found to be almost uniformly hæmorrhagic in certain places; in others there were numerous punctiform hæmorrhages close together, some reaching the size of a pin-head. The brain-substance affected was disintegrated, soft and of a pulpy consistence. At the base there was here and there purulent infiltration of the pia mater, especially round about the chiasma and in the Sylvian fissure. The rest was normal.

In this case we really had a basilar meningitis combined with extremely rapidly progressing hæmorrhagic encephalitis.

The physicians most experienced in the diseases of children, who have had a very large amount of material at command—Guersant, Becquerel, Billard, Rilliet, and Barthez—all acknowledge that they have seen very few cases of simple cerebral hæmorrhage, understanding as such those which could be clinically recognised; for I have myself often enough met with small capillary hæmorrhages due to tuberculosis of the brain, tubercular meningitis, sinus-thrombosis, and other diseases. But since these capillary hæmorrhages reveal their presence by no symptoms whatever, they have only a pathological interest. I have hitherto had no experience of larger cerebral hæmorrhages in children confirmed by post-mortem examination. The few cases which I have published elsewhere[1] cannot be regarded as quite conclusive, seeing that they were not observed up to the end. The same holds good of the following cases, although the diagnosis is probably that of hæmorrhagic apoplexy.

[1] *Beiträge zur Kinderheilk.*, *N.F.*, S. 62.

Boy of 7 years, fell suddenly from his chair, during a meal, and was at once paralysed on the right side of his body. Later on, steady lessening of the paralysis, which I was able to follow for 10 months. The lower extremity improved more quickly and decidedly than the upper, in which the rigid contraction of the flexors of the fingers gave a claw-like appearance to the hand and rendered it almost useless. Dipping the hand in warm water removed the contractures, and the extensors then acted pretty freely. At first there was also aphasia, which so far passed off that after 10 minutes the boy could speak a few words. When the tongue was put out, it inclined distinctly towards the paralysed side. The sensibility and intelligence were completely normal, likewise the organs of circulation, as far as could be ascertained by examination.

In the case of a child of 1½ years the course was quite similar. Suddenly on a hot summer day, while in perfect health, he became unconscious while lying in his perambulator, and at once showed right hemiplegia of the body and face. In course of time, after about a year and a half, power of movement was almost quite restored to the leg by electric treatment, while the arm still showed partial paresis. The facial nerve recovered soon after the attack. There were never any symptoms of irritation in the paralysed parts, and the general health was always quite good.

In these cases the cause of the disease remained unknown; but in a child of 3 years who suffered from very severe whooping-cough, I saw convulsions and coma occur after a particularly violent attack. These lasted for 9 hours, and left behind them hemiplegia of the left side. This continued several weeks, the arm and leg being flabby and quite incapable of movement. The face was unaffected. Other writers also[1] have published similar cases which ended favourably, and considering the hæmorrhages which so often occur from whooping cough, in the connective tissue of the eyelids and conjunctivæ, from the nose, and even from the ears—we may assume almost with certainty that this was really a case of cerebral hæmorrhage.

In the following case I believe that we must assume a hæmorrhage in the brain as the result of an injury:—

Boy of 4 years. On 7th August, 1879, he fell from a bridge about twelve feet high, on to the railway lines. Loss of consciousness, and hæmorrhage from the mouth and nose. After he was

[1] Finlayson, *Jahrb. f. Kinderheilk.*, x. 400., *Oesterr. Zeitschr.*, 1876, ii., S. 138. —Casin (*Gaz. des hôp.*, 37, 1881) found under similar circumstances 6½ oz. of fluid blood between the bones and dura mater over the left occipital fossa (cephalhæmatoma internum).

taken home, frequent vomiting of matter mixed with blood. On the 8th, admitted into the ward; then quite conscious. Ecchymosis behind the right ear. Incomplete ptosis on the right side. Marked dilation and sluggishness of the right pupil, and paresis of the right arm. Pulse somewhat irregular, 80—92. Temperature 98·1° F. Steady improvement from the fifth day. Ptosis and paresis of the arm disappeared after 8 days. Difference of the pupils still noticeable on 24th. On this date he left his bed, and dragging of the right leg was noticed. On 31st he was discharged quite cured. Treatment: ice-bag to the head, repeated doses of castor oil.

In a few cases of purpura hæmorrhagica also, apoplexy has been known to occur in children. Mauthner publishes a case of this kind with a post-mortem. I have only one case recorded, which, however, was not fully confirmed as there was no post-mortem.

Child of 7 years. Scarlet fever 4 years before, followed by dropsy. During the last year, purpura with repeated hæmorrhages from the mouth, nose, ears, eyes, bowel and kidneys. At the same time great weakness and loss of appetite. No enlargement of the spleen. After treatment for 9 days, sudden violent convulsions and coma. Soon after, left hemiplegia with paralysis of the facial. Death in the evening. Post-mortem not permitted.

Whether the extravasation—which in this case cannot of course be doubted—occurred in the brain matter itself, as in Mauthner's case, or between the membranes, must remain undecided. That the latter may happen we learn from an English case[1] in which effusion of blood was found between the dura mater and the arachnoid in a boy with purpura, who died in a state of coma.

In the following case, also, in which aphasia was the only symptom, I think we must certainly assume the presence of a limited cerebral hæmorrhage :—

On May 29th, 1878, I was consulted in the neighbourhood of Berlin, about a boy of 3 years who had suffered for 10 weeks—including an interval of 3 weeks—from intermittent fever. The last attack of intermittent fever had occurred a fortnight before, just one day after the boy had suffered a concussion of the brain from a fall on the head. His relatives being unwilling to defer a projected journey into the country, the boy had to travel

[1] *Journ. f. Kinderkrankh.*, iv., S. 318.

during the hot stage and was seized in the railway carriage with eclamptic convulsions, which continued almost without intermission for 7 hours. When he awaked from the coma, a marked interference with speech was at once noticed, which passed after 24 hours into complete aphasia. At first there was also headache and increased temperature of the head, which, however, soon disappeared after iced-compresses, and the use of calomel. With the exception of the aphasia, the child was quite well; no paralytic symptoms were ever noticed. On the day of my visit the boy had pronounced the word "auf" for the first time, but was still unable to give any answers to my questions, although he was quite sensible and intelligent, and he could only indicate what he meant by signs. The comforting assurance of rapid recovery which I gave the parents was speedily confirmed. After a very few weeks the power of speech gradually returned, and recovery was complete in a fortnight.

If we consider the concurrence in this case of various circumstances favouring hyperæmia of the brain—the previous concussion and the exciting railway journey during the hot stage of intermittent fever—we cannot but assume the occurrence of hæmorrhage in consequence of extreme hyperæmia, and its site would probably have been found in the second or third left frontal convolution. The absence of other paralysis cannot be regarded as weighing much against this supposition, for examples are not wanting in which small blood extravasations in the brain (confirmed post-mortem) only revealed their presence by quite localised paralysis—for example, of the facial nerve. We need not be surprised that the supposed cerebral hæmorrhage in this case, as in some of the others just given, manifested itself first by violent convulsive symptoms, seeing that these occur in young children much more commonly in connection with cerebral hæmorrhages than in adults. The small extravasations already mentioned, which are found in the form of clusters of red spots, or in a mass as large as a pea—especially in the tissue of the pia mater and the cortex, more rarely in other more central parts of the brain—often give no evidence during life of their existence except convulsions, which are not sufficient for a certain diagnosis. This is true not only of the capillary hæmorrhages of the brain and pia mater observed in asphyxiated new-born children and in the first weeks of life, but also of those which we frequently find in older children in the capillary form, or in the form of spots, as a result of severe con-

stitutional diseases (typhus, diphtheria, scarlet fever, &c.), or localised brain diseases (especially tuberculosis of the brain and tubercular meningitis). All of these hæmorrhages cannot be diagnosed, because their symptoms cannot be separated from those of general diseases, and there may often be no symptoms at all. In tubercular meningitis, particularly, I have frequently found considerable extravasations in the pia, several times also in the substance of the brain—*e.g.* in the commissures of the third ventricle—without any corresponding change in the ordinary symptoms. I therefore consider it is not worth while to linger any longer over these conditions as they have no clinical value. The rare cases of larger hæmorrhages, however, occurring in older children with sudden hemiplegia do not present either anatomically or clinically any difference worth mentioning from the apoplexy of adults. The same is true of the hæmorrhages which occasionally occur suddenly in the space between the dura mater and the arachnoid from external injuries (apoplexia meningea). At the same time I would remark that the disease described by French observers (Legendre, Rilliet and Barthez) by the name of "hæmorrhagies dans la cavité de l'arachnoide," is not now regarded amongst us as simple hæmorrhage, but as pachymeningitis, that is, as inflammation of the inner surface of the dura mater accompanied by small hæmorrhages.

In childhood, as among adults, cerebral paralysis may take place suddenly from embolic processes. Although this is far less common, still medical literature contains a number of cases in which (with the well-known symptoms) clots were carried from the left side of the heart or even from the pulmonary veins through the circulation into the carotid and its branches, especially the Sylvian artery, and occasioned a more or less extensive patch of softening in the area of brain supplied by it. Since in such cases the paralysis makes its appearance with apoplectic symptoms, owing to the sudden anæmia which takes place in the affected areas of the brain, we encounter here the same difficulties of diagnosis as in adults, and it is only possible to determine approximately whether we have to do with an embolism or a hæmorrhage, if we are able, by examining the heart, to find something that supports the diagnosis (endocarditis, valvular disease). If we find no murmur in the heart, this by no means

excludes the possibility of an embolism, for the thrombus from which the embolus has arisen may also have been situated between the trabeculæ of the left ventricle, in the left auricular appendix, or even in the pulmonary vein, and may have found its way from these into the left side of the heart and into the aorta. A case of this kind was under observation in my ward. The patient was a boy of 2½, suffering from chronic pneumonia and caseous degeneration of the bronchial glands, in whom right hemiplegia had suddenly appeared along with contracture. After death we found embolism of the left Sylvian artery, with extensive softening of the corresponding cerebral hemisphere. The source of the embolus was not the heart—which was quite normal—but one of the branches of the right pulmonary vein, which was filled with thrombi.

In another case, to which I shall return later, left hemiplegia occurred during the stage of collapse in diphtheria. The cause revealed by the port-mortem was the formation of a thrombus in the left auricular appendix, and an embolism in the Sylvian artery which had proceeded from it.

Thrombi of this kind also frequently occur at the time of death, owing to the diminished propulsive power of the heart. In a girl of 9 years with tuberculosis I found, along with buffy clots in both cavities of the heart, obstruction of one of the principal branches of the right pulmonary artery, of both vertebral arteries, and of the right Sylvian artery by embolism, without any further alteration of their tissue.

Finally, the sudden onset of hemiplegia may also be due to diseases of the brain, which either may have remained quite latent for a considerable period, or may have revealed their presence by other cerebral symptoms, especially by convulsive attacks. Among these diseases, the one we have next to consider occupies decidedly the first place.

XII. *Cerebral Tuberculosis.*

Of all chronic diseases of the brain occurring in childhood, this is undoubtedly the most frequent; indeed its frequency is so great that we will seldom go wrong if, when chronic cerebral

symptoms exist, we make the diagnosis of tubercle. Tubercle occurs in the brain, as well as in other organs, in children at a very early period of life. The assertion of Rilliet and Barthez that this disease is never observed before the third year, is to be explained by the circumstance that these authors only saw children over two years of age in their hospital. Among 14 of my cases there were 12 between 9 months and 2 years of age, and Demme has found a tubercular nodule the size of a hazel-nut in one of the cerebellar hemispheres in a child of 23 days, whose mother had tuberculosis.[1]

The diagnosis of cerebral tubercle is supported by a characteristic group of symptoms and circumstances. In the first place, the children affected are almost never quite healthy, but generally bear traces of scrofula or tuberculosis. I have repeatedly met with eczematous eruptions, ophthalmia, otorrhœa, enlarged lymphatic glands, osteomyelitis in the fingers and toes or in other bones, and—above all—caries of the petrous bone, as accompaniments of cerebral tuberculosis. Of course these morbid conditions are not always present at the moment when the cerebral symptoms commence, but it is quite sufficient that the children should have suffered from them at an earlier period, that traces are still discoverable, or even that other members of the same family have died of "lung- or gland-disease." These points in the history make the diagnosis very much easier, and thus it happens that this may often present greater difficulties in a hospital—where children concerning whom we have no history often come under treatment—than in a polyclinic or in private practice. If we inquire carefully of the relatives, we will find in almost every case that the child has not been perfectly free from "scrofulous" symptoms.

Following upon conditions of this kind, there now suddenly occurs, in many cases, an epileptic fit, which may recur after an indefinite interval. In children who are still in the period of the first dentition, or especially in those who are rachitic, it is scarcely possible to distinguish these convulsions from the comparatively harmless ones already described (p. 161). We must therefore pay particular attention to the child's condition during the intervals, which may even last for many months. Every cerebral symptom observed during these intervals is important

[1] 17. Jahresber. d. Berner Kinderspitals.

for the diagnosis. Even very young children, but more commonly older ones, often complain of headache occurring in fits like migraine, not uncommonly along with vomiting, which forces the children either to lie still or to support the head with the hands. In others a squint hitherto unobserved appears, usually in one eye, and this among poor people is often either not noticed at all or else put down to a bad habit. Suddenly, after one of the above-mentioned convulsive attacks —occasionally also without their occurrence—there occurs paralysis of a single limb, or hemiplegia, with or without implication of the facial or ocular nerves. Here, as in all central forms of paralysis of the facial, only certain branches —especially those to the lips—are wont to be paralysed. Paralysis of the third is indicated by ptosis, divergent strabismus and dilatation of the pupil; that of the sixth by an inward squint and inability to turn the eyeball outward. This paralysis may also disappear after some days or weeks, and one who is inexperienced is very apt to regard them as the remains of the epileptic attack, until there is a repetition of the symptoms, which may very possibly prove rapidly fatal.

Martha M.,[1] 2 years old, rickety and scrofulous. Repeated convulsive attacks, inability to hold the head upright, irritable temper. On 29th June, 1864, a renewal of the convulsions confined to the left side of the body, which was found to be paralysed immediately afterwards. Cranial nerves and sensibility normal. I diagnosed tuberculosis of the right hemisphere, with hyperæmia in the neighbourhood. Calomel, gr. ½ every 2 hours, and 4 leeches applied to the head. Marked improvement by 1st July; by the 8th, the paralysis has quite disappeared. On the 26th again violent convulsions on the left side, lasting three hours, followed by coma but without paralysis. On 16th October another fit, lasting 5 hours. A short fit in February, 1865, and on the 30th March a very severe one ending in coma and death.

Post-mortem: marked hyperæmia of the pia mater, especially on the left side. Small ecchymoses at some points. Some serum in the ventricles. In the posterior lobes of the right hemisphere, in the white substance, a greyish-yellow tubercle of the size of a pea, surrounded by a thin capsule of connective tissue. No tubercular meningitis. Miliary tuberculosis of the pleura, and caseous enlargement of the bronchial glands.

---

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

I would specially draw your attention in this case to the one-sidedness of the convulsions already spoken of (p. 166), which still further justifies the diagnosis of serious disease of the opposite hemisphere, when—as was the case here—it leaves behind a paralysis of the side on which the convulsions occurred. This case is also an example of the so-called "solitary tubercle"; for nowhere else in the brain was there any similar formation. You must not, however, be misled by this into the belief that it is only in the case of solitary tubercles, or when the disease is confined to one half of the brain, that unilateral convulsions and hemiplegia occur, as we certainly might expect. The following case shows, on the contrary, that tubercular disease of both hemispheres may be accompanied by hemiplegia.

Otto A., 2½ years old, admitted into my ward October 24th, 1876. A convulsive attack a year before. Four days before admission, sudden left hemiplegia with implication of the left facial nerve. During the next few days development of tubercular meningitis. Death on 30th.

Post-mortem: numerous adhesions between the dura and pia mater. Many tubercular nodules, from the size of a hazelnut to that of a walnut in the cortical substance of both hemispheres (6 in the right, 4 in the left), and an equally large one in the posterior part of the left half of the cerebellum. Tubercular meningitis.

You see that in this case it was only the tubercular masses in the right hemisphere that produced paralysis of the opposite half of the body, while those in the left, although they proved on anatomical examination to be exactly the same as on the right side, exerted no influence on the motor functions. This brings us to a weighty point in the pathology of cerebral tuberculosis—namely, its latency. As, in the case just given, tuberculosis of the left hemisphere was not revealed by any symptom during life, in like manner even more extensive cerebral tuberculosis may remain completely latent during life, and only be discovered incidentally at the post-mortem. Indeed my own experience inclines me to hold that multiple tuberculosis is far more subject to this latency than the solitary form. The following cases observed by me may serve as examples.

Boy of 4 years, with phthisis pulmonum. No cerebral symptoms ever observed. Death from rapid basilar meningitis.

P.-M.—Besides the meningitis, a tubercular mass, the size of a pigeon's egg, on the convexity of the right frontal lobe; one of similar size on the anterior surface of the right corpus striatum; finally, a mass of tubercle as large as an orange between the cerebellum and the tentorium cerebelli—soft, fissured in the inside, and slightly adherent.[1]

Child of 14 months. Caries of the right petrous bone with paralysis of the right facial nerve, and numerous enlarged glands. No cerebral symptoms ever observed. Phthisis. Death from rupture of a small apical cavity and pneumothorax. At the *P.-M.* a much fissured and softened tubercular mass, the size of a walnut, was found on the surface of the right frontal lobe; a still larger one on the surface of the occipital lobe, and a third of equal size at the periphery of the latter near the base. Also on the surface of the left hemisphere numerous large tubercular nodules with cavities filled with detritus and calcareous concretions of the size of a pea. The left lobe of the cerebellum almost entirely converted into a soft caseous mass.[2]

Child of 2 years, admitted to my ward on April 17th, 1874, with caries of the right upper and lower limbs. Anæmia and emaciation; otherwise no striking symptoms. Development of tubercular meningitis dating from April 29th. Death on 5th May. *P.-M.*—In the vermiform process of the cerebellum, extending into both its hemispheres, a tubercular mass the size of a walnut, with numerous recent tubercles in its neighbourhood. In both occipital lobes, nodules from the size of an almond to that of a hazel-nut.[3]

Child of 1 year, admitted September 28th, 1878. Hitherto always healthy, but 10 days ago took ill with repeated convulsions, followed rapidly by left-sided hemiparesis. On admission, all the symptoms of tubercular meningitis in the last stage (coma, pupils dilated and no longer reacting, pulse 160 and very small, &c.). At the same time frequent spasmodic contractions of the left side of the face, hemiparesis and rigidity of the limbs on the left side. Abdomen tense and distended. Death on 8th October with great rise of temperature (106·2° F.). *P.-M.*—The pia mater on the left side of the convexity of the brain infiltrated with caseous matter in a space as large as a sixpence just outside the median fissure. The caseous nodules extended for some millimetres into the grey substance of the cerebral cortex The rest of the brain free from tubercle. Extensive tubercular meningitis of the base and convexity with acute hydrocephalus. Likewise caseous degeneration of the bronchial glands, miliary

[1] *Beiträge, N. F.*, S. 67.
[2] *Journ. f. Kinderkrankh.*, viii., 1847, S. 166.
[3] *Charité-Annalen, Jahrg.*, iv., S. 498.

tuberculosis of the left lung, of the liver and spleen, and chronic adhesive tubercular peritonitis.[1]

A rickety child of 1 year, admitted on June 10th, 1878, with all the symptoms of tubercular meningitis. Was said to have been always healthy. Illness began 8 days previously with repeated convulsions. No paralysis, but almost continuous chorea-like movements of the right arm and leg (flexion and extension, pronation and supination, and movements in all directions). Death on 26th. *P.-M.*—Tuberculosis of the lungs and pleura, liver, spleen, kidneys, of the diaphragm and bone-marrow. Caseous degeneration of the bronchial glands, caseous masses in the left lung. Miliary tuberculosis of the basilar dura mater, tubercular meningitis, and masses of tubercle the size of a hazel-nut in the middle division of the left optic thalamus.

In these and other similar cases there was always present at the same time an advanced tuberculosis and caseation of other organs; and it has already been maintained by Rilliet and Barthez that it is just under such circumstances that cerebral tubercle is most frequently latent. I therefore still adhere to the opinion which I expressed as early as 1868,[2] that in children suffering from extensive tubercular degeneration of the lymphatic glands, lungs, abdominal organs or bones, who die with symptoms of tubercular meningitis of normal—or more frequently abnormal—course, tuberculosis of the cerebrum or cerebellum may also be assumed with sufficient probability, even should this never have revealed its existence by any definite symptoms. This probability is all the greater if the petrous bone is one of those that are carious.

The occurrence of cerebral tuberculosis with repeated epileptiform attacks and accompanying hemiplegia is, however, only one of the forms under which the disease presents itself. In another class of cases, paresis of one side gradually appears, steadily increases, and is often combined with tremor or contracture of one or both limbs. Or, the disease may begin with strabismus, localised contractures (either of the limbs or of the muscles

[1] The striking fact that in this case the paralytic and convulsive symptoms occurred on the same side on which the cortical tubercles were situated, does not require for its explanation the assumption of an incomplete crossing of the pyramidal fibres. To my mind the solitary tubercles—which were quite latent—had nothing whatever to do with these symptoms; for the latter might have occurred in the course of any tubercular meningitis, even if there had been tubercle in the brain-substance.

[2] *Beiträge, N. F.*, S. 69.

of the neck) and other cerebral symptoms—*e.g.* attacks of headache with vomiting, momentary loss of consciousness, without accompanying paralytic symptoms, aphasia and hallucinations of hearing. Not until many months, or even years, have passed—during which the condition has undergone many variations, does the fatal issue take place in the form of violent convulsions or tubercular meningitis. The following cases observed in my wards and chosen from among many others[1] will illustrate this form to you better than a detailed description.

Carl Sch., 3 years old, admitted on January 15th, 1874. Thin and pale. The disease commenced 7 months before with a tremor of the right hand. Two months later paresis of the whole right side of the body, and of the right facial nerve. Since November, 1872, almost continuous contracture of the right arm at the elbow-joint. On admission, rigid contracture of all four extremities, right-sided paralysis and tremor of the left hand. Development of tubercular meningitis. Death on 21st. *P.-M.*—A tubercular deposit the size of a walnut near the posterior surface of the right hemisphere of the cerebellum. On the convexity of the left frontal lobe, a caseous nodule $\frac{2}{3}$in. in diameter extending inwards right through the convolution to the white substance. Hydrocephalus internus. At the posterior part of the left corpus striatum 3 tubercular masses the size of a pea, close under the ependyma. Both optic thalami converted at their upper part into a nodular caseous mass.

Wilhelm J., 2 years old, admitted on April 3rd, 1875. Coughing and wasting for the last 6 months; rickety. Continuous trembling, frequently also more marked spasmodic contractions of the right arm and side of the face, the mouth being drawn upwards and to the right. No paralysis to be observed. Sensibility apparently normal. Symptoms of consolidation in the lungs. After a few days, increase of the tremor, the head and right lower limb being then also affected. The muscles of the chest and abdomen, as well as the cremaster on the right side, presented distinct spasmodic contractions recurring at short intervals. Slight paresis of the right arm. On April 6th, continuous contracture of the right thumb. On the 7th, nystagmus of the right eye. Death with high temperature and collapse. *P.-M.*—Œdema of the pia mater, especially on the convexity of the left hemisphere, and in it numerous miliary tubercles are embedded. Right in front of the fissure of Rolando, about its middle, a yellow tubercular mass of the size of a hazel-nut in the cerebral substance, which was to some degree softened in its neighbourhood. Pulmonary phthisis, &c.

[1] *Charité-Annalen, Jahrg.*, iv., 492 *et seq.*

The duration of the disease, as far as we can judge, may vary much. In some cases many months or even years may elapse, from the appearance of the first symptoms, before death occurs; while in others the first symptoms are observed a comparatively short time before death. In these cases, therefore we must assume that the disease has remained latent until reaching its last stage. I have frequently seen the first symptoms of cerebral tubercle—*e.g.* convulsive attacks with or without hemiplegia—pass almost immediately into symptoms of tubercular meningitis, which was the immediate cause of death and was as a rule marked in these cases by an unusually violent course. Other cases end in an extremely protracted and violent attack of convulsions, or death may be due to the advance of concomitant tuberculosis of other organs without meningitis.

The cases given have already illustrated to you the pathological conditions. Tubercle of the brain appears most frequently as greyish-yellow caseous nodules, ranging from the size of a pea to that of a hazel-nut, usually globular, but sometimes also uneven in shape, most frequently situated in the grey substance of the brain, in the cortex, the great ganglia, the pons Varolii and the cerebellum, but is by no means unknown in the white substance, corpora quadrigemina, crura cerebri, &c. The tubercles of the cortex which lie immediately under the arachnoid and pia mater can scarcely be distinguished from those which originate in the membranes themselves and penetrate from them into the cortical substance—which, clinically, comes to the same thing. In both cases we find the arachnoid and dura mater overlying the cortical tubercles more or less adherent to one another, so that on our removing the dura mater a portion of the tubercle is apt to remain attached to it. Sometimes the size of the nodules is much greater. I have myself seen them as big as a walnut, and even bigger; and these when cut into, usually no longer presented a homogeneous caseous appearance, but contained fissures and cavities filled with a whey-like fluid. In one child I even found on the outer surface of the right optic thalamus, a tubercular mass as large as a hen's egg, full of fissures, and in other cases there was diffuse caseous degeneration of the cortex or caseous metamorphosis of an entire cerebellar hemisphere. Calcification of cerebral tumours is not a common occurrence; I have observed only two cases of it,

one of which has already been mentioned. In the other case a tubercular nodule of the cerebellum contained very hard calcareous particles.

In large tubercular masses we can generally, on careful examination, distinctly make out that they have arisen from the confluence of small nodules lying close together. The interior—apart from the fissures already mentioned—is partly firm and homogeneous, partly granular and friable. The outer layer is often thin, greyish-white, and transparent, and numerous miliary nodules can be discovered in it. It is partly through the confluence of these, and partly by a chronic caseous encephalitis that the larger nodules seem to be developed. Smaller tubercles are not unfrequently encased in a thin capsule of connective tissue, while the larger ones are usually more diffuse and are imbedded in the extremely vascular, moist and softened brain-substance. The number of brain tubercles varies greatly. Most rarely we find only one (solitary tubercle), usually several scattered through different parts of the brain, occasionally very many (a dozen or more), and of this I have already given examples. In most cases we also find symptoms of tubercular meningitis and accumulation of serum in the ventricles —which we shall speak of later on—and not uncommonly small ecchymoses in the pia mater or brain-substance. I have repeatedly observed that the accumulation of miliary nodules in the pia mater was most marked in the immediate neighbourhood of the caseous nodules, especially on the convexity. More or less advanced tuberculosis and caseation of other organs is generally, but by no means invariably, present also. In the case already mentioned (p. 276) in which a dozen large tubercles were found in the brain, only a few miliary nodules were found in the right lung, while all the other organs were perfectly free from disease.

The question, whether we can diagnose from the symptoms in what part of the brain the tubercular mass is situated, does not strictly speaking concern us here, seeing that the conditions are the same as in adults. I refer you, therefore, to a paper of mine published in the Charité-Annalen (Jahrgang IV.) from which you will find that in spite of the knowledge recently acquired by experiments, the diagnosis of the localisation of cerebral tubercle is still far from being established; and the latency already dis-

cussed affords another proof of this. I have, however, records of three cases in which a solitary tubercle of one frontal lobe resulted in symptoms of irritation or paralysis on the opposite side of the body; and we may certainly conclude from this that these symptoms may be caused by disease affecting exclusively the convolutions I have mentioned. I say advisedly "may," for it is not a matter of necessity. I have often enough seen exactly the same morbid conditions—hemiplegia and contractures—in cases at the post-mortem of which this area of the cortex was perfectly normal, while there were tubercular masses in the greatest variety of other situations in the cerebrum or cerebellum. Although the large number of these masses must make all efforts to arrive at a local diagnosis vain, still even solitary tubercles often present symptoms at variance with the results of experimental research. I therefore advise you to exercise the utmost caution in local diagnosis—and especially not to over-estimate the "motor centres of the cortex" of which so much is being made at present—if you do not wish to find yourself unpleasantly mistaken at the post-mortem. It would be useless labour to discuss here certain cases of solitary tubercle from which conclusions have been drawn as to the functions of different parts of the brain; for on this subject the greatest diversity of opinion exists on all sides. I will only refer here to the case, mentioned on p. 278, of solitary tubercle of the left optic thalamus, which was accompanied by chorea-like movements of the right side of the body. Quite independent of the fact that the latter only set in during the final meningitis (and, according to my experience, can only be ascribed to this), I have often seen cases of tuberculosis of the optic thalami in which no movements whatever of this kind appeared. One of these may be given here.

Hedwig F., 4 years old, admitted on April 24th, 1881. Healthy until middle of February. Seemed out of sorts after a fall on the forehead. A fortnight later, left internal strabismus, frequent vomiting, giddiness. Later, retraction of the head, and contractures at the hip and knee-joints, which disappeared under chloroform, sometimes also vanished spontaneously. Headaches, drowsiness. In May, short epileptic attacks. 1st June, slight left ptosis, increasing amblyopia with nystagmus. 14th June, neuro-retinitis in both eyes. On 5th August, commencement of tubercular meningitis. Death

on the 9th with external high temperature at the last (104·7—106·2° F.).

Post-mortem: basilar tubercular meningitis, acute hydrocephalus. The left optic thalamus reddened and nodular, the right smooth; both contain several caseous nodules surrounded by a greyish-red transparent layer. One of these in the left thalamus, is the size of a hazel-nut and reaches to the surface. In the vermiform process of the cerebellum there is a caseous nodule with soft centre, of the size of a small walnut, and in each hemisphere of the cerebellum a tubercular mass of the size of a hazel-nut Spinal cord normal.

On the other hand I have observed choreic movements in one case in which the central ganglia of the brain were quite unaffected, and only the cerebellar peduncle was the seat of the tubercular mass.

Child of 2 years, admitted on August 6th, 1883. Well-nourished. Scarlet fever 8 months ago, soon followed by chorea-like movements in the left side. Slight left convergent strabismus, tremor of the tongue when extruded, contracture of the left arm at the elbow, and of the left leg at the knee-joint. Athetosis-movements of the fingers and foot of left side. These ceased during sleep, but continued constantly when the child was awake. Both extremities were paralysed, the cervical glands swollen, some of them suppurating. In the left orbicularis palpebrarum there were continuous spasmodic movements when awake. From 29th September, fever, vomiting, increasing coma. On the 30th, death, with temperature 104·9° F.

P.-M.—Solitary tubercle the size of a hazel-nut in the right cerebellar peduncle.

The regions of the pons and corpora quadrigemina seem to me to be those in lesions of which an approximate diagnosis is soonest possible, from the simultaneous or successive affection of several nerves whose nuclei are situated in this region. The simultaneous paralysis of one or both oculo-motor nerves, of the optic, facial, and abducens—which are either principal symptoms or at least precede the hemiplegia—strongly favour this local diagnosis; and in this connection I would refer you to some observations I have published on tuberculosis of the corpora quadrigemina and pons,[1] in connection with which I have discussed the other cases of the kind which have been published. I shall add to these another case of tuberculosis of the cerebral peduncle, which shows that here, as in tumours of the pons,

[1] *Beitr. z. Kinderheilk., N. F.*, S. 72.—*Charité-Annalen*, Bd. iv.

owing to pressure on the neighbouring oculo-motor nerve, paralysis of it may occur along with crossed paralysis of the extremities.

Max Sch., 3 years old, admitted on 26th March, 1883. Of healthy parents, but himself scrofulous, and for a long time sickly. For 9 weeks tremor of left hand, which had gradually spread to the whole arm, combined with contracture at the elbow-joint. For six weeks tremor of left leg also. This became aggravated on the attempt to grasp anything, but ceased during sleep. Fingers flexed. No paralysis. At the same time ptosis of the right eyelid, marked dilatation of the right pupil, and divergent strabismus, so that the right eyeball was turned outwards and could not be brought inwards beyond the middle line. Facial nerve unaffected. After recovering from an attack of scarlet fever in the ward, in the middle of April the boy became steadily more apathetic and uninterested. On 25th he also became affected by ptosis, mydriasis, and divergent strabismus of the left eye; and died on 8th May of measles and broncho-pneumonia.

P.-M.—In the right crus cerebri a hard tubercular mass the size of a cherry, projecting into the third ventricle. At the base, the right oculo-motor nerve is flattened by the pressure of the tubercular mass and is thinned and greyish in colour. In the apex of the left lung, a cavity the size of a walnut in which there is a large half-dissolved caseous plug. Broncho-pneumonia, laryngitis. No tubercle elsewhere.

I have yet to treat of a pretty common sequela of cerebral tubercle, namely, chronic hydrocephalus. It is supposed that the tubercular nodules, especially those situated in the middle lobe of the cerebellum, or between it and the tentorium cerebelli, may, by pressure on the veins of Galen and their chief branches, produce engorgement and exudation into the ventricles. This may be indicated even during life by increased size of the head, even when the sutures are already closed. The first case of this kind that I met with, was that of a girl of 3, in whom enlargement of the head, impaired intelligence, and blindness of both eyes were added to the symptoms of cerebral tubercle. Von Graefe discovered neuro-retinitis as the cause of blindness, along with marked swelling of the papilla, and tortuosity of the veins. As no post-mortem was made, however, it was not ascertained whether the tumour which produced this result by pressure on the veins was tubercular or of some other nature. In two other cases[1] tuberculosis of the middle lobe of

[1] *Charité-Annalen*, iv., S. 498, 499.

the cerebellum was found along with a moderate distension of the ventricles. However, only to the second of these cases can much value be assigned in this connection, as it alone presented no tubercular meningitis. The following case, observed in my ward, is more to the purpose :—

> Clara G., 3 years old, formerly healthy. For about 6 months, gradually increasing enlargement of the head, to which had been added a slowly increasing right-sided hemiplegia. The latter no longer so marked as formerly, so that the right arm especially could now be pretty well used. She had had whooping cough for 7 weeks. Admitted into hospital on January 4th, 1879. Head hydrocephalic, circumference 21¼ inches; fontanelle widely open and extending into the sutures; tense and elastic. Eyes somewhat protruding. Drowsiness. Violent attacks of whooping cough, diffuse bronchial catarrh, remittent fever increasing in severity until death, which took place on the 15th. Temperature towards the end 105·1° F. Pulse 160 and somewhat irregular. Post-mortem: very marked chronic hydrocephalus of the ventricles, with compression of the brain-substance, flattening of the convolutions and extreme distension of the skull. The distance between the parietal eminences was about 6 inches; the sutures extremely wide with very marked serrations gaping in some places, and fibrous. The left hemisphere of the cerebellum converted almost entirely into a homogeneous yellowish-white caseous mass, surrounded by a narrow border of healthy substance. Nothing else of importance.

This tubercular mass had undoubtedly existed in a latent condition for a considerable time, before it occasioned hemiparesis and produced engorgement by the increasing pressure on the veins. The mesial position of the nodules in the line of the vena magna is consequently not absolutely necessary; for any tumour lying to the right or left of it may, by increasing the lateral pressure, produce engorgement in the area of distribution of the neighbouring veins. This can be made out in the most various cerebral tumours by means of the ophthalmoscope. We must, however, consider whether the mechanical explanation of chronic hydrocephalus as being due to compression of the veins is the only one which will account for all such cases; or whether a state of irritation originating in the pia mater covering it, and transmitted through the velum interpositum to the ependyma of the ventricles, may not also have to be considered as a factor in the causation of the serous exudation.

To speak of effective treatment of cerebral tubercle is, of course, out of the question. Neither by iodide of potash (the favourite drug), nor by other anti-scrofulous remedies can we remove caseous nodules from the brain when they are once developed. We must, however, acknowledge that a natural cure is possible—especially in the case of solitary tubercle—and you may therefore always, although only with very slight prospect of success, attempt to favour this process as much as possible by a tonic line of treatment (iodide of iron, codliver oil, saline baths, fresh air, nourishing diet), and by preventing the patient from being exposed to injurious influences. A temporary improvement (disappearance of the paralysis, long intermission of the fits, &c.), must not—as some of the cases I have given will show—lead you to suppose that recovery has taken place. And indeed such a supposition is generally prevented by the accompanying tuberculosis of other organs. The case, however, becomes quite hopeless whenever the first certain signs of tubercular meningitis appear. Epileptiform attacks, with or without febrile symptoms, which occur suddenly in the course of the disease, and are followed by coma or even local paralysis, are always to be regarded with suspicion; because tubercular meningitis not uncommonly begins with these very symptoms. We must remember, however, that the same symptoms may arise from sudden hyperæmia or localised encephalitis in the immediate neighbourhood of tubercles. Therefore we must not neglect to order some leeches to the head, iced compresses and purgatives (Form. 7). Under this treatment the threatening symptoms occasionally pass off, till after some time death is caused by a fresh attack or by tubercular meningitis.

XIII. *Tumours of the Brain.*

I have but little to tell you of cerebral tumours in children, as they resemble, in all respects, those occurring in later life. The different forms of sarcoma are those most frequently found; and they develope either in the middle of the cerebral substance—especially in the pons Varolii and its neighbourhood—or grow from the cranial bones, and in that case interfere with

the brain by pressure. I have myself records of several such cases with post-mortems, and others which are incomplete from there having been no examination of the body.

Alice G., 6 years old, admitted into the ward, July 16th, 1874.[1] Violent headaches for some months, especially in the left frontal region. Bilateral amaurosis for 6 weeks, which developed within a few days. On examination we found incomplete ptosis on the left side, complete immobility of the left eye, the pupil of which was dilated and did not react. The right eye could be well moved, the pupil equally dilated. Neuro-retinitis in both eyes. Occasional pain in the left nasal cavity, from which there was a greyish purulent discharge. General health good till the 24th when the child became affected by a severe attack of scarlet fever. Death on August 2nd.

Post-mortem: a myxo-sarcoma—half the size of the fist, originating in the bones of the middle cerebral fossa, and completely filling it—had grown into the upper part of the left nasal cavity after penetrating the lamina cribrosa, and had surrounded the optic chiasma and all the ocular nerves on the left side. Brain and meninges normal, but pressed upward a little.

The post-mortem explains perfectly the amaurosis of both eyes, the paralysis of all the muscles of the left, and the purulent secretion from the left nasal cavity. The absence of all paralytic symptoms in the extremities, in spite of the compression of the brain substance from the base, is worthy of note.

Anton H., 11 years old, brought to the hospital on June 26th, 1872.[2] Formerly healthy except for occasional headache. Six years before, excitement and chill during a fire. A week later complete right ptosis, swaying gait, increase of headaches. On examination there was ptosis of the right side, moderate dilatation of both pupils, stupid look, great restlessness, frequent rotatory movement of the head, especially from right to left. Upper extremities could be used, though only feebly. Could not walk without support. When supported under both armpits he could shuffle along laboriously in an ataxic manner. When lying, the lower extremities could be freely moved. The sensibility diminished at some places on the right leg. Speech faltering, scarcely intelligible. Swallowing difficult. Vision unaffected, intelligence unimpaired. P. 54—84. After some days speech even less distinct, the movements of the head more forcible, the

[1] *Charité-Annalen, Jahrg.*, i., S. 561.

[2] *Charité-Annalen, Jahrg.*, i., S. 562, and Scheibe, *Inaugural-diss. über Hirngeschwülste im Kindesalter:* Berlin, 1873.

mind confused. On July 4th, sudden loss of consciousness and asphyxia. Artificial respiration and faradisation although continued steadily for 2 hours, had but a passing effect (pulse-rate increased, diminished cyanosis). Death in the afternoon.

P.-M.—Dura mater very tense, brain flattened. In the region of the pons Varolii a large shapeless tumour of the size of a testicle, involving the pons and the left superior cerebral peduncle, reddish-grey soft; within it a cavity, the size of a cherry-stone, filled with a spongy, sulphur-yellow mass. Chronic hydrocephalus of the ventricles. Under the microscope the tumour was found to be a large-celled sarcoma, the processes of which could be traced right into the crura cerebri.

Anna D., 11 years old, admitted into the ward on May 4th, 1876. Had always been healthy except for an attack of pneumonia 4 years previously. For a considerable time (?) increasing uncertainty of gait. Since April of that year squinting of right eye, and giddiness. Nausea, occasionally vomiting. On examination, her gait was found to be exceedingly uncertain and staggering, especially when the eyes were closed. Motility and sensibility almost unimpaired. Paralysis of the left abducens with internal strabismus and inability to turn the eye outwards. Pupils normal and brain unaffected, but great apathy and dulness. Speech nasal and indistinct. Fluids sometimes returned through the nose when she was drinking. Soft palate hanging loose, but little moved in breathing and phonating. During the next few days vomiting, very difficult defæcation, retention of urine (met by the introduction of a catheter), speech less distinct, and swallowing daily more difficult. On the 8th, the right abducens also paralysed. Intelligence steadily decreasing, drowsiness. Pulse usually 80—100, occasionally falling to 64 and under, and irregular. From the 24th onwards, complete apathy. Nutrient enemata because of inability to swallow. Sinking of strength. Death on 29th from œdema of the lungs. By comparing with the previous case I was led to make the diagnosis of tumour of the pons Varolii.

P.-M.—The pons enlarged to twice its usual size. The medulla oblongata—especially on the right side—also enlarged, but only to a slight degree. Pons soft, fluctuating at certain points. On section several tumours from the size of a bean to that of a cherry, of medullary consistence and greyish-red colour, not circumscribed from the surrounding tissue. On examination these were found to be sarcomatous. No other abnormalities anywhere.

The two last cases, on account of their having a number of symptoms in common (bilateral paralysis of the abducens, paralysis of the muscles of the palate with difficult swallowing

and indistinct speech, ataxia of the lower extremities) may claim a certain importance in connection with the diagnosis of diseases of the pons.

The occurrence of gummatous tumours in the brain in children is also occasionally mentioned, and indeed one can see no reason why childhood should be exempt from these manifestations of syphilis. I should, however, point out to you that the diagnosis of these tumours from tubercle is often very difficult, and that even the microscope may fail us, so that many tubercular masses in the brain may pass for gummata, and *vice-versâ*. Even tubercle-bacilli cannot be regarded as quite certain criteria in such cases; since they perish in old caseous nodules, and on the other hand similar microbes have been found in syphilitic products. In such cases the caseous condition of other organs —especially of the lungs and bronchial glands—put the presence of tubercle beyond doubt. If indubitable signs of syphilis are not present at the same time, and the complete absence of tubercle in the other organs is ascertained by a very careful post-mortem, I should be very slow, especially in children, to diagnose gummata in the brain at the post-mortem; for they are extremely rare at this age compared to tubercle. I have hitherto met with only one undoubted case, so far as I am aware, and it has already been given (p. 110).

Other varieties of tumour (glioma, medullary sarcoma, echinococci, cysticercus), which have occasionally occurred in the brain in children, do not present anything characteristic, nor do the encephalitic focal lesions, which end in softening of the brain-substance or in the formation of abscesses. All these conditions are the same in children as in adults, and I therefore think it unnecessary to discuss them further here. Abscesses of the brain are not very uncommon in children, since injuries —which are a very common cause of them—are more frequently encountered at this age than in later life. Besides, we have to take into account here, the greater frequency of caries of the petrous bone, the tendency of which to cause abscesses is well known. My personal observations are confined to the already-mentioned association of cerebral tubercle with disease of this bone. On the other hand I have seen an enormous abscess of the brain in a scrofulous girl of 12, which involved almost the whole frontal lobe of the right hemisphere, occurring along with

caries of the lamina cribrosa of the ethmoid. In this case there had for many weeks been violent attacks of neuralgic pain in the region of the right supraorbital nerve, which could only be relieved by the injection of morphia, while the intervals were almost quite free from morbid symptoms, and only the pressure on the orbital margin—especially towards the nasal side—caused pain. Quite suddenly violent epileptic convulsions, coma and hemiplegia set in, and caused death within a few days.[1] You see from this that the diseases of the nasal cavity (chronic rhinitis) in children should be treated with no less care than those of the ear, the dangerous character of which has long been recognised.

XIV. *Atrophic Cerebral Paralysis.*

A cerebral form of infantile paralysis may, like the "spinal," persist to a late period of life, and then for the first time come under the physician's observation. It is much oftener, however, seen first during childhood, even during the early years of life. The children present the symptoms of more or less complete hemiplegia, with or without implication of the facial or other cranial nerves. The upper extremity is generally more seriously affected in regard to its movements than the lower, the latter being often still used in walking, although dragging somewhat. The paralysis is either congenital, that is, appears immediately after birth, or it arises in the first period of life, between the 3rd and 12th months, or even later; and the parents usually tell you that it came on after an attack of "inflammation of the brain," *i.e.* as a rule, after a febrile comatose preliminary stage, lasting from a day to a week, with more or less violent convulsions, which—as we have seen above (p. 248)—but seldom usher in spinal infantile paralysis. In course of time, however, contracture and atrophy of the paralysed parts gradually develope in the cerebral paralysis we are speaking of also; and these parts finally appear not only colder, thinner, and more shrivelled than the healthy ones, but also shorter and stunted in growth. This disease differs from the spinal form, first, in its invariably unilateral character, secondly, and more especially by the long

[1] *Berl. klin. Wochenschr.*, 1882, No. 15.

persistence of the electrical reaction in the paralysed muscles, which does not disappear till their atrophy is extremely advanced —until, in fact, scarcely any normal muscular tissue is left. In the cerebral form the atrophy of the limbs takes place, almost always very slowly, and only after the disease has lasted many years, and it rarely reaches the extreme degree which spinal paralysis so often presents. Still, in many cases I have seen a very marked shortening of the affected limb and diminution in size of the hand and fingers.[1] Sensory disturbances are in these cases also rarely observed. In one case (a boy of 7 years) in which the disease had begun at the age of 18 months, anæsthesia of the paralysed arm was said to have been present at first, and to have afterwards disappeared. Here also as in the spinal form, the head of the humerus sometimes falls out of the glenoid cavity, so that the finger can be inserted between the joint and the head of the bone. The paralysed upper extremity frequently shows athetosis-movements of the fingers, especially on purposive muscular action. The development of speech also suffers more or less, likewise that of the intelligence, which may present all the intermediate stages from slight stupidity to regular idiocy. Very frequently epileptiform attacks are also added, which complete the clinical picture of the disease. As already mentioned, such children, who are only a burden to their relatives, may reach the age of 20 years or more; but usually they die sooner, either in a convulsive attack, in coma, or from the results of a chance complication.

The invariably incurable character of this disease is due to the anatomical conditions present. For we have here an atrophy or complete absence of certain areas of the brain; for example, of some of the convolutions of one hemisphere, of a half or a whole lobe, of the great cerebral ganglia, &c., which are replaced in such cases by an accumulation of serum, often accompanied by a thickening of the cranial bones. I described a most typical case of this kind in my graduation thesis.[2]

> Girl of 19 years, healthy at birth. Convulsions at the age of 3 months, leaving behind them right hemiplegia. Later, atrophy of both the affected limbs, which were regularly stunted. Sen-

[1] *Cf.* Seeligmüller, *Jahrb. f. Kinderheilk., N. F.*, xiii., S. 356.—Förster, *Ibid.* xv., S. 268.

[2] Henoch, *De atrophia cerebri:* Berolini, 1842.

sibility normal. Cranial nerves not paralysed. Fingers flexed. Intelligence almost at the point of idiocy. Speech monosyllabic, but quite free. Death from phthisis.

P.-M.—The left half of the skull ½ inch smaller than the right. The left frontal bone thickened. The middle and upper part of the left hemisphere was quite absent, being replaced by a cyst filled with serum, which extended to the lateral ventricle. The latter was much dilated and filled with serum. The corpus striatum and the optic thalamus were shrivelled to half their normal size. This atrophy was continued partially in a cross direction, the optic tract, the corpus albicantium, the crus cerebri, the left side of the pons and the pyramid on the right side, appearing considerably thinner; of the pyramid, especially, scarcely ¼ part remained.

In all these cases we find an atrophy of the pyramidal tracts caused by a retrogressive metamorphosis (with a breaking down into granular cells), which arises in the atrophic area of the brain, and may be followed as it crosses over into the opposite half of the spinal cord. On the other hand we have no thoroughly clear insight into the essential nature of the disease itself. Cases like that given above, may also be congenital. It would seem then that we have a meningo-encephalitic and hæmorrhagic process arising either during fœtal life or just after birth, by which the affected part of the brain is disintegrated. In course of time, owing to an inflammatory reaction in the immediate neighbourhood, a capsule like the wall of a cyst forms round the disintegrated mass of brain substance; which gradually undergoes fatty degeneration, and is absorbed, leaving in its place a more or less clear serum as the contents of the cyst.[1] In other cases we find varieties differing more or less from this form, the development of which, however, must be explained in the same way.

Elizabeth R., 12 years old, admitted into the hospital on 8th January, 1879. Had suffered since early childhood from irregularly-recurring epileptiform fits; sometimes 3—5 in one day, then weeks without any. There had also existed as long as she could remember, paralysis of the right side, especially of the arm. She was admitted on account of phthisis pulmonalis. The fits which were observed in the hospital were of a distinctly epileptic character, and affected chiefly the right (paralysed) limbs, the head, eyes, and right facial nerve. The paralysed right arm could only be used to a very limited extent, and was wasted to a

[1] Kundrat, *Die Porencephalie. Eine anatomische Studie:* Graz, 1882.

moderate degree and slightly flexed at the elbow. Death took place on 25th, and at the *P.-M.* the following changes in the brain were found:—pia mater on the convexity of both hemispheres markedly œdematous. All the convolutions on the left side very small and narrow, sulci very deep. In the sulci between the 2nd and 3rd frontal convolution, as well as in the central fissure, the pia mater was thickened, and could only be removed with very great difficulty. The affected convolutions were extremely narrow, and presented a depression and a rust-brown colour, which was evidently due to former hæmorrhages. On the right side everything normal. The above-mentioned depression was filled with serous fluid, over which the arachnoid stretched.

In this case an original fault in formation (smallness of all the convolutions of the left convexity) seems to have been accompanied at a later period by a hæmorrhagic and exudative affection of the membranes in the situation described. From the pressure of the exudation there resulted atrophy and depression of the frontal convolutions, and they were gradually replaced by serum. Both in this case and in the following one, we see symptoms of paralysis and irritation arising on the opposite side of the body from the affection of the anterior part of the cortex; while in the second of the following cases, the temporal and parietal lobes seemed to be far more affected than the frontal region.

Margarethe G., 5 years old, admitted into the hospital July 11th, 1875. Healthy till 18 months old. About this time a violent shock caused by the breaking of an axle during a drive. Some days later, sudden left hemiplegia after a warm bath. In course of time improvement, and good mental development. Disturbance of speech, dating from spring, 1875; words which were formerly spoken fluently could no longer be pronounced. At the same time, change of character; destructive mania and great violence. Gait staggering and uncertain. The hemiplegia was now only manifested by less energy of the left limbs, which are atrophied. Death on 24th September from diphtheria. *P.-M.*—Pia mater on right side over the upper frontal convolution considerably thickened, whitish, and opaque, adhering at this place extremely firmly to the brain substance, which seemed wasted and very hard. The whole right upper frontal convolution markedly atrophied; the atrophy of the rest of frontal lobe was somewhat less, but still very noticeable. The atrophied convolution was likewise peculiarly transparent and of a very bright red colour, except in its posterior third, which was whitish and very uneven. Everything else normal.

Georg St., 5 years old, admitted on July 23rd, 1877. "Inflammation of the brain" 3 years before, after which aphasia, mental hebetude, and right hemiplegia gradually developed. In the 2 years following, repeated epileptic attacks, sometimes 3 or 4 times in one day. A robust and well-nourished child. Stupid expression of face, divergent strabismus of right eye. Intellect confused. The patient only answered with inarticulate noises without understanding what was asked him; speech quite lost. Hearing and sight unaffected. Right hemiplegia, with rigid contracture of the flexors, which could only be overcome with difficulty. Faradic excitability of the flexors retained, that of the extensors weakened. Analgesia of the paralysed parts (to pricks with a needle), gait uncertain and reeling. The right leg was dragged, and only the fore part of the foot used in walking. Right arm flabby and wasted, circumference $\frac{3}{8}$ of an inch less than that of the left. Motions and urine passed in bed. No fever. On the 5th August, scarlet fever; on 13th, double broncho-pneumonia; on the 20th, death.

P.-M.—Partial synostosis of the coronal and sagittal sutures. Inner surface of the cranial bones shows here and there a very slight white thickening, especially on the frontal bone. Dura mater, especially on the left side, very flaccid, forming wide folds, on both sides adherent to the pia mater at many points. The pia mater on the right side much reddened, very vascular. Here and there very opaque and thickened, especially at the border between the parietal and occipital lobe. The left hemisphere strikingly diminished in size. The pia mater in this position presents much fibrous thickening all over. The temporal lobe appears especially atrophied and the pia mater over it, with the immediately subjacent cortical substance, is œdematous, greyish-red, and transparent, like a bladder full of water. Pia mater at the base thickened, especially in the Sylvian fissure. Vessels unaffected; ventricles, especially the left, much distended with serum, their walls much thickened and papillated on the surface. On more careful examination we find that the atrophy extends from the temporal lobe backwards to a considerable part of the parietal lobe and forward to the lowest portion of both central convolutions. On incision into these parts we find that there was also a considerable atrophy of the white substance. In this situation the brain-substance was reddish-grey, tough, very vascular, studded with pale, hardish nodules (sclerosis). The right hemisphere unaffected, with the exception of atrophy of a part of the parietal lobe.

More rare than the cases I have just given are those with bilateral atrophy of the brain substance, which may cause symptoms on both sides of the body.

A boy of 6 years old,[1] admitted on July 20th, 1874. Had had measles when 6 months old. Soon after, "convulsions," which were frequently repeated during 8 days, then became less frequent, and finally only occurred very seldom. Immediately after the first convulsive attack the present disease had developed. Actual paralysis was nowhere noticeable, although there was extensive rigidity of the muscles. When he was lying, both lower extremities were stiff, with slight flexion at the knee-joints. Any attempt at flexion or extension was rendered difficult by the state of tension of the flexors and extensors. The upper extremities, especially the right, flexed at the elbow-joint. Extension was very difficult, and could not be effected by the patient himself. As soon as the boy was placed on his feet and encouraged to walk, there at once resulted a rigid contraction of the calf-muscles, with the foot in a position of pes equinus, and at the same time marked dorsiflexion of the toes, so that standing and walking were absolutely impossible. Slight contracture also in the joints of the hands and fingers. Purposeless, chorea-like movements noticeable on the attempt to take hold of anything. Speech stuttering, laborious, difficult to understand; mental energy much weakened. Otherwise normal in all respects. Death from diphtheria on the 12th. *P.-M.*—The right arm, measuring from the axilla to the styloid process of the radius, was shortened to the extent of one inch; and there was atrophy of the muscles. The cranial vertex slightly asymmetrical, the right parietal bone being more arched and larger than the left, and the oblique diameter (from the left side in front to the right behind) larger than the corresponding diameter on the other side. Dura mater normal. Pia mater on the frontal lobes on both sides of the incisura magna thickened, opaque, and distended like a bladder by a clear fluid, and when this was let out the affected part of the brain appeared sunk in. The first and partly also the second frontal convolution on both sides was atrophied, being scarcely $\frac{1}{3}$ as broad as in the normal state; very soft and uniformly greyish-red on section. The adjacent parts of the medulla had the same characters and were also atrophied. The third frontal convolution only affected to a slight degree; insula normal. Corpus callosum, fornix and septum lucidum considerably atrophied. Lateral ventricles much dilated, distended with serum, especially their anterior horns, which took up more room than all the rest of the lateral ventricles. Ependyma of all the ventricles much thickened, tough, and covered with little prominences. Otherwise nothing abnormal.

Thus many cases of "spastic spinal paralysis" which have not been examined post-mortem may have depended on such a bilateral deficiency of the brain substance, especially those

[1] *Charité-Annalen, Jahrg.* i., S. 567.

that were accompanied by a weakened or quite ruined intellect (p. 263).

On microscopic examination of the atrophied gyri in these cases, we find sclerosis taking place, *i.e.*, destruction and finally disappearance of the real nerve elements, in the place of which there is an interstitial growth of the neuroglia, fatty granular cells and more or less numerous corpora amylacea. There are also often hæmatoidin crystals which indicate that there had originally been a hæmorrhagic process. These areas of sclerotic atrophy seem, then, to represent the last remains of inflammatory and hæmorrhagic processes, which have occurred during fœtal life or during the earliest childhood; and to them there may also be added an exudative inflammation of the pia mater which exerts pressure on underlying tissue. The cases I have given, and many others, show that under these circumstances the prognosis must be absolutely bad, and treatment of no avail. If you wish to do something in deference to the parents' wishes, the use of electricity is the only thing that remains, and in these cases it may arrest the muscular atrophy even better than in infantile spinal paralysis. Frictions of the limbs, stimulating baths, and gymnastics may also be used in this as in the latter disease. The physician, however, loses heart in those cases much sooner, on account of the impairment of the intellect which takes place at the same time, and which may progress to idiocy, and the unfortunate child is finally left to his fate as a burden to his family. That sclerotic patches (sclérose en plaques) may occur in children in other parts of the brain besides the cortical layer, is a fact,[1] although it is of rare occurrence and I have never myself observed it. The majority of the cases published are, however, of no value; because no accounts of post-mortems are given. In the symptoms described I find nothing that is characteristic, at least nothing which might not have been observed also in other chronic diseases of the brain in children (tubercle, tumours, chronic meningitis). The connection with syphilis, which Moncorvo assumes, is in no way proved.

[1] Ten Cate Hoedemaker, *Deutsches Archiv f. klin. Med.*, xxiii., S. 443.—Förster, *l. c.*, S. 272.—Pierre Marie, *Revue de Méd.*, 1883, No. 7.—Moncorvo, *Contributions à l'étude de la sclérose multiloculaire chez les enfants:* Paris, 1884.—Richardière, *Sclérose encéphalique primitive de l'enfance:* Paris, 1885.—Kast, *Arch. f. Psychiatrie*, xviii., H. 2.—Unger, *Ueber multiple inselförmige Sclerose*, &c.: Leipzig u. Wien, 1887.

XV. *Chronic Hydrocephalus.*

The only certain symptom of this disease is the more or less rapidly growing size of the head due to increasing pressure of fluid filling the cerebral ventricles (more rarely the space between the dura mater and arachnoid). Slight degrees of hydrocephalus in which there is no enlargement of the head, are beyond the reach of diagnosis. More than 3 ounces of fluid may be found in the dilated ventricle of children who have died from various cachectic diseases, especially tuberculosis, without the suspicion of this condition having been suggested by any sign during life. But it is not of such cases that we have here to speak.

On the other hand, however, an unusually large size of the head must not of itself mislead you into at once assuming the presence of hydrocephalus. Children have often enough been brought to me who, to their parents' alarm, had been declared hydrocephalic by medical men because their head was very large, and the fontanelles and sutures not yet closed; and yet I was soon able to comfort the parents with the assurance that their anxiety was groundless—that there was no hydrocephalus, but only a rickety form of the skull which had misled the doctor. I grant that the diagnosis is occasionally difficult if one takes into consideration the large size and arrested ossification of the skull; but careful observation of the mental condition, the movements and the looks, will soon guide you to a conclusion. Only in those cases in which there is a combination of rickets with hydrocephalus can there long exist any doubt as to the diagnosis.

Most children with chronic hydrocephalus come under medical observation during the first six months of life. For the steady increase of the size of the head, with which the growth of the rest of the body does not keep pace, soon rouses the relatives' attention. The increase of size is not very considerable at first, so that one may make the mistake of denying it altogether, and taking for granted that the mother has been misled by the comparatively large size of the head which is a constant feature of early childhood. Measurements, however, soon decide the matter. They are taken with a centimetre-measure in the following way: (1) the circumference of the head (the glabella and occipital

tuberosity being taken as the central points),[1] (2) the transverse diameter (from one mastoid process to the other, over the vertex), and (3) the longitudinal diameter (from root of the nose over the vertex to the occipital tuberosity). We can then from time to time make out an increase of one centimetre and more. Most hydrocephalic crania are distinguished by a marked prominence of the frontal bone, and a lateral bulging of the parietal bone, which is especially noticeable when one looks at the skull from above. Only exceptionally have I met with a dolichocephalic shape, *i.e.*, an elongation of the longitudinal diameter with lateral flattening of the skull combined with extreme hydrocephalus. We almost always see the subcutaneous veins of the head standing out like blue cords. On palpation of the skull, we generally find that the process of ossification is arrested. All the fontanelles, especially the great one, are widely open, the sutures gaping so that we can feel the fibrous membrane which stretches between the bones pressed outwards by the pressure of the cerebral fluid, elastic and more or less distinctly fluctuating. Sometimes—but only in very extreme congenital cases, or those which have arisen very early, in which the formation of bone was still extremely deficient—I have felt scattered islands of bone within the fluctuating membrane joining the cranial bones. In one of these cases the membrane just above the flat portion of the occipital bone, was prolonged into a round diverticulum the size of a walnut, which—as we found on puncture—was filled with fluid and was evidently a meningocele. The assumption that there was also external hydrocephalus in this case—*i.e.*, an accumulation of fluid under the dura mater—was confirmed by the post-mortem.

Much more rarely—indeed only in quite exceptional cases—have I met with a normal condition of the ossification, or even an unusual thickness of the skull although it was so much enlarged. In one case this appeared chiefly in the region of the temporal bone and gave to the skull a striking appearance of breadth.

Owing to the marked increase in size, the head gradually becomes so heavy that the child cannot hold it up. When it is

[1] In new-born children the circumference of the head is, on an average, 39—40 ctm., from 6—12 months about 40—45 ctm., and then gradually increases to 50 ctm., which it reaches about the twelfth year (Steffen).

not supported it follows the law of gravity and sways from side to side. The huge proportions of the head contrast strongly with the small size of the face, which owing to the steady wasting becomes still smaller, and assumes an almost triangular shape. At the same time one is struck by the peculiar staring look, or by the downward direction of the eyeballs (which was pointed out by the old physicians) so that the iris is covered by the lower lid and a considerable extent of the upper portion of the sclerotic is always visible. The view that this position of the eyeball—which, moreover, is not always present—always arises from a pressing downward of the orbital plate of the frontal bone is doubtful for this reason, that in that case a diminution of space in the orbit and consequent exophthalmus would always result. As a fact we not uncommonly find a certain degree of the latter, and then we also find by palpation that the bony roof of the orbit is as thin as a piece of parchment, and at the post-mortem this appears to form an extremely obtuse angle with the other portion of the frontal bone. But this does not always occur; for in two cases in which the direction of the eyes certainly gave one reason to assume such pressing downward of the orbital plate, I found to my surprise at the post-mortem that the latter was in its normal position; although it is conceivable that the very thin bone which was pressed downward during life had returned to its normal position after the removal of the brain that was weighing upon it. The downward direction of the eyeball may, however, also be caused by a partial paralysis of the oculo-motor nerve, namely, of those branches which supply the rectus superior; so that the action of the rectus inferior preponderates. The paralysis of other branches of the same nerve also occur, and occasion instead of a downward direction of the eye, a divergent squint or some other abnormal position, or more or less well-marked ptosis. It is but very rarely that we find both the direction of vision and the position of the eyeball quite unaffected. On examination with the ophthalmoscope we generally find a pressure-atrophy of the optic disc and dilatation of the retinal veins owing to the interference with the backward flow of blood into the cavernous sinus which the compression causes. In most cases the mental development remains in a very backward condition. The children are extremely apathetic, seem neither to see nor hear distinctly and do not recognise

those about them; and not uncommonly they present all the symptoms of regular idiocy—the saliva trickling from the half-opened mouth and macerating the skin of the lower lip and chin. This, however, is by no means always the case; indeed we are sometimes astonished at the amount of intelligence and mental energy retained even in advanced cases of hydrocephalus. Thus I have observed a child of 1½ with a very extreme form of the disease who recognised those around him, called "papa" and "mamma," and followed with his eyes anything held before him. Even a few weeks before death, which took place with violent convulsions, the vision was quite unimpaired, the child spoke as before, recognised his mother and smiled to her. I have frequently met with similar cases, and they may serve to warn us not to lay too great stress, in cases of chronic hydrocephalus, on there being necessarily complete arrest of the intellectual development.

The power of motion in the upper limbs is usually not much affected. But we may perhaps notice that the children on attempting to grasp anything, make incongruous movements which have a distinct resemblance to those of chorea. On the other hand paraplegia is very often present, both legs being either completely paralysed, or at least unable to bear the weight of the body. Standing and walking—and often even sitting—unsupported, is out of the question, and most of the children, if you try to set them on their feet, cross their legs (which are loosely hanging down) without attempting a single step. But to this rule, also, there are exceptions; and cases have been published in which the power of movement in the lower extremities was almost perfectly retained. Convulsive attacks of various kinds, spasmus glottidis, strabismus, nystagmus, convulsive contraction of the body with a tendency to fall forwards, and, finally, general epileptiform fits and contractures, are often added. At the same time, all the organic functions—respiration, circulation, and digestion—may remain perfectly normal for years. Yet the state of nutrition suffers considerably, and the children finally become wasted, which causes the size of the head to become all the more striking by contrast. In a child of six months a large bed-sore formed on the right parietal bone and ear owing to the heavy head lying continually on the right side. Now, although most of these children die during the first years of life from atrophy and marasmus, or in a

convulsive fit—still you must give a cautious prognosis as to the duration of the disease. Apparently desperate cases have in not a few instances attained the age of 5—6 years and more; and we have also examples of the patients living to adolescence, or even much longer. Rarely cases have been observed to end in the fluid rupturing into the space between the dura mater and arachnoid, or even externally through the vault of the cranium. I have, however, no personal experience of this ending.

At the post-mortem we find, firstly, a more or less marked thinning of the cranial bones due to the pressure of the distended brain, and it may often be recognisable during life by palpation. In a child of 9 months, who was not backward to any extent in his intelligence, and did not show anywhere a trace of paralysis, I found this thinning very marked, the diploë gone, and the bone so transparent that we could make out distinctly through it the colour of the dura mater and its blood-vessels. Further, the fontanelles and sutures gape widely, and the spaces of the latter are closed by fibrous membrane of a finger-breadth or even more in width, which contain scattered points of bone. The cerebrum consists of two more or less flaccid undulating sacs—the enormously distended lateral ventricles filled with serous fluid and surrounded by a shell of condensed brain substance, which is sometimes only a few centimetres in thickness. The amount of fluid averages from 9 to 18 oz., but may even reach 2 pints or more. Albumen is generally only present in small quantities. In the surrounding shell to which the brain-substance of the hemispheres is compressed, we still see the limits of the grey and white matter. Both the convolutions and the large cerebral ganglia are flattened by the pressure. The third and fourth ventricles are frequently dilated and filled with fluid. I have also seen the fifth ventricle repeatedly affected by this dropsical dilatation. We almost always find the central structures (corpus callosum, fornix, &c.) of unusually firm consistence if the brain be examined in as fresh a condition as possible. The ependyma of the ventricles generally has a finely granular surface, and appears as if dusted over with extremely fine, grey, transparent granules, which under the microscope are found to be due to hypertrophy of its tissue. Only seldom do we find fragments of

fibrinous lymph obstructing the foramen of Monro, and consequently interfering with the communication of the ventricles with one another, or inflammatory thickening of the choroid plexus. The degree of the morbid changes described vary, of course, very much; the dilatation of the ventricles and the thickness of the compressed brain-substance presents the greatest differences. The following case may serve as an example of an unusually extreme condition :—

> Anna P., 3 months old, admitted March 26th, 1877, with chronic hydrocephalus. Tolerably well-nourished. Circumference of the head 45, longitudinal diameter 25, transverse diameter 27 centimetres. Eyeballs directed downwards. No nervous symptoms noticed. The child took the bottle quite normally, cried much and lustily, and its whole behaviour differed in no way from that of a healthy child. Collapse and broncho-pneumonia, commencing on April 3rd; death on 7th.
>
> *P.-M.*—After removal of the upper portion of the very thin dolichocephalic cranium, and division of the dura mater, we saw into a ventricle completely filled with fluid, at the bottom of which an elongated lump represented the remainder of the brain. On closer examination we found that the cerebral hemispheres had almost entirely disappeared. Under the dura mater, which had remained normal, there appeared—only in certain places—very thin plates, bands and strips, covered by a membrane resembling the pia mater—all that remained of the hemispheres which had disappeared and were replaced by clear watery fluid filling the whole cranial cavity. The amorphous mass on the floor of the cranium consisted of the remainder of the great cerebral ganglia, and the cerebellum and spinal cord were connected with it in the normal way. These parts, as well as the cranial nerves and vessels, were quite unaffected.

Although in this child the compression of the substance of the hemispheres had gone on until they had almost quite disappeared, we yet see all the functions acting normally, and the whole condition differing in no way from that of a healthy child of the same age. Exactly the same condition was found at the post-mortem of another case, in which the power of motion was just as little interfered with. A "psycho-motor centre" was certainly out of the question here. The cases, therefore, furnish a clinical proof of the view that the actions of the newborn child must be regarded as involuntary (reflex, automatic).

The pathology of chronic hydrocephalus is still by no means thoroughly explained. It is certain that in a number of cases

the disease is congenital, *i.e.* developes during fœtal life. Under these circumstances a serious obstruction to birth may arise, which must be removed by operation. In these cases we sometimes find various kinds of arrested development—defects of the corpus callosum, fornix, &c.; likewise spina bifida, club-feet and hands, &c. Much more frequently, however, the children come into the world apparently healthy, and it is some months after birth before the relatives are struck by the unusual growth of the cranium. What, then, is taking place here? The peculiar granular hypertrophied condition of the ependyma —which can sometimes even be torn off from the wall of the ventricle in tough strips—points to the occurrence of an insidious inflammatory condition of it, which either begins in fœtal life, or else not until some time after birth, and is so little noticeable that the first sign of the disease is the distension of the head by the steadily increasing pressure of the fluid in the ventricle.[1] This inflammatory theory does not, however, fit all cases, because the granular condition of the ependyma may be absent, and with it everything that is in favour of an irritative process having existed within the ventricles. In the same way causes of compression (*e.g.*, tumours, of which we have already spoken on p. 284) are met with in a very small number of cases; and least commonly in those that are congenital, or have arisen very early, and we have then nothing left but to content ourselves with the unsatisfactory supposition of a "malformation," or of an excessive "secretion of cerebro-spinal fluid." Those who support the inflammatory theory usually go upon the rare cases of hydrocephalus which may arise in somewhat older children, *i.e.* about the second half of the first year, after symptoms of meningitis. I have myself seen some cases of this kind, but they are only of value as proof when the position of the fluid within the ventricles and the alteration of the ependyma is verified by a post-mortem. Should this not take place, we remain in doubt as to whether the case was really one of hydrops ventriculorum, or of an accumulation of fluid between the membranes (hydrocephalus meningealis *sive* externus) — to distinguish

[1] I cannot find any connection with congenital syphilis such as is occasionally assumed; at least I have not seen the slightest effect from specific treatment in the very few cases of chronic hydrocephalus in which syphilis could be ascertained (Sandoz, *Revue mens*, Januar, 1847, p. 42).

which, clinically, from internal hydrocephalus is a matter of the greatest difficulty. I always think that the absence, or at least the slight development of the cerebral symptoms—especially a satisfactory state of the mental powers—are points in favour of a diagnosis of hydrocephalus externus.

Some French writers (Legendre, Rilliet and Barthez, and others) have described this hydrocephalus meningealis, as I have already remarked (p. 272), as the second stage of "hæmorrhage into the cavity of the arachnoid." According to our present view, we have to do here not with a primary hæmorrhage, but with an inflammatory process accompanied by blood-extravasation on the inner surface of the dura mater (pachymeningitis), which occurs pretty often in children with moderate severity and extent, though not so commonly as in old people. I have under various circumstances found more or less thick fibrinous deposits, coloured with blood, on the inner surface of the dura mater, along with the accumulation of a varying amount of reddish serum between this and the pia mater, although during life there had been no definite series of symptoms suggesting the presence of such a condition. There were present only the ordinary symptoms of meningitis—drowsiness, strabismus, dilatation of the pupils, cervical rigidity, screaming, &c., and it is proved by experience[1] that these also may be absent even in extreme cases. On the other hand, I have hitherto met with only two cases of pachymeningitis which corresponded to those observed by the French writers, *i.e.*, where the mass of the exuded fluid had by its pressure distended the cranial capsule so as to produce hydrocephalus.

Otto R., 10 months old, extremely atrophic and anæmic, admitted September 5th, 1883, with a considerable degree of hydrocephalus (very large head, widely open fontanelle, intelligence backward) and rickets. Death on September 15th.

P.-M.—Vault of the cranium strikingly large, especially the parietal bones which corresponded in size to those of a child of 2 or 3 years. On sawing through the skull, about 10½ oz. of clear yellowish-red fluid escaped, although the brain itself was not in the slightest degree damaged. On removal of the vault of the cranium a third membrane was found between the dura and pia mater which enveloped the whole brain with the exception of

[1] Moses, *Jahrb. f. Kinderheilk.*, 1873, vi.—Rilliet et Barthez, *loc. cit.*, and others.

the posterior fossa, and could be raised up from the internal surface of the dura mater as a transparent, colourless and but slightly vascular membrane. Pia mater delicate and thin all over. In some places adherent to the false membrane. Vessels empty of blood. The brain did not fill the cranial cavity, but, after the fluid had been run off, the convexity appeared to be several centimetres from the bones. Ventricles of moderate size; empty, brain very anæmic, otherwise unchanged. All the other organs normal with the exception of rickety changes in the bones, and perisplenitis adhesiva.

In this case, therefore, the origin of the hydrocephalus in pachymeningitis was proved beyond doubt by the post-mortem (false membrane, blood-stained serum). Likewise in a girl of six months with extremely hydrocephalic cranium, backward intelligence, and contractures of the muscles of the eyes and extremities, we found after death "pachymeningitis pseudomembranacea hæmorrhagica," with fibrous thickening of the arachnoid and pia.

In the following case, which ended in recovery, the inflammatory origin of the hydrocephalus is beyond a doubt, but it must remain unsettled whether the fluid was in the ventricles or in the meningeal space, or in both places at once.[1]

Paul W., 3 years and 2 months old, brought to me for first time on February 14th, 1861. Formerly healthy. During last 8 weeks complaints of pain in the head and neck, tendency to head-retraction, irregular rise of temperature in the evening, pallor and emaciation. On examination we found: inability to hold up the head, which was retracted; pain in the neck on pressure and movement; frontal headache. Walking and standing impossible, but no paralysis. Anorexia and obstruction. In the afternoon moderate fever, pulse 96—100 and regular. Antiphlogistic treatment (leeches and inunctions of mercurial ointment into the occiput and neck) produced by the 19th an improvement in the posture of the head. But on the 20th, got worse again: vomiting, violent pain in the forehead and neck and marked retraction of the head (calomel, gr. ½ thrice daily). Exacerbations of the pain occurred, especially between 11 and 3 P.M., along with rise of temperature (blister the size of half-a-crown on the occiput). Slight improvement from the use of quinine, but vomiting, grinding of teeth during sleep, and a certain degree of incontinence of urine appeared for the first time. It was not till March 22nd that the fever and attacks of pain had quite disappeared and the head could then be

[1] *Beitr. zur Kinderheilk., N. F.*, S. 28.

moved forward; but it now appeared markedly enlarged, and on examination we found diastasis of the parietal bones. These appearances increased daily, so that on the 26th the child was obliged to wear one of his father's hats instead of his own. The sagittal suture gaped, and yielded somewhat on pressure, although the mother had observed that this as well as the other sutures had been firmly closed in the second year of life. Weak pulsation in the position of the fontanelle. Intelligence quite normal, the right arm weaker than the left, which was almost exclusively used. Pulse regular (calomel, gr. ¼ twice daily and ung. hydrarg., grs. vi. to be rubbed into the scalp daily). After 21 days (April 16) the diameter of the head unchanged, but it was held well up and no longer retracted. The right arm was now easily moved. General health unaffected. (Treatment on the same lines continued for 4 weeks with codliver oil, a dessert-spoonful twice daily.) In the middle of May the child began to walk, and on June 11th, excepting for the large size of the head, every trace of the disease had disappeared. The sutures already showed commencing ossification. In May, 1863—*i.e.*, 2½ years after the beginning of the disease—I again saw the child, in perfect health; all the cranial sutures had become ossified.

What seems to me especially remarkable in this case is the fact that even at the age of three, when the ossification of the sutures and fontanelles was already completed, the intra-cranial pressure was sufficiently strong to force these once more apart. I have seen the same thing happen, but limited to the coronal suture, in a boy of 7, in whom hydrocephalus had developed with considerable increase of the size of the head, as a result of a fall two years before. Goelis, Rilliet and Barthez, and others mention this very rare occurrence. Perhaps this very circumstance, which relieves the brain from a part of the pressure, must be looked upon as favourable in so far that it may prevent the onset of serious cerebral symptoms. At any rate, our case shows that even when the amount of fluid present is very large, re-absorption and final recovery is still possible—if the starting point of the disease was meningitis. It is very hard to conceive in what manner the empty space, which must be created by the re-absorption of a considerable amount of fluid in the ventricles, can possibly be filled up, since the brain substance which had been compressed to a mere shell could hardly expand into its former bulk. This consideration is, to my mind, in favour of the above-mentioned case being one of external hydrocephalus from pachymeningitis, the brain, which

was only slightly compressed from the outside, having no difficulty in again expanding after the re-absorption of the fluid. The ossification of the fibrous tissue of the sutures then took place gradually, spreading partly from the margin of the cranial bones and partly from Wormian bones. I found exactly the same condition in a Russian girl, nine years old, of excellent mental powers, whose head had enlarged very greatly after an attack of meningitis in her second year but finally became completely ossified.

The case I have given illustrates to you the treatment which you should employ in this disease. At first local blood-letting is to be recommended by means of a few leeches applied behind the ears or to the temples, also iced compresses to the head, and purgatives, the best being calomel (gr. $\frac{1}{4}$—$\frac{1}{2}$) several times daily. Later on, when the inflammatory stage is passed and our chief object is to bring about the absorption of the fluid, I should recommend the continued administration of small doses of mercury, inunction of blue ointment into the head and neck (grs. x. in the day) and painting with iodoform-collodion (1 : 15). Also iodide of potash (Form. 13) continued for a long time is useful in such cases. Antiphlogistic treatment, however, is only suitable for the very rare cases in which we have ourselves had the opportunity of observing the early inflammatory stage. On the other hand, I consider chronic hydrocephalus of the ventricles as invariably incurable when it has caused considerable increase in the size of the head. The results which Goelis thinks he has obtained from his mercurial inunctions, appear to me, from my experience, extremely doubtful and founded on fallacy. Read, for instance, his fourth case,[1] which was said to be completely cured after 30 days' treatment, and which certainly was not hydrocephalus at all, but only diphtheritic paralysis. I, at any rate, have obtained no results whatever with Goelis's method, nor from iodide of potash nor from painting the head with tincture of iodine or with iodoform-collodion, and I can promise you no better success from the compression of the cranium by strips of sticking-plaster, or from puncture through the fontanelle (to one side of the middle line). In the cases where these proceedings have been

[1] *Prakt. Abhandl. über die vorzüglicheren Krankheiten des kindlichen Alters*, ii., S. 214.

of use (and they were exceptional) it is possible, for the reasons given above, that there may have been only external hydrocephalus.[1] Those who have an inclination to operate may gratify it, as the danger of meningitis is not very great; but one will do well to abandon from the very beginning any hope of a radical cure. In five cases in which we performed puncture, it had no effect whatever.

Gustav P., 3 months old, admitted into the hospital on July 13th, 1878. A few weeks after birth, increase in size of the head, and spasmodic twitching of the eye-muscles. On admission distinct hydrocephalus. Circumference of head 40½ ctm., longitudinal diameter 24 ctm., transverse diameter 23 ctm. On the 18th, puncture of the right lateral ventricle with a hypodermic syringe and evacuation of more than an ounce of a slightly albuminous fluid. Pressure applied immediately after by strips of sticking-plaster. By the 21st, no after-symptoms. On this date a second puncture; introduction of a medium-sized exploring canula one inch from the middle line in the lateral angle of the large fontanelle and right into the left lateral ventricle, followed by the application of Dieulafoy's aspirator. 4¼oz. of fluid removed. Convulsions in the course of the following night. Death on 22nd.

P.-M.—Chronic internal hydrocephalus, also fluid between the dura and pia mater. No trace of the puncture to be found. No meningitis.

A child of one year, admitted on June 21st, 1883, with (congenital) chronic hydrocephalus and rickets. Circumference of the head 59, transverse diameter 21 ctm. At the parents' request 6 punctures were made in the coronal suture with an aspirating needle about three finger-breadths to the right or left of the middle line.

First puncture on 23rd June. 3½oz. of clear fluid removed, which (according to Prof. Salkowsky's examination) had a neutral reaction, remained clear on boiling, but when it was heated and acetic acid and sod. chlorid. were added, it became cloudy and showed traces of albumen; it gave no sugar reaction. Convulsions in the evening lasting 3 hours; temp. up to 103·5° F. Fever, contractures and tremor lasting 9 days; after that the child seemed well.

Second puncture on July 6th. About 7 oz. removed. The fontanelle subsided considerably.

Third puncture on July 12th. About 7 oz. removed. 13¼

[1] Rehn, "Ueber einfache chronische Hydrocephalie im ersten Kindesalter": *Verhandlungen d. Congresses f. innere Med.*, iv.

drachms of the fluid mixed with 15 minims tinct. iodi injected through the trocar. The child remained well, with the exception of slight rigidity of the limbs.

Three other punctures on July 19th, August 1st and 9th. Each time $8\frac{3}{4}$—$10\frac{1}{2}$oz. of fluid removed. Injection of tinct. iodi as above. No cerebral symptoms. Circumference of the head unchanged. After the 15th, broncho-pneumonia and diarrhœa. Death on 29th. Post-mortem refused.

I shall take this opportunity of saying a few words on acute hydrocephalus, which formerly occupied such an important place in pædiatrics. The vast majority of the cases described under this name are really cases of tubercular meningitis, and I shall discuss them later on in considering it. Far more rarely, acute hydrocephalus accompanies simple basic meningitis, spreading into the ventricles along the choroid plexus. If one subtracts these cases there remain but few in which you can speak clinically of a rapidly-recurring exudation into the ventricles, or between the meninges. One finds, to be sure, at the post-mortem of many children, effusion of serum with moderate distention of the ventricles, which, if one may judge from the symptoms, can only have taken place within a short time of death—a few days or even less; and it is especially children with acute miliary tuberculosis, Bright's disease, and scarlatinal dropsy who most frequently present this form of acute hydrocephalus. Such cases, however, cannot be diagnosed with certainty, for exactly the same symptoms may be caused, without accumulation of fluid in the ventricles, by œdema of the pia mater, or of the brain itself—which is not uncommon under the same circumstances. Coma, convulsions, fatal issue within a few hours or days—all these are not in themselves sufficient to form a special disease, as, for example, Goelis has endeavoured to make out with his "Serous Apoplexy" (hydrocephalus acutissimus). Let us rather admit that our powers are, as yet at least, limited here, and that acute serous effusion within the cranium—whether into the ventricles, between the membranes, into the pia mater or into the substance of the brain—may be suspected from the circumstances in which the patients die, but cannot be diagnosed with any certainty from the above-mentioned cerebral symptoms.

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

We learn from post-mortem examinations that the amount of blood contained in a child's brain varies very much—that all conceivable degrees occur, from a slight filling of the vessels of the pia mater and a pale anæmic colour of the grey substance to the most minute injection of the vessels with numerous points of blood seen on section of the brain. It is, however, vain to attempt to connect these different states of the vascular system with definite symptoms. One can only smile when some writers go so far as to pretend to distinguish even clinically hyperæmia of the pia mater from that of the brain. We must also always remember that hyperæmia found post-mortem may just as well be the result as the cause of fatal cerebral symptoms—*e.g.*, of very violent and protracted convulsions. Even pure reflex convulsions may, by the accompanying interference with the respiration, finally occasion engorgement of the cerebral veins, ending in œdema of the pia mater and brain, with serous exudation into the ventricles or between the dura and pia mater.

Hyperæmia of the brain and its membranes may arise, like any other hyperæmia, either from increased blood pressure in the arteries or from an engorged state of the cerebral veins. The former we may expect in hypertrophy of the left ventricle and as the preliminary state of inflammatory processes (meningitis), along with which it falls to be considered clinically. Likewise, local causes of irritation (tubercular masses or tumours) appear capable of causing "meningitic" symptoms (fever, vomiting, drowsiness, convulsions) by exciting hyperæmia from time to time in their immediate neighbourhood. These symptoms rapidly subside either spontaneously or under antiphlogistic treatment; but they may also, by their frequent repetition, lead to "inflammatory-hæmorrhagic" softening or to encapsulation from proliferation of the connective tissue. Thus far we stand on the firm ground of pathology. But we not uncommonly meet with cases in practice, which—when we take all the circumstances into account—can hardly be explained otherwise than by arterial hyperæmia of the brain, although the exact mode of its occurrence is not always quite clear and there is fortunately no opportunity for anatomical confirmation. Among the causes of this condition

which here demand our consideration, injuries are those most frequently met with. The child may become torpid or completely unconscious immediately after a fall on the head. We do not yet know for certain on what state of the brain the symptoms depend which are usually known as "concussion of the brain." In three such cases which I have published elsewhere,[1] the children were perfectly well immediately after the fall, and the symptoms only set in after some hours or days. These were as follows:—continuous headache, apathy, drowsiness, yawning, change of colour, restlessness at night, anorexia, repeated vomiting, and fever, the pulse rising to 140—160 in the minute but remaining regular. One of these children suffered at the same time from attacks of night-terrors, so that he jumped out of bed and ran to the light (probably owing to terrifying dreams); and these recurred from time to time for some weeks after recovery. The rapid onset of these symptoms after an injury to the cranium, and especiaily the surprisingly rapid result of antiphlogistic treatment, make the diagnosis in this case certain, I think. The application of a few leeches behind the ears (the bites of which I did not allow to bleed afterwards, in order to avoid excessive loss of blood) was sufficient to give considerable relief to the symptoms. The hæmatophobic line of treatment which has come into fashion in our time is here to be avoided. We can draw blood directly from the cranial cavity by means of the emissaria Santorini, and we must not hesitate to do so; because these preliminary symptoms if neglected may result in regular meningitis. At the same time we must apply an ice-cap continuously to the head and produce copious evacuations by giving calomel or mist. sennæ co., and syrupus rhamni (Form. 7). Under this treatment I have seen complete recovery after 36—48 hours. In the two following cases, also, hyperæmia due to cerebral concussion seems to have been the cause of the symptoms.

Boy of 9 years, remained unconscious for 24 hours after falling from a vehicle on to the back of his head. No wound discoverable. Eyes fixedly directed to the right, pupils did not react. No fever; temperature 98·2° F. Pulse small, 100 and irregular; repeated vomiting. After 24 hours, headache, frequent vomiting and irregularity of pulse remained. Otherwise

[1] *Beitr. zur Kinderheilk., N. F.*, S. 2.

well. These symptoms lasted for a whole week and then disappeared, leaving the child perfectly well. Treatment:—4 leeches behind the ear, ice-cap, calomel.

Boy of 6 years, after a fall from a high stair on April 29th, 1881, loss of consciousness and vomiting, lasting through the night. Next morning return of consciousness, but apathy and double vision. Œdema, ecchymoses and desquamation of the skin over the right half of the face, a considerable cephalhæmatoma over the right parietal bone. Pulse 84, somewhat irregular. Still occasional vomiting; otherwise well. Continuous application of an ice-cap, repeated purgatives. Recovery by 12th May, but a slight thickening is still noticeable in the situation of the cephalhæmatoma.

In this case I thought that I might omit local blood-letting on account of the severe hæmorrhage which had taken place from the vessels of the pericranium. As a matter of course this must generally be omitted while the actual symptoms of concussion (unconsciousness, great pallor, small pulse, coldness of the skin) last, and stimulants are rather to be used.

If you consider that the symptoms of hyperæmia of the brain occur after a fall on the head only in a comparatively small number of children, while the majority remain quite free from them or are only slightly stunned, you may assume that besides the severity of the concussion an individual pre-disposition to dilatation of the small blood vessels is an important factor. As a matter of fact, a certain number of my patients had shortly before recovered from whooping-cough or chronic pneumonia, or else came of a tubercular family. The conformation of the cranium must also be considered; for little children with membranous fontanelles and sutures seem generally to escape the bad effects of concussion more easily than older ones, whose cranial bones are already completely ossified.

In a smaller series of cases we see symptoms of hyperæmia of the brain come on without any discoverable traumatic cause (and we may even be able to exclude such causes entirely), especially in children about the period of the first dentition. These symptoms are—fever, drowsiness alternating with great restlessness, bad temper, apathy, frequent convulsive movements of the body, inability to hold up the head, tense and strongly-pulsating fontanelle, elevated temperature of the head, and likewise vomiting. I only mention this as a fact, without being able to prove that these

symptoms depend on dentition; but I would remind you that we often find along with it extreme hyperæmia of the buccal mucous membrane, increased secretion of saliva, erythema and papules on the skin and the face, and catarrh of the conjunctiva and bronchi. Purgatives (small doses of calomel) and cold compresses to the head are in these cases usually sufficient to remove the symptoms within a few days. Still, we do not always attain our end so easily. Every physician has had cases in which the symptoms have gradually got worse and assumed the characters of meningitis by the addition of convulsions, head-retraction and coma.

Finally, excessive mental exertion must be mentioned as a source of cerebral hyperæmia. This occurs as the result of over-excitement of an organ which is in a state of development. Although under these circumstances the hysterical symptoms, already considered, and neuralgic headaches are wont to occur more frequently, still there are plenty of cases in which hyperæmic symptoms also have made their appearance after mental exertion. I have elsewhere[1] published the case of a boy of 9, who from such a cause was affected not only by violent headache and photophobia, but also by giddiness, anorexia, nausea, sighing, constipation, pains in the neck, intermittent pulse, and staggering gait. Emetics and quinine had no effect whatever, but the application of five leeches and of an ice-bag to the head and the use of purgatives were followed by rapid improvement.

The second form of cerebral hyperæmia is caused by mechanical engorgement of the intracranial venous system. Valvular disease of the heart, with dilatation of the right ventricle, compression of the large venous trunks by enlarged glands inside the thorax or in the throat, but especially thrombosis of the cerebral sinuses, may gradually give rise to this hyperæmia; and extreme cardiac debility, from exhausting diseases, may cause it in a more acute form. In cases of this latter kind, anæmia of the brain is often assumed during life as the cause of the symptoms. As a matter of fact the debilitated cardiac muscle is unable to drive the normal amount of arterial blood into the small cerebral arteries, and the consequent retardation of the circulation causes a venous engorgement which finally leads to œdema of the pia mater and serous effusion into the ventricles. The clinical picture of "hydro-

[1] *Beitr. z. Kinderheilk., N. F.*, S. 8.

cephaloid" sketched by Marshall Hall is made up therefore of the symptoms of arterial anæmia along with those of venous hyperæmia of the brain. Its characteristic symptoms are: advancing apathy and drowsiness, half-closed eyes, flattening or depression of the great fontanelle, opacity of the cornea from fragments of mucus and drying up of the tissue, great weakness of the pulse and fall of temperature (especially at the extremities) —symptoms which depend only partially on venous hyperæmia of the brain, and partially on the cardiac debility and general collapse. The development of this series of symptoms is caused especially by continuous diarrhœa or very acute cholera infantum.

Child of 6 months. Diarrhœa for nearly 3 months. Admitted on October 3rd, 1873, in a state of extreme collapse. Drowsy, with waxen pallor; eyes sunk in, staring, and sometimes turned upwards. Thready pulse. During the next few days fall of temperature to 96·8° F. in spite of stimulating treatment; pulse almost imperceptible, dimness of both corneæ; coma. Death on October 5th. *P.-M.*—Enlargement of Peyer's patches. Catarrh and thickening of the mucous membrane of the large intestine, especially in the descending colon and rectum, with numerous follicular ulcers. Fatty liver and fatty degeneration of the renal epithelium. Heart and lungs normal. All veins of the pia mater enormously engorged, pia mater œdematous. Numerous points of blood on section of the brain. All the sinuses quite unaffected.

The treatment of such cases must not, of course, be depressing, or else it would only further diminish the heart's energy, thereby increasing the venous engorgement of the brain. Our chief endeavour must be rather to strengthen the heart's energy in order to restore the circulation as soon as possible to its normal condition. Repeated doses of wine (a teaspoonful of Hungarian wine, port or sherry every 1—2 hours), warm baths (95° F.) rendered stimulating by the addition of mustard, with cold compresses to the head or douching of it with cold water, are to be used. We must of course treat by suitable remedies any source of collapse that still continues; in most cases this is diarrhœa. In many cases, however, this has already ceased by the time the cerebral symptoms make their appearance. We may, then, at once attempt to strengthen the heart by stimulants. According to my experience, the best of

these is camphor (grs. $\frac{3}{4}$—grs. iii. every 2 hours, according to age, in the form of powder or emulsion, Form. 14). Should neither camphor nor wine be sufficient to keep the heart going, I do not expect to succeed with any other remedies. Musk, and especially the much-praised preparations of ammonia, I have found practically useless. Milk and strong beef-tea, yolk of egg beaten up with wine must be given to the child at short intervals. The prognosis, however, is always extremely serious, and a large number of these children, in spite of all our exertions, die in a state of coma often with convulsions.

The retardation of the venous blood-stream leads not unfrequently to complete stagnation and coagulation of the blood in the large cerebral sinuses—to "marasmic" thrombosis. We most frequently find the longitudinal, less frequently the other sinuses, filled with more or less decolourised tough thrombi, which may be followed to a greater or less distance into the communicating veins, and must considerably increase the venous engorgement in the brain and pia mater as well as the danger of serous effusion. Any other sinus-thrombosis acts, of course, in the same way, whether it is caused by compression of the sinus, or by inflammation spreading from the neighbouring cranial bones. The petrosal and transverse sinuses especially are exposed to the influence of the adjacent petrous bones when carious, and the thrombi in them occasionally extend far into the jugular vein. That this process may take place without any change being visible on the free surface of the dura mater, is proved by the following case :—

Girl of 9 years, admitted into the hospital on February 2nd, 1877. Otitis media since her first year; perforation of the membrane, through which one could see a red pulsating surface covered with pus. Constant severe headache; no fever. Ear washed out under chloroform. In the night between the 4th and 5th February, suddenly great restlessness, delirium and screaming. On the 5th, coma; pulse 116, regular; temp. 101·3° F. Next day continuation of the same condition, convulsive contraction in the limbs on the right side. Pulse 132, small; deep coma; temperature 100·4° F.; resp. 60. Copious perspiration. Death. *P.-M.*—Marked œdema of the brain, pia mater normal. Transverse sinus and right inferior petrosal sinus containing thrombi. The right petrous bone carious. The caries extends to close under the dura mater, in which situation there was an abscess the size of a pea. The dura mater itself was perfectly unaffected.

Parenchymatous nephritis. A portion of the ileum, nearly 30 inches long, dark-red in colour and covered with a diphtheritic membrane. Liver fatty.

I have frequently observed the fact that caries of one of the cranial bones, especially the petrous, may extend so as to reach close up to the dura mater without affecting that membrane itself. It remains for a long time unaffected and glistening, and yet the neighbouring petrosal sinus may be the seat of a thrombosis, which is to be explained, either from small thrombi having been carried into it from the veins in the bones, or from their having projected into it. The sinus-thrombosis which is occasionally observed as the result of severe suppurating eczema capitis is also to be explained in the same way (continuous formation of thrombi through the emissaria Santorini).

Much trouble has been taken to render the diagnosis of sinus-thrombosis possible. Gerhardt and Huguenin lay especial stress on the fact that in thrombosis of the transverse sinus, or at the commencement of the internal jugular vein, the external jugulars appear less filled on the affected than on the healthy side, because their contents are more easily discharged into the empty internal jugular. Again, in thrombosis of the cavernous sinus, the engorged condition of the ophthalmic vein is said to be indicated by venous hyperæmia of the fundus of the eye, slight exophthalmos, and œdema of the upper lid or of the whole side of the face. Although I have repeatedly looked out for the symptoms recorded I have never been able to convince myself that they really occur,—perhaps because (as Gerhardt himself admits) the cervical veins do not always present the degree of turgescence necessary for making out the difference between them. Still it appears to me that careful examination of the veins of the throat and eyes, and close examination and careful noting of any unilateral œdema in the face, promise more for the diagnosis in cases where there is a suspicion of sinus-thrombosis than do the signs which these writers give for thrombosis of the pulmonary artery. That this condition and its results (hæmorrhagic infarct) may occur from thrombosis of the sinus by means of embolism, is indeed beyond doubt, and it has also been proved anatomically; but in a child the diagnosis of this embolism under the circumstances in which it occurs (*i.e.* when various kinds of cerebral

disturbances are present) is so difficult that it is only in very exceptional cases that we can establish during life that it is connected with sinus-thrombosis. Under these circumstances treatment is of course out of the question, since even in a case where the diagnosis has been put beyond doubt, no one would expect to be able to remove the thrombosis.

XVII. *Tubercular Meningitis.*

This is one of the commonest and most fatal of the diseases affecting childhood. As soon as you observe the first certain signs of it, you may confidently foretell a fatal issue; and, although in doubtful cases the physician leaves no stone unturned in order to arrive at a sure diagnosis, this is not, unfortunately, because he has any successful treatment in view, but only to assure himself of the certainty of the sad issue for which he has to prepare the patient's friends. If we compare the relatively numerous successful results given by the authors of the older works on "hydrocephalus acutus" with our own, we see at once that physicians formerly described and treated under that collective name a number of different morbid conditions (simple cerebral hyperæmia, meningitis simplex, typhoid). Now-a-days, however, when our diagnosis has become more exact, and we limit our conception of acute hydrocephalus to tubercular meningitis, we can only look back with a smile to the modes of treatment which were recommended and in their time held in high estimation. The incurability of this form of meningitis is indeed expressed in the very designation "tubercular." Meningitis of this nature is fatal, from its combination with tubercle of the pia mater and of many other organs. It is not a merely local disease, but one which extends over many important parts—in a word, it is a "terminal" form of tuberculosis.

The description of this disease is difficult on account of the numerous variations in its course; and, in spite of the large amount of material at my command, I can scarcely hope to be able to give you a complete, comprehensive, and clear account of it. I think it will be most suitable to describe to you first of all the usual "classical" form of the disease, as I may call it, and later on describe its varieties.

The real outbreak of the disease is in many cases preceded by a premonitory stage, which may last for weeks or even for months. The child becomes emaciated and flabby; the mother notices this in washing him, and cannot account for it. The general health is often meanwhile unaffected, while in other cases various derangements occur—capricious appetite, lassitude, varying temper, irregular rises of temperature—indefinite symptoms the significance of which, in spite of the most careful examination, the physician is at a loss to estimate. These symptoms announce the slow development of tubercle in various organs; and therefore, in taking the history in such cases we must always investigate whether there is a hereditary tendency to tuberculosis; for the discovery of this may serve to shed some light on the obscure significance of the symptoms. We must not, however, forget that a family tendency to tuberculosis is by no means necessary; for hypertrophy and caseation of the bronchial and mesenteric glands may exist as the result of chronic catarrh, whooping-cough, measles, typhoid, or repeated attacks of diarrhœa, and may finally form a centre of miliary tubercular infection. Caseous processes in superficial lymphatic glands or in bones (spondylitis and osteomyelitis) may have a similarly important influence. We must hold to these facts which are the result of innumerable well-established observation from the clinical point of view, and leave their connection with tubercular bacilli to be determined by further investigations. It can hardly be doubted that invasion of the bacilli may take place from the intestine, the lungs, the skin (eczema), or the nasal mucous membrane, and finally lead to meningeal tuberculosis. In this matter the nose deserves special attention,[1] as its lymphatic spaces communicate with the meninges through the ethmoid bone. Moreover, the above mentioned preliminary symptoms are not at all constant. In spite of careful investigations I have often enough been told by mothers that their children had been perfectly well up to the time of the actual commencement of the disease; and their thriving, well-nourished appearance supported the statement.

[1] *Cf.* Demme's case (*Klin. Wochenschr.*, 1886, No. 15), in which a tubercular ozæna with discharge containing bacilli preceded the meningitis by a long time, without any hereditary predisposition, and without there being any caseous deposit found.

The onset of the disease occurs almost suddenly, with complaints of headache, especially in the forehead, and with vomiting—usually repeated several times during the first few days, and sometimes occurring after every attempt to eat or drink. Definite characters have been ascribed to this kind of vomiting, but I cannot confirm them. I have seen it take place in the upright as well as the horizontal position, sometimes without warning and sometimes accompanied by much retching. I cannot therefore see any real difference in the characters of cerebral vomiting from that which is gastric. It is just this point in the diagnosis, however, that we are first called upon to consider. The symptoms of the first half or whole week are in very many cases so like those of a slight case of gastric fever, that many experienced physicians who have seen numbers of such children die are by no means secure from such mistakes. The general apathy, the loss of inclination for play, the headache, the tendency of the head to become retracted, and especially the inclination to lie down, the more or less thickly-coated tongue, the loss of appetite with vomiting and constipation, and, finally the irregular rises of temperature—all of these symptoms are so equivocal that we may be in doubt whether the case is one of commencing meningitis, or some feverish stomach-complaint, or whether it is not even the commencement of typhoid fever. In tubercular meningitis the children often show a striking persistence in picking at their lips, boring in their nose and rubbing their eyes; but even this peculiar and inexplicable symptom is common to all the conditions just mentioned. As long, therefore, as you are not quite certain you must beware of telling the parents that the matter is one of no importance, and that it all arises from nothing but a "bad stomach"—a mistake which the inexperienced readily fall into. It is much better to leave the possibility of cerebral disease open, for parents never forgive a physician for a false prognosis, even although he afterwards tries to shield himself by saying that the "stomach-complaint" has finally gone on to hydrocephalus.

The uncertainty, however, generally lasts—for the experienced physician at least—only a few days. By the end of the first week at latest more unmistakeable signs of the danger threatening generally set in, and cannot but attract your attention. Among these I reckon especially a frequently recurring deep sighing—

which has almost never deceived me—and the characteristic alteration of the pulse—both of these being of course caused by irritation of the origin of the vagus at the base of the brain. The pulse becomes slower, and at the same time irregular, likewise unequal in the strength of its individual beats. This symptom I regard as decisive under the circumstances I have described even if its appearance is only transient. There is scarcely any other disease of children in which the pulse varies so much in its character as it does in this. In the course of one day its rate changes repeatedly and considerably. Slight movements are sufficient to cause an increase of 20 or more beats, while the varying temperature—to which I shall presently return—has no influence on the pulse. The rate varies much between 96 and 120, and occasionally falls to 80, 72, and even less. But although this symptom is so important, we must also bear in mind that just the same may also occur in trifling stomach complaints, owing to reflex irritation of the vagus. Of this, however, I have only seen one instance, viz., the following case:—

> In a boy of 9 years, whom I treated in April, 1867, at the beginning of an attack of febrile dyspepsia, the pulse fell, on the day following the use of an emetic, from 120 to 80, even when awake and in the sitting posture; during the next few days to 52—48, and presented at the same time marked intermissions. The persistent frontal headache, sleepiness, and indolence made me very anxious; but complete recovery of the gastric condition took place after a week under the use of sod. bicarb. with tinct. rhei, the pulse at the same time regaining its normal rate and regularity.

On the other hand I have frequently met with irregularity of the pulse without any great retardation, due to gastric or intestinal disturbance; for example, in a girl of 7 years who was feverish for only 24 hours (temp. 103·3° F.), had repeated vomiting and purging, and presented herpes labialis on the upper lip. The pulse in this case was 88—96, when the temperature fell, and was very irregular, intermitting after every third or fourth beat. This lasted for 9 days with diminishing distinctness and then suddenly disappeared. Occasionally even in meningitis the retardation of the pulse is absent, and we only notice its irregularity. Of this I have elsewhere published some

examples.[1] Such cases are, however, rare upon the whole; and where irregularity is combined with retardation you may always be prepared for the further development of tubercular meningitis. The hardness and vibrating character of the pulse (pulsus tardus) pointed out by Rilliet and Barthez, I regard as in no way characteristic, although I have frequently been able to observe it in the radial artery, and likewise just as distinctly over the great fontanelle when it was still open. The retardation and irregularity of the pulse usually last till about the middle of the second week, and then give place to a steadily increasing rapidity with regular rhythm. During this time the symptoms already described gradually increase in severity. The headache is rarely so violent as to make the children cry out and press their hands to their foreheads. Many scarcely complain at all of their head, but of pain in the ears, in the throat, the abdomen, the knee or other parts, although nothing abnormal can be found in them on examination. When the headache is present, it is generally aggravated by coughing. Occasionally also there seems to be a feeling of giddiness making the children think they are going to fall, even when they are sitting or lying down, and they beseech those standing by to keep hold of them. The apathy and drowsiness slowly increases, being sometimes interrupted by restlessness, loud screaming, also perhaps by slight delirium. If we wake the child when in this condition—which we can still easily do—we find the intellect clear so that it answers questions, and puts out the tongue when desired. The disappearance of childish obstinacy and the indifference towards the physician who used to be received with screaming, and towards his manipulations, is always a bad sign, and may, especially in doubtful cases, become important from a diagnostic point of view. The influence on certain secretory and trophic processes at this stage is also remarkable. Actively suppurating eczema on the head or other parts not uncommonly dries up, copious secretion from the nasal mucous membrane becomes arrested, previously existing diarrhœa ceases, and in two cases I have seen well-marked enlargement of the cervical glands, which had existed for a considerable time, disappear within a few days under the influence of meningitis.

In many of the patients (though by no means in all) we observe,

[1] *Beitr. zur Kinderheilk., N. F.*, S. 51.

about the middle of the second week, or perhaps even earlier, symptoms of irritation of certain of the cranial nerves which have become directly affected by the inflammatory irritation of the base, most frequently convergent strabismus and grinding of the teeth. Whether the chewing movements which begin about the same time and are somewhat characteristic of the disease, are also to be referred to irritation of the portio minor of the fifth nerve, seems to me to be doubtful, because in this case we would rather expect trismus (which as a matter of fact, does occasionally occur). Slight retraction of the head is sometimes noticed even at this stage. The colour of the face changes, sudden flushes passing over it from time to time. The drowsy condition very gradually passes into coma; it becomes more and more difficult to waken the child, until at last it lies in a state of complete unconsciousness, making no response when called to. The eyes are half closed, one leg generally stretched out while the other is flexed at the knee, the hands lying on the genital organs, which are occasionally in a state of erection. The child utters deep sighs from time to time, or even a piercing cry (the well-known but by no means constant "*cri hydrencéphalique*" of Coindet). About this time the pupils dilate, often one more markedly than the other, and they react to light either very sluggishly or not at all. On the conjunctiva bulbi we see leashes of enlarged blood vessels running towards the cornea, and fragments of mucus; gradually also cloudiness of the cornea appears, especially of its lower segment which is not covered by the half-closed eyelids, and is exposed continuously to the air, owing to the absence of motion in the lids. The reflex sensibility of the skin disappears like that of the conjunctiva, so that, *e.g.* a gentle stroking on the inner side of the thigh no longer occasions contraction of the cremaster. In addition we have automatic movements of the hands to the head, pendulum-movements of one upper or lower extremity, and rigid contracture of the muscles of the neck, and of those of mastication, so that it becomes difficult to give the child a drink. On more careful examination we also not uncommonly find some rigidity or paralysis of one or other side of the body. When there is paralysis the limb on being raised falls down without resistance, and lies motionless as if dead, while that on the other side is often jerked about in

all directions as in chorea. The constipation which has generally been present up to this time, and which yields only with difficulty to purgatives, is often replaced in this last stage of the disease by involuntary loose motions. The abdomen steadily sinks in in the region of the umbilicus, so that it comes to have a hollowed out appearance, with the costal margins and iliac crests projecting, and the vertebral column can be easily felt through it. Retention of urine sometimes occurs to such a degree that a catheter has to be used. The pulse-rate continues to increase from about the middle of the second week, and its rhythm again becomes regular. The rate gradually increases to 180—200 and more, and the pulse becomes smaller and more difficult to feel. The respiration, the implication of which has already been indicated by the above-mentioned deep sighing, almost always presents during the last 24—48 hours the Cheyne-Stokes character—either in its well-known classical form or else modified to some extent. Thus I have seen, after a pause in the breathing lasting for a quarter of a minute, first a deep sighing inspiration occur, followed by 2—3 superficial breaths and then another pause. The number of respirations in the minute may therefore be only 7—5, and this infrequency of the respiration, along with the extreme weakness of the heart (pulse 180—200 scarcely perceptible), explains the cyanotic discolouration of the face, of the visible mucous membranes, and of the points of the fingers and toes, which often comes on about this time. In many cases the face becomes dark red during the last few days, and profuse perspiration covers the forehead and cheeks in clear drops. On the other hand, I have had but few opportunities of observing the skin eruptions which other writers have mentioned (erythema and papules)—I saw one child of 2 years who in the last few days presented an erythema annulare extending over the whole body. To these symptoms, which indicate the fatal termination of the disease, there are very often added epileptiform convulsions in the last 24—48 hours. These either affect the whole muscular system of the body in violent paroxysms; or they occur only on one side, being sometimes confined to the facial muscles; or they consist merely in weak contractions of the limbs. In many cases rigid contractures of the muscles of the extremities and of the neck also occur, or a condition of tremor seen most

distinctly in the movements of the hands which continue after the onset of coma. It is always well to warn the parents of the possible occurrence of convulsions towards the end, even although no spastic phenomena have been observed during the previous course of the disease. I have but rarely found them entirely absent. The death-agony is always unusually long, whether convulsions occur or not. It frequently lasts for several days and—what is all the more painful for the parents—it occasionally happens that in the midst of this last hopeless stage there suddenly appear surprising and inexplicable signs of apparent improvement. The unconscious and comatose patient suddenly manifests a return of his mental activity. He turns his head to his mother when she calls to him, opens his eyes, takes his food once more, or may even begin to sit up again and catch at toys held in front of him. I have several times convinced myself of the correctness of this old observation. I therefore warn you not to over-estimate these favourable signs. After a few hours the child relapses into his former condition, and dies from progressing collapse (paralysis of the heart) with convulsions or deep coma—a fortnight or three weeks, as a rule, after the first occurrence of the vomiting.

We have yet to mention shortly the relations of tubercular meningitis with regard to temperature. Investigations which I have instituted during the last few years, and which I have already partly published,[1] go to establish the fact that this disease possesses no characteristic temperature-curve at all, but that very considerable variations occur throughout its whole course. The evening temperature nearly always exceeds that of the morning, more or less; it is rarely the same, and only exceptionally somewhat lower. At the same time the temperature is always about a medium height, rarely exceeding 102·2° F., and in many cases reaching this level only on a very few days. I have indeed observed cases in which during the whole course of the disease, or at least for several days, the temperature did not rise above the normal at all, or only did so very slightly. On the other hand, according to my observation the temperature rises rapidly—in the majority of cases, although not invariably—on the day before the last or else on

[1] *Charité-Annalen, Jahrg.* iv., S. 505.

the last day of the disease to a considerable height—to 104° F. or even to 107·6° F. It nearly always remains at this level till death, in rare cases falling just before the end to 100·4° F.—102·2° F. I have not yet investigated the condition of the temperature after death.[1] This sudden rise of temperature just before or during the death-agony, cannot possibly be regarded as an ordinary exacerbation of fever, for during the whole course of the disease the fever plays only a secondary part, and therefore we cannot suppose that it would suddenly rise to such a high degree just at the very last when the symptoms of collapse—heart failure (pulse 200, small)—were setting in. Nor yet can we regard the final convulsions or any chance inflammatory complication in the respiratory organs as answerable for it. I think I have proved this conclusively in my paper (l. c. page 510). Two or three times I have observed violent convulsions some days before death, with a temperature of 100·8° F., while during the last days there were no convulsions, although the temperature was 104° F. and over. In a few cases, also, where recent pneumonia was found at the post-mortem, I have noticed that this final elevation of temperature did not occur, while in all the other cases acute affections of the respiratory organs were not found, and yet this rise of temperature during the death-agony took place. This symptom—which occurs not only in tubercular meningitis, but also in adults who die with

[1] I give a few temperature charts as examples:—

Louise S., 1 year old, admitted on 29th Sept., 1878:—

	M.	E.
29th Sept.	100·4	101·3
30th ,,	99·7	101·3
1st Oct.	99·7	100·8
2nd ,,	100·4	100·4
3rd ,,	99·7	99·7
4th ,,	100·6	102·2
5th ,,	100·6	102·0
6th ,,	101·8	102·2
7th ,,	104·0	105·8
8th ,,	106·2	Death

H., 4 years old, admitted on 6th April, 1878:—

	M.		E.
6th April	—		101·3
7th ,,	99·5		100·4
8th ,,	99·0		98·2
9th ,,	101·1		101·3
10th ,,	98·2		99·5
11th ,,	100·4		100·6
12th ,,	100·8		101·5
13th ,,	101·3	11 o'clock	102·6
		4 ,,	103·6
		6 ,,	104·5
		9 ,,	107·2

In a child of 2 years, admitted on July 16th, 1881, the temperature was found to be 100·8° F. only on the evening of the 16th and 17th. From then to the 27th it was always normal or even subnormal. On the evening of the 27th the temperature suddenly rose to 104·4° F. (pulse 180), and on the 28th (the day of death) to 107·6° F.

These examples may suffice; very many of my cases presented similar conditions.

paralysis of the cerebral functions—I can only explain by the assumption that there is paralysis of the supposed heat-controlling centre, which is situated at the junction of the brain and spinal cord. If this is paralysed, the temperature of the body, which is now no longer kept in check, must reach an extraordinary height. You will find this subject further worked out in my paper already referred to, in which I have also gathered together the results of experiments which supported my view. Less commonly the temperature falls abnormally low before the end (from 96·8° F. to 82·4 F.[1]), which is to be referred to paralysis of the heat-producing centre.

In describing the course of the disease I have disregarded the customary division of the disease into regular stages; because I consider all attempts at such a division as useless, whether they rest on anatomical or clinical principles. We may perhaps distinguish a stage of irritation and one of paralysis; but even this division is by no means thoroughly justified. For, as we have seen, irritative symptoms—*e.g.*, convulsions—often enough appear for the first time during the last stage. If we take into consideration, moreover, the cases with an abnormal course and the numerous varieties, to which I shall presently recur, we see that the division into stages is misleading and had best be abandoned.

The variations from the typical normal course in this disease are, in fact, so numerous that we are much less likely to be correct in our diagnosis if we try to form our judgments according to one model case. Even physicians who think they know meningitis thoroughly are always coming upon new variations in its course which may cause confusion and cannot be explained anatomically. I have occasionally found a series of symptoms lasting for 10—12 days which resembled those of infantile typhoid very closely. Sometimes the children utter a piercing cry—day and night, almost without intermission—driving the parents to distraction, and then they suddenly fall into a state of coma. The initial vomiting, which is justly regarded with apprehension, may be entirely

[1] Gnändinger, *Jahrb. f. Kinderheilk.*, 1880, xv., S. 459.—Turin, *ibid.*, xv 1880, S. 24.—Loeb, *Deutsches Archiv f. klin. Med.*, 1883, S. 443.—Balaban, *Ueber den Gang des Temperatur bei Meningitis tub.*, &c.: Heidelberg, 1884.—Bokai, *Jahrb. f. Kinderheilk.*, Bd. xxi., S. 440.

absent, while in other cases it continues with the greatest violence for 9—10 days or longer, and there may be such slight symptoms of any other kind pointing to cerebral disease that the physician who sees the child once or at most twice a day may quite overlook them. One child of this kind I used always to find sitting up in bed when I visited him, apparently taking an interest in everything and eagerly looking at picture books. His eyes were clear and there was not the slightest drowsiness, nor anything but the obstinate vomiting, to cause anxiety to the parents or physician. The inequality and irregularity of the pulse, however, confirmed the diagnosis, which was soon established. When the vomiting is thus obstinate, the children often complain also of pain in the region of the stomach, and this may still further mislead the physician. Especially in little children in the first and second years of life, obstinate vomiting without any other threatening symptom seems to me to deserve the fullest attention. For in such children it is most likely to be regarded as due to dyspepsia, until after some time the sudden occurrence of drowsiness, squint, ptosis and convulsions, clear up the mistake in a very unpleasant manner. Even the obstinate constipation which we generally have to deal with is not a symptom always to be depended on. I have repeatedly met with cases which began with vomiting and diarrhœa and were therefore regarded as cholera infantum, until after 24—36 hours obstruction set in, while the vomiting either persisted or likewise disappeared. I have also occasionally seen diarrhœa due to follicular or tubercular ulceration of the intestine persist in spite of the development of meningitis. Instead of the usual hollowing out of the abdomen, I have sometimes observed a more or less extreme condition of flatulent distension, which is generally due to a concomitant chronic tubercular peritonitis. The rule which is applicable to the pulse (moderate acceleration during the first few days followed by retardation and irregularity, and finally increasing rate and regularity of the beats) only holds good in the majority of the cases. I have already previously (p. 320) pointed out to you the variations in the character of the pulse, and I would add that in several cases in the very last stage when epileptiform convulsions had already set in, I have found a pulse-rate of only 70, 76, 92, and 96. In one child of two years, a marked diminution in the secretion of urine took

place, and for two or three weeks formed the only premonitory symptom. This child only passed its water (which was normal) once in the 24 hours, and the bladder was not distended. It was only the increasing apathy and drowsiness that determined me to make the diagnosis of meningitis, which was confirmed by the further progress and by the post-mortem.

According to Legendre and Rilliet and Barthez the character of the symptoms suffers material modification according as the meningitis affects an apparently healthy child or one already affected with advanced tuberculosis or phthisis. Only in the former case does the above described "classic" course take place, while in the latter, the disease has a much more violent onset, with much quicker succession of the symptoms, resembling meningitis simplex. In my own practice I have frequently had the opportunity of confirming their statements.

> Anne H., 3 years old, brought to me on October 2nd, 1862. Since August, diarrhœa, weakness, and anæmia, steady wasting, cough, dulness, with sharp râles and bronchophony in the left supra-spinous fossa; fever, eczema on many parts of the body. On 24th November, sudden epileptiform convulsions; in the evening, vomiting, cessation of the diarrhœa, rapid irregular pulse. The eczema rapidly disappeared. Within the next few days drowsiness, coma, repeated convulsions. Death on the 28th—that is, on the 5th day after the first appearance of cerebral symptoms. *P.-M.*—Basilar tubercular meningitis, internal hydrocephalus, extreme amount of tubercle in both lungs. Cavities in both upper lobes; follicular enteritis, &c.

I have most frequently observed this very acute course ushered in by violent epileptiform convulsions in cases which were complicated with tuberculosis of the substance of the brain itself. I have indeed often been able to diagnose from such a course the presence of this complication before the post-mortem took place, even though I was unacquainted with the former condition of the child. You will find several cases of this kind brought together in my paper on cerebral tuberculosis.[1] Exceptions to this rule, however, are not uncommon. On the one hand the disease may take its usual course notwithstanding the presence of a considerable degree of tuberculosis of the brain or of advanced phthisis; while on the other hand it may have an unusually acute course where there is as yet no real phthisical

[1] *Charité-Annalen, Jahrg.* iv., S. 489.

disintegration.[1] This course, which very closely resembles that of purulent meningitis, is particularly apt to occur in little children in the first or second years of life; take for instance the following case, in which the whole process ran its course in 6 days.

> Karl M., 9 months old, admitted on March 18th, 1879. Healthy child. Took ill 2 days before, refusing the breast, vomiting, feverish. Drowsiness and extreme apathy. Temperature, 101·1°—101·8° F.; pulse, 132, regular. On the 19th and 20th increase of the drowsiness; pulse, 156; eyes often fixed, turned upwards; almost continuous twitchings of the upper limbs. In the lungs nothing to be made out but catarrh. On 21st, pulse 200; temp. 106·2° F.; rigid extension of the arms, with tremor; respiration frequent and noisy. Death on 22nd, with temperature of 106·2° F. and imperceptible pulse.
>
> *P.-M.*—Pia mater near the longitudinal fissure greyish-yellow, cloudy, very thickly studded with miliary nodules, still more marked at the base, especially in the Sylvian fissure. Ventricles distended by a large quantity of clear serum. Brain slightly œdematous. Miliary tuberculosis of both lungs and of the liver and spleen. Bronchial, tracheal, and mesenteric glands caseous.

We are not able sufficiently to explain the variations in the course of the disease from its pathological anatomy. The post-mortem conditions seem to be just the same whether the disease has a normal or an abnormal course; and the differences must therefore consist in finer modifications of structure which can scarcely be demonstrated. These affect sometimes one part of the brain, sometimes another, although their occurrence has not yet been proved beyond a doubt. In support of this idea I shall only refer to the observations of Rendu,[2] who in a series of cases found thrombosis of the Sylvian artery resulting from the surrounding tubercular inflammation, and little patches of softening in its area of distribution (corpus striatum &c.) with which he was able to connect the paralysis observed during life. In several cases characterised by an unusually acute course suggesting simple meningitis, I have myself found the inflammatory products deposited on the convexity of the hemispheres to a greater extent than on the base which is generally its favourite seat. In one of these children, indeed, this part was almost entirely unaffected. From this it follows that we must not regard the terms "meningitis

[1] *Vide* my "*Beiträge zur Kinderheilk., N.F.*," S. 44.

[2] *Recherches clin. et anat. sur les paralysies liées à la méningite tuberculeuse*: Paris, 1874.

tuberculosa" and "meningitis basilaris" as quite equivalent; but the variations in the course of the disease cannot depend on this alone, for I have also often enough found the convexity affected in the same way in cases with the ordinary prolonged course.

In the great majority of cases the affection of the basis cerebri is certainly the characteristic feature of the disease. In these cases we find a cloudy greenish-grey gelatinous infiltration of the pia mater, in the space between the optic chiasma and the medulla oblongata, which surrounds the cranial nerves as they pass out and may undoubtedly give rise directly to symptoms of irritation and paralysis in them. In this neighbourhood, especially inside the Sylvian fissure there is a cloudy, œdematous infiltration, and here also particularly we find more or less numerous grey or greyish-yellow miliary tubercles imbedded, about the size of a pin's head or less; and these are most clearly seen when we draw the pia mater carefully out of the fissures. According as these tubercular granulations are recent or old they are smooth and soft or somewhat hard and projecting. Similar miliary tubercles of the pia mater are also not uncommonly met with, often in very great numbers, in the choroid plexuses of the ventricles, on the convexity and inner surface of the hemisphere, —the pia mater at the same time often appearing extremely cloudy owing to serous infiltration, and streaks of exudation being deposited along the larger veins, either as greyish-yellow pus or in the form of caseous masses. I have but rarely met with small miliary nodules on the inner surface of the dura mater also. On microscopical examination of these nodules we find almost invariably the tubercular bacilli. The vessels of the pia mater are, as a rule, more or less congested, and when it is drawn out of the fissures little particles of softened cortical substance are apt to remain firmly adherent to it. We also find here and there, strips of adhesion between the arachnoid and dura mater or accumulation of serum between the two membranes, or blood-stained infiltration into the pia mater. The brain substance itself is generally anæmic, rarely hyperæmic; the ventricles are markedly distended by the accumulation of serous fluid and their walls as well as the central structures of the brain (corpus callosum, septum &c.) are often—but by no means always—very much softened or even broken down into a cream-like mass

floating in the cerebral fluid. In rare cases I have found little ecchymoses, especially in the neighbourhood of the third ventricle. These conditions are not, however, invariably found, as the accumulation of serum in the ventricles and their dilatation may also be absent; so that tubercular meningitis is not necessarily accompanied by "acute hydrocephalus." In this case the cream-like softening in the neighbourhood of the ventricles is also absent, and indeed it can only be regarded as a post-mortem appearance due to maceration by the accumulated serum.

In a small number of the cases, although we find indications of inflammation in the pia mater of the base and likewise of the convexity—diffuse cloudiness and thickening, œdema or gelatinous infiltration with or without hydrocephalus of the ventricle —yet in spite of the most careful investigation we nowhere discover miliary nodules in the pia mater, although they may be widely distributed in other organs (spleen, liver and lungs). I have myself met with such cases, and Rilliet and Barthez, who observed eleven of the same, put them down as tubercular meningitis—most properly, I think, because the presence of miliary tubercles in other organs, and the peculiar character of the inflammatory product marks them as such. It follows from this that these inflammatory products may also occur spontaneously, apart from the irritation of the miliary granulations; just as there is also, on the other hand, no lack of cases of acute tuberculosis in which, in spite of numerous miliary tubercles, there are no signs of inflammation at all to be made out in the pia mater. I shall return to these cases when discussing tuberculosis.

I have seen only a single case where the tubercles were limited to the pia mater to the exclusion of all other organs; and although similar observations have been published by other writers, *e.g.* by Bouchut, we cannot help suspecting that the post-mortems were not quite as exhaustive as they might have been. I will only recall the fact that we have repeatedly found tubercles in the marrow, which would assuredly have been overlooked by the older observers. I have also only in rare cases found the disease very limited in its extent; for example, in a child of $2\frac{1}{4}$ years with numerous tubercular masses in the brain and tubercular meningitis, there were only very few scattered miliary nodules in the right lung. Again, in a child of 2 with

tubercular meningitis of the base and convexity, I found only one single caseous deposit in the mesenteric glands; in a child of 9 months only one caseous mass the size of a hazel-nut in one of the bronchial glands; in a boy of 11 years, only one indurated bronchial gland the size of a hazel-nut containing small calcareous particles, all the other organs being perfectly normal. Far oftener I have found tubercular changes simultaneously in many other parts of the body, the most constant feature being a more or less extensive caseous degeneration of the bronchial glands. Tuberculosis and caseous processes are also found in the mesenteric and other lymphatic glands, in the brain, lungs, pleuræ, peritoneum, spleen, liver, and kidneys, and even in the epididymis and in the genital organs in little girls. In more recent times tuberculosis of the choroid has excited great interest, because at first when the fact of its occurrence was announced by Cohnheim and von Graefe it was thought that an absolutely certain criterion had been found for the diagnosis of tubercular meningitis and acute miliary tuberculosis. The ophthalmoscopic examination began therefore to be regarded as the most important diagnostic proceeding in this disease; and the discovery of one or more greyish-white granules or patches in the fundus was held to be decisive in all cases where the diagnosis was doubtful. The latter opinion is, indeed, quite justified, and I have frequently been able to convince myself of the importance of this examination. By it I have frequently found tubercles in the choroid a considerable time before the onset of the serious cerebral symptoms, and while the disease was still in the preliminary stage of vague indisposition; and I was thus enabled to realise the serious nature of the case. Unfortunately the choroid, as was afterwards found, is by no means constantly affected[1]; and of this I have been frequently convinced by post-mortems. We must, therefore, by no means regard a negative result of examination of the eyes as disproving the presence of meningitis; but, at the same time, a positive result may certainly be regarded as of the greatest diagnostic

[1] Heinzel (*Jahrb. f. Kinderheilk.*, Bd. viii., 1875, S, 355) in 31 cases of basilar tubercular meningitis did not once find choroid-tubercle either during life or after death, although in 15 cases there was neuro-retinitis and "choked disc"—the latter being probably caused by the pressure of the hydrocephalic ventricles.—Money (*Lancet*, xix., 1883, Vol. ii.), found tubercle of the choroid only 12 times at the post-mortem out of 42 cases of tubercular meningitis.

significance. The spinal cord also does not escape; for its pia mater often presents eruptions of tubercle and inflammatory products. In a boy of 8 we found the spinal arachnoid markedly thickened on the posterior aspect as low down as the lumbar enlargement and infiltrated with pus, but free from tubercle as far as could be seen on naked-eye examination. Probably this complication would be found more frequently if we would take the trouble to open the vertebral canal at every post-mortem.[1] The assumption that the onset of violent convulsions, contractures, and hyperæsthesia depends only on such an affection of the spinal membranes, is, however, unfounded; for in one case where the predominance of these convulsive symptoms was marked, the spinal cord was found to be perfectly normal at the post-mortem. We have often found considerable accumulations of fæces in the large intestine; in one boy of four the whole cœcum on both sides of the ileocœcal valve was distended by a fæcal mass an inch and a half in length.

As to the etiology of the disease I have only a few words to add. Although children with a hereditary predisposition to tuberculosis, or those who are suffering from scrofulous conditions, phthisis, or chronic suppurations connected with bone, are most liable to the disease, you will nevertheless very often see well-nourished and apparently healthy children fall victims to it. It is only since the discovery of the tubercle-bacillus that we have recognised the possibility of these cases arising from direct infection; positive proof of this will very seldom be found possible. In general, all the ways in which the bacilli may enter the body and set up tuberculosis are also of significance as regards the origin of tubercular meningitis (the mucous membrane of the digestive and respiratory tract, and the skin). Of especial importance is the fact, which innumerable observations have confirmed, that the bacillary infection of the pia mater may start in apparently quite healthy children from very limited caseous, tubercular deposits in the lymphatic, mesenteric, or bronchial glands, which have existed for many months or even years without giving rise to any symptom whatever.

The assumption of a traumatic cause, especially of a fall

[1] F. Schultze has carefully examined these spinal changes microscopically in 3 cases of basilar tubercular meningitis—which, however, occurred in adults (*Berl. klin. Wochenschr.*, 1876, Nos. 1 and 2).

on the head (to which the parents always incline), is usually quite mistaken under these circumstances, and is generally based on a mere chance coincidence. At the same time it cannot be denied that a concussion of the brain is more likely to be followed by other hyperæmic conditions and their results in children with a tubercular tendency than in others (p. 312).

I have, unfortunately, nothing favourable to tell you as to the results of treatment. All physicians who go thoroughly into the diagnosis will agree with me in this, that they regard every case of tubercular meningitis as lost from the beginning; and they are not mistaken in this prognosis. The few cases of recovery which have been published are therefore to be received with the greatest reserve. The possibility of recovery certainly cannot be denied. When we remember that in tubercular subjects every pleurisy or peritonitis does not prove fatal, and, further, that the danger of the disease does not arise from miliary nodules in the pia mater, which are not uncommonly quite latent, we can only refer the enormous mortality of meningitis to two causes. The first of these is the concomitant, acute tuberculosis of many other organs; the second is the local changes which the brain suffers, both from softening of the grey substance immediately under the pia mater, and from the increasing pressure of the dilated ventricles. When it has once reached this stage any idea of recovery is of course out of the question. On the other hand I do not regard it as impossible to bring about recovery by opportune treatment at the beginning of the case when the miliary tuberculosis is not general but localised, as our main object at this stage is to arrest the commencing inflammation of the pia mater, and to prevent a more extensive exudation, which might affect the cortical substance of the brain. It is true that this attempt only succeeds in extremely few cases; but I believe, nevertheless, that it is always worth while to make it, except in cases where, owing to the presence of advanced phthisis or of the signs of tuberculosis of the brain itself, it is evidently useless from the very first.

I have elsewhere[1] published some cases which presented all the symptoms of the first stage of tubercular meningitis, and were cured by energetic antiphlogistic treatment. One of these cases—that of a child of $1\frac{3}{4}$ years—ended fatally from an attack

[1] *Beiträge zur Kinderheilk.*: Berlin, 1861, S. 13, and *Neue Folge*, 1868, S. 55.

of meningitis three years after the first illness; a brother of his having in the meantime died of this disease, this fact seemed to me to be in favour of the correctness of the diagnosis. Rilliet and Barthez record two cases in which death took place from a second attack occurring two or three years after recovery from the first one; and at the post-mortem the old and the recent eruptions of tubercle in the pia mater could be clearly distinguished. Politzer[1] also describes the case of a child who had suffered three years previously from an attack of basilar meningitis, and who—except for persistent emaciation—completely recovered. At the post-mortem, besides the recent basilar meningitis, an obsolete indurated patch was found on the pons. Although, therefore, these exceptional cases seem to show that even after recovery has taken place a fatal return of the disease is always to be feared sooner or later, this apprehension must not cause the physician to take up a passive attitude. I therefore order, to begin with, the application of 3—6 leeches behind the ears (according to the patient's age), and an ice-cap to the head; I also give calomel, gr. $\frac{3}{4}$ every 2 hours, and —if the bowels are not freely opened—follow it by mist. sennæ co., or syrupus rhamni, and have blue ointment (grs. v.—x.) rubbed into the neck and throat several times daily. Although in about fifteen years I have seen no result from this mode of treatment, I still consider it my duty to carry it out, and it will certainly do no harm in a disease which, if left to itself, is inevitably fatal. It is of course only to be tried during the first few days of the disease; at the later stage neither this nor any other kind of treatment can be of any avail. I have also abandoned the extremely painful inunction of tartar emetic ointment into the head, which used to be so strongly recommended; and the application of fly blisters to the neck. Further, the continuous use of iodide of potash which I have tried in innumerable cases, and the repeated and long-continued painting of the head and neck with iodoform-collodion, have been equally far from yielding successful results.

[1] *Jahrb. f. Kinderheilk.*, 1863, vi., S. 40.

XVIII. *Purulent Meningitis.*

The frequency of purulent meningitis, whether affecting the membranes of the brain alone or those of the spine also at the same time, is not great compared with that of the tubercular form. Only those physicians who have had the opportunity of observing epidemic cerebro-spinal meningitis have any considerable material at their command; for under ordinary circumstances the number of cases to be observed is always very small.

Anatomically the disease is generally characterised by the absence of all tubercular formations in the brain and its membranes, as well as in any other organs. This does not, of course, exclude the possibility of a tubercular subject being affected accidentally by simple meningitis—*e.g.* as the result of a fracture of the skull. Apart from these cases and a few others —*e.g.* those due to pyæmia—almost every case of meningitis in tubercular subjects assumes the anatomical and clinical characters described in the last chapter; and even the absence of miliary tubercle in the pia mater does not violate this rule (p. 331). Simple meningitis affects the convexity of the hemispheres far more frequently and more severely than does the tubercular form; but the inflammation often spreads to the base also, and extends from this over the medulla oblongata more or less deeply into the vertebral canal (cerebro-spinal meningitis). From the base the sero-purulent infiltration may spread even as far as the tissue behind the eyeballs, thus occasioning exophthalmos. In addition to marked hyperæmia of the pia mater, ecchymoses of various sizes, and localised adhesions of the dura with the pia mater, you find the tissue of the latter infiltrated with yellow or yellowish-grey pus. This partly follows the course of the larger blood-vessels, partly spreads out so as to form a layer, and also a varying amount of it may occur free between the pia and dura mater. The grey cortical layer of the brain is frequently adherent at many points to the pia mater, softened at its periphery by imbibition of serum, also hyperæmic in places and with capillary hæmorrhages scattered through it. Although the ventricles are empty as a rule, this is by no means invariably the case. I have occasionally found them

distended by turbid serum containing streaks of purulent matter, while the ependyma was at the same time loosened, but showed no important change. In a child of 2 months both the lateral and the fourth ventricles were filled with thin yellow pus, and much dilated. When the spinal cord is implicated we find a quite similar purulent infiltration of its pia mater and of the loose meshes of the arachnoid, the posterior surface of the spinal cord being most severely and extensively affected. Also the inner surface of the dura mater both in the cranium and in the spinal canal is in many cases congested and covered with pus and blood (pachymeningitis). All the symptoms occur equally in the epidemic and sporadic cases of the disease.[1]

I have as yet had no opportunity myself of observing the epidemic infectious form on any considerable scale, although many times cases of this disease have followed each other so rapidly here in Berlin that, taking them along with cases simultaneously observed by other medical men, I have been obliged to regard them as examples of a miniature epidemic. Two cases which came into my ward immediately after one another in the summer of 1885—one of which ended fatally—occurred even in one family. At any rate the so-called sporadic cases were at least as common. As far as my experience goes, a very acute course—which was ormerly held to be in favour of this meningitis in contradistinction to the tubercular form—is by no means a sure criterion; since, as we have seen, there are cases which last as long, in fact much longer, than those of the tubercular form. The clinical symptoms also may vary so much in their severity and combinations that it is impossible to sketch a clinical picture which will apply to every case.

The following may be mentioned as being the main symptoms which can in general be traced like a red thread running through all its varying manifestations: headache in children who are old enough to complain of it, vomiting, stiffness of the muscles at the back or sides of the neck, contractures of the extremities, convulsions, delirium, coma, and more or less high

[1] With regard to the occurrence of specific bacteria in the pus of this form of meningitis, the statements of writers vary. Many speak of micrococci, others (A. Fränkel) of a form identical with the pneumo-coccus which he has described. Attempts at cultivation which were made in the Pathological Institute with the pus from one of my cases gave an entirely negative result.

fever. Of these symptoms, however, either one or more may be absent, or else their presence be so slightly marked as to be readily overlooked. The order in which they occur also varies. In a series of cases, well-marked brain symptoms set in at the very beginning—delirium, coma, vomiting, convulsions and cervical rigidity which at once put the diagnosis beyond a doubt. Such cases occasionally have an extremely violent and acute course.

A girl of 5 years suddenly, in the midst of perfect health and without discoverable cause, became affected by violent headache and vomiting. After three hours, general epileptic convulsions and deep coma. The convulsions ceased after about 12 hours, while the coma persisted; there was high fever. The convulsions then recommenced and lasted till death, which took place 48 hours after the commencement of the illness. *P.-M.*— The whole convex surface of the brain covered with a yellow purulent exudation, infiltrating the pia mater, which formed a coherent layer over the frontal lobes, further back followed the course of the vessels and penetrated deeply into all the fissures. Also at the base purulent infiltration in the neighbourhood of the optic and oculo-motor nerves. Ventricles empty. The remaining organs healthy.

In a boy of 1½ years vomiting and general convulsions suddenly began in the early morning, lasting till 5 P.M. They then ceased for 5 full days, during which there was fever and coma, and then recommenced on the day before death (the 6th day of the disease).

The younger the children are, the oftener does the disease begin with convulsions, which occur one after the other, and are rapidly followed by coma. In many cases, however, even at this early age an extremely high temperature forms the chief symptom, and for a considerable time supports the diagnosis of typhus until at last unmistakable cerebral symptoms set in.

Agnes W., aged 8 months, healthy, child of a medical man, took ill on March 8th, 1877, with a single violent fit of vomiting. The child was pale, unwilling to take the breast, and, contrary to its usual custom, very quiet. On the following day, however, there was still nothing really morbid to be found. She laughed and jumped in her father's arms almost as happily as ever. On the 10th and 11th the child again became apathetic and very feverish, and in the evening the temperature was 105·4° F., so that we looked for a scarlet fever-rash. On the 4 following

days, up to the 15th, the high fever formed the only important symptom. The temperature was as follows :—

	M.	E.
On 12th March	104·0	105·8
„ 13th „	104·7	107·2
„ 14th „	105·1	104·4
„ 15th „	104·2	101·8

The fall of the temperature during the last 2 days was effected by two cold packs, two doses of quinine (grs. iii. and grs. vi.), and finally by a bath of 86° F. The diagnosis wavered between typhus and meningitis; and on the occasion of my first visit (on the 15th) I did not venture to make up my mind; but on the 16th—that is, 8 days after the commencement of the vomiting—a moderate amount of rigidity appeared in the muscles of the neck, along with turning of the head towards the left and a slight contraction of the right arm at the elbow-joint. Neither by continued ice-compresses to the head nor by cold baths given twice daily, and enemata, containing quinine (grs. viiss), were we now able to bring down the temperature. This kept between 104° F. and 106·5° F., and only fell temporarily during the 2 last days to 101·3° F. Pulse between 130 and 160, always regular. As now (on the 18th) the neck seemed to be again more easily moved, and the spleen was found to be much enlarged on palpation; as, further, the child—in spite of the continued high temperature—responded readily when called to, and grasped at a watch held in front of it, we again hesitated in our diagnosis of meningitis. But on the 19th the vomiting returned, and the cervical rigidity and contracture of the right arm again set in, making the diagnosis certain. Convulsions of the whole body, with dark redness of the face and profuse perspiration occurred for the first time on the evening of the 21st. During the night frequent screaming and repeated vomiting. On the following day, at 3 P.M., an epileptiform attack, lasting for half an hour; later, energetic chewing and sucking movements; convergent strabismus, congestion of the conjunctivæ. The convulsions recurred on the 23rd, from 3—6 P.M., and again at 10 P.M., after which they continued till death ensued, at 3 P.M., on the 24th. Pulse at the last, 200, thready.

P.-M.—Very severe cerebro-spinal meningitis. About a tablespoonful of free pus on the surface of the brain; purulent exudation $\frac{2}{5}$ inches thick between the meshes of the pia mater; softening of the brain substance, extending about $\frac{2}{5}$ inches into the grey substance of the brain. Ventricles empty. Spleen enlarged to about thrice its normal size. All other organs normal.

In this case we find the convulsions beginning on the 13th day of the disease when there had previously existed nothing but a very

high temperature, a certain amount of cervical rigidity, contracture of the right upper arm, and palpable enlargement of the spleen. For these symptoms, which are sufficient for a diagnosis, we are in some cases kept so long waiting that we think sooner of the development of tubercular, than of purulent meningitis. This mistake is especially liable to be caused by a persistent low temperature (about 101·3° F.) and a not very rapid pulse-rate (64—90) and the pulse may also be irregular.

Thus in a child of 9 months,[1] rickety but quite free from tubercle, vomiting occurred after every meal for a fortnight before cervical rigidity made its appearance. At the same time there was high fever (pulse 152 regular) almost continuous screaming and contractures of the fingers. During the 5 last days, continuous coma and almost uninterrupted epileptiform convulsions. Accompanying these, there was a return of the vomiting, sinking-in of the fontanelle, dilatation and immobility of the pupils; pulse small and too rapid to be counted, breathing irregular. Death after 3 weeks. At the *P.-M.* we found purulent meningitis of the convexity and base, which had extended to the pia mater of the cervical cord. Ventricles dilated, filled with turbid serum and pus. Otherwise all organs normal. No tubercle anywhere.

The following case also looked like one of tubercular meningitis, although it began with an attack of convulsions, which was referred to a complication with tubercle of the brain.

Max Th., 7 months old, rickety; admitted June 11th, 1884. After a cough which had lasted for some time, suddenly, 2 weeks ago, an epileptiform attack occurred. Since then, retraction of the head, sometimes more marked than at others. The head and spinal column formed an acute angle; the former could not be bent forward. At the same time great apathy, left convergent strabismus, right pupil somewhat dilated but reacted well. Bilateral otorrhœa especially on the right side. Catarrh of the large bronchi. These symptoms had persisted for nearly three weeks unchanged. Apathy and drowsiness daily increasing; extreme emaciation. During the last days, coma, pericorneal injection, fragments of mucus on the conjunctiva; temperature never above 100·4°—101·8° F., in the last days almost normal. Ophthalmoscopic examination negative. Death on 29th in coma without convulsions.

P.-M.—No tubercle in any organ. Moderate basilar purulent meningitis, extreme dilatation of the lateral and of the fourth

[1] *Beitr. zur Kinderheilk., N.F.*, S. 13.

ventricles, which were filled with thin yellowish pus. Ependyma swollen. Brain anæmic, a hyperæmic zone round the ventricles. Purulent otitis media in both ears with purulent infiltration of the surrounding bone.

In this case the basilar meningitis may have arisen from otitis media and then spread along the choroid plexus into the ventricle. The disease lasted for five weeks altogether, convulsions occurred only once, at the beginning of the meningitis. Convulsions may, however, be entirely absent during the whole course of the disease, and in that case there occur in their stead contractures either of the muscles of the neck and back only, or also of the limbs (especially the lower) presenting more or less rigid resistance to extension, and when they are extended the child screams loudly. In one case (a boy of 10 years) there was also an extremely tender diffuse swelling of the left hand and right knee-joint, which slowly disappeared under the use of mercurial inunctions.

Ernst P., 7 years old, admitted in November, 1872, with catarrh of the larger bronchi and typhoid symptoms. Coma, tongue dry and red, soon becoming brown, lips blackish: spleen and liver normal in size. Temp. 102·2°—103·1° F., later 101·8° F. From the 6th day after admission cervical rigidity and stiff flexion of the lower limbs, dilatation of the left pupil, frequent loud screaming; later, flexion of all the fingers and supination of the hands. Temp. varying from 97·9°—100·8° F. On the 12th day, improvement, tongue moister, tremor of the legs, intelligence returning, appetite better. During the next 2 days, symptoms worse again. Temp. normal. After the 16th day intelligence quite clear, temp. 101·3°—102·2° F. After the 22nd day all spastic symptoms disappeared, and the child seemed well. Free from fever. Pulse during the whole illness varying between 104 and 132. Only once (on the 28th day) was the pulse 46 and temperature 98·2° F.

Otto K., 7 years old, admitted in December, 1872, with gastric symptoms, pains in the head and body and the abdominal wall extremely tense. From the 3rd to the 7th day violent delirium, drowsiness, complete apathy, temperature normal. After the 7th day, marked improvement, intelligence brighter till the 11th, when the child got worse again and complained of violent pain in the neck. Moderate cervical rigidity and contracture of the adductors of the thighs. Temp. 97·7° F. and pulse 60—64 till the evening of the 12th day. The symptoms continued to get worse and considerable hyperæsthesia of the lower extremities came on, with repeated vomiting and severe pain in the

back and loins, the temp. rising at the same time to 103·5°—104·7° F., the pulse 110—142. On the 14th day all the symptoms subsided and the temp. and pulse gradually returned to their normal condition.

The treatment in both cases consisted in the repeated application of leeches to the head and wet-cupping along the spinal column, warm baths (in the first case with cold douche to the head and back), inunction of mercurial ointment; internally, calomel and other purgatives.

Gottfried Sp., 7 years, ill for 3 days. Admitted on May 23rd, 1885, with violent headache, followed by pains in the neck and left knee; drowsiness, slight delirium, extreme rigidity of the neck and spinal column, which increased when he was set up. Pupils normal. Temp. 100·8° F., pulse 100, but soon fell to 84 and became irregular. Lower limbs slightly contracted in a position of flexion; no hyperæsthesia.

Treatment—12 wet cups, inunction of ung. hydrarg. grs. x.; calomel, gr. ½ every 3 hours. On 24th, 8 more wet-cups. On 25th, herpes labialis. Temp. 101·1°—103·1° F. Drowsiness alternating with free intervals. On 26th, disappearance of the contractures, pulse 120 regular. Temp. 101·3° F. The symptom described by Kernig[1] could be distinctly observed, and continued with diminishing distinctness into the period of convalescence. It only disappeared entirely on June 6th. After 3rd June, patient free from fever. The cervical rigidity, which was then moderate, did not disappear till the 9th. Latterly pot. iod. was given. Discharged cured.

In these and several other cases which I have lately met with, I observed the symptom described by Kernig, *i.e.* rigidity of the lower extremities at once came on when we set the patient up in bed, even when there was no rigidity when lying quietly on the back. Further, when the patient was lying on the side, this rigidity was generally set up whenever the thigh was placed at anything like a right angle to the body; there thus occurred at once a condition of contracture at the knee-joints in an attitude of flexion, which firmly resisted extension but disappeared at once when the patient was put back into the horizontal position. I cannot say, however, that this symptom is constant; for it was absent in one severe case in which the diagnosis was confirmed by a post-mortem—at least so long as the case was under clinical observation. Even although the symptom is not pathognomonic, as it occurs in other cerebral affections also, still it cannot be denied that it has some diagnostic value. It was also

[1] *Berl. klin. Wochenschr.*, 1884, No. 52.—Bull, *ibid.*, 1885, No. 47.

very well-marked in a case of tubercular meningitis which was complicated by a considerable purulent spinal arachnitis. I must agree with Bull that we may often discover a slight degree of this phenomenon even in healthy people, especially if we place the thigh at an acute angle.[1]

If all cases presented the violent symptoms which I have described, it would be possible, generally, to give a pretty certain diagnosis. This, however, is not always the case; for there is a moderately severe chronic form of meningitis, especially of the infectious variety, which by its long duration and the varying severity of the symptoms, may mislead the physician, especially if he is inexperienced. Usually, the course is as follows. The children who have hitherto been healthy, take suddenly ill with more or less high temperature, which in the afternoon or evening hours may reach from 103·1° to 104·4° F. There is violent headache from the beginning, generally frontal; and this even in young children is often indicated by their catching at the head, moaning and whimpering. Vomiting occurs often, but not always. Cervical rigidity with retraction or lateral obliquity of the head (caput obstipum spasticum) is constant, and the latter in one boy was so severe and persistent that a bedsore formed on the right ear, on which he always lay. Every passive movement of the head gives rise to expressions of pain. Less frequently, rigidity of the muscles appears in the extremities also, especially the lower ones, and both active and passive movements are interfered with. However, I have very seldom been able to make out distinct hyperæsthesia in these parts, and it was absent even in some of the very acute cases. The patellar reflex in several cases which were carefully examined in this particular, was well-marked. After about 1½—2 weeks the fever diminishes considerably, may even temporally dis-

[1] Dr. Sachs of Brieg has, in a letter, drawn my attention to the following remark of Landois: "The long extensors of the leg arising from the tuber ischii are too short to allow of complete extension at the knee joint when the hip is bent at an acute angle." Henke discusses this circumstance more fully (*Handatlas u. s. w.:* Berlin, 1888, i., S. 175). The three muscles here concerned, semitendinosus, semimembranosus and biceps, are distinctly pennate, and contain very many short fibres. They are therefore quite incapable of such a stretching as they would require to undergo if the hip joint were actively flexed and the knee extended at the same time. Even in the dead body this is not possible. At any rate however the resistence of the muscles in meningitis is even greater than in the normal condition, perhaps because the muscular tone is here morbidly exaggerated.

appear, and the appearance of health which now sets in seems to justify the relatives in entertaining the highest hopes. But the cervical rigidity which persists although in a diminished degree, shows that recovery is not yet complete. The fever in fact recommences after an interval of one or more days, the general condition again becomes worse, the headache and cervical contracture become more prominent without any cause for this aggravation of the condition being discoverable. Such remissions and exacerbations may be frequently repeated. The children become steadily more emaciated and weaker, and the physician already suspects tuberculosis of the brain or cervical vertebræ, till after a course of seven, ten, or more weeks, recovery at last takes place. I, at least, have never observed a fatal termination of such cases, except in some in which the disease had run its course, without the characteristic remissions, with almost equal severity of symptoms for a number of weeks.[1]

Unfortunately, however, recovery from meningitis is not always complete. Like many other writers I have repeatedly seen deafness or amaurosis in young children, also deaf-mutism, remain permanently. We refer these derangements of the organs of sense to neuritic changes, which are supposed to depend on the inflammation spreading from the meninges to the optic and auditory nerves. More recent observations make it probable that this extension may also take place through the bands of the dura mater which pass into the petrous bone to its spongy tissue and from thence to the semicircular canals, setting up hæmorrhagic inflammation there.[2] Children who become deaf at a very early age, before they have begun to speak, as the result of meningitis, naturally remain dumb also, because hearing is absolutely necessary if the child is to learn to speak. In very rare cases amaurosis or deafness may also set in during the disease as a symptom which passes off after a few days. In a girl of eight years contracture of the muscles on the right side of the neck (torticollis) persisted unchanged two months after recovery from the meningitis.

Among the causes of meningitis—next to epidemic influences, to which I shall shortly return—the most important are injuries and diseases of the cranial bones. Even

[1] *Vide* my paper on this form in the *Charité-Annalen*, Bd. xi.: Berlin, 1886.
[2] Lucae, *Virchow's Archiv*, Bd. 88, 1882, S. 556.

after severe concussion of the brain from a blow or a fall symptoms of cerebral hyperæmia may set in, as already mentioned (p. 311) and may end in meningitis. Far more dangerous are fissures and fractures of the cranial bones, which, besides the meningitis, may give rise to more or less severe hæmorrhage into the cranial cavity.

Max E., 5 years old, admitted on 1st July, 1875. Had tumbled from a ground-floor window on to the street 3 days previously, falling on his head. Stupor, right pupil smaller than the left, urinary bladder distended, reaching to the level of the umbilicus. The head was turned to the right and rotation to the left was anxiously avoided and guarded against. T. 103·6° F., P. 120 regular, R. 30. Bladder emptied by catheter; leeches and ice-bag to the head; purgatives. On the following days active delirium, violent pain on swallowing in spite of the stupor and the normal condition of the pharynx. After 3rd July, profound drowsiness, but screaming when raised up. Moderate cervical rigidity, slight spasmodic contraction of the arms, increasing pulse-rate, finally uncountable. Death, on evening of 4th, in a state of coma.

Temp. on 2nd July	103·3	103·6
„ „ 3rd „	104·2	104·9
„ „ 4th „	106·7	104·5

P.-M.—Marked hyperæmia of the pia mater and on the convexity; extensive purulent infiltration of it, especially on the left side. Sides of the Sylvian fissure adherent to one another; in the pia mater, especially on the left side, large purulent patches in this situation. In the bones of the base of the cranium on the left side three fissures traversing the frontal bone, the greater and lesser wings of the sphenoid and the temporal bone. Blood extravasion between the dura mater and the bone corresponding to these fractures.

In this case the absence of all motor derangements—with the exception of slight twitchings of the arms, and some contracture of the muscles of the neck—is worthy of notice. The pain on every movement of the head and on swallowing, may indeed be explained merely by the inevitable movement of the fractured fragments of bone; especially the pains on swallowing, by the action of the pterygo-pharyngeus and stylo-pharyngeus which must have exerted some traction on the fractured base of the cranium. In this case, also, we found a very high temperature persisting (to 106·7° F.).

We may also have meningitis due to chronic diseases of

the cranial bones. Still, I have myself but seldom met with purulent meningitis post-mortem, in spite of the fact that I have observed numerous cases of caries of the petrous bone. I have more frequently found the above-mentioned (p. 315) thrombosis of the neighbouring sinuses with purulent disintegration and pyæmic symptoms, or the hæmorrhagic form of inflammation of the inner surface of the dura mater which is known by the name of pachymeningitis, and on which I have already touched (pp. 272, 304). To this class also belong those cases of meningitis which arise as the result of a direct injury to the membranes of the brain or spinal cord, *e.g.* from an operation. I have met with this after puncture of a large hydro-meningocele at the occiput and of a lumbar spina bifida.

> The latter case was that of a child of 2 months with a defect of the sacrum and of the 3 lower lumbar vertebræ. There was already gangrene of the skin covering the tumour, and when this was excised the sac was opened and 2 tablespoonfuls of serum evacuated. A suture was inserted and an iodoform dressing applied. After 2 days, death ensued with spasmodic contractions of the lower limbs and a few general convulsions, and at the post-mortem we found a fibrino-purulent infiltration of the whole spinal pia mater up to the base of the brain. The temperature in this case had sunk to 93·7° F., during the disease; which is a further proof of the fact that during the first period of life even acute inflammations may run their course with subnormal temperature (p. 17).

Meningitis sometimes developes secondarily in the course of various acute diseases; *e.g.* pneumonia, nephritis, pyæmia, and septicæmia. As a rule, however, the symptoms are under these circumstances so complicated by those of the original disease that a definite diagnosis is very difficult or even impossible. At any rate, the complication of scarlet fever or pneumonia with actual meningitis is rare, and the cerebral symptoms which come on in these and other infectious diseases are—as we shall see later on—to be regarded either simply as the result of the considerable elevation of temperature or of the virulence of the disease. In these cases generally the presence of meningitis is out of the question, although formerly this designation was very freely bestowed. I would especially point out to you that violent cerebral symptoms with vomiting and even convulsions may arise in children from otitis media or even otitis externa, and may occasion a false diagnosis of

meningitis until a large quantity of pus suddenly escapes from the ear, and the dangerous symptoms rapidly pass off. In all cases, therefore, where head-symptoms are present we must bear in mind this possibility and carefully examine the external auditory meatus at least. Firm pressure on the tragus is often sufficient in such a case to set the child a-crying. I must, however, according to my experience hitherto, regard as rare the cases in which the symptoms of otitis are really such as to be mistaken for those of meningitis, and as even rarer those in which meningitic symptoms are caused by rhinitis. Twice—in a boy of three and a girl of four years—I have observed after a fall on the nose, in addition to the local symptoms (swelling, tenderness of the nose and interference with breathing), violent frontal headache, high fever, and restlessness, nocturnal delirium, which came to an end with the rupture of the abscess, and the discharge of blood and pus from the nose. In a number of the cases we are unable to make out any of the causes named, but the disease arises, as the saying is, "from a whole skin," in the midst of perfect health, and it is in those cases that the suspicion of an infectious origin at once occurs to one. The proof of such an assumption is only possible, however, when at the same time and in the same family, or at least neighbourhood, one or more analogous affections have recently occurred or actually prevail. Such cases I have met with in no small number, particularly in the summers of 1879 and 1885, and, especially during the latter season, almost all the cases came to the hospital from one quarter of the town. It has been already mentioned (p. 337 *note*) that in spite of the most recent bacteriological investigations, we are still very uncertain as to the nature of the infecting material. From a clinical point of view I should remark that this form frequently has a protracted course interrupted by great remissions, and that after the disappearance of the regular cerebral symptoms a temperature rising to 103·1° F. may persist for days and weeks with marked morning remissions, or complete intermissions, quite similar to what occurs in typhoid fever. In one case, after apparently complete recovery, death ensued from inanition and increasing collapse, against which all stimulants and tonics proved ineffectual.

In the treatment we must seemingly be guided by the stage of the disease and by the state of the patient's strength. At the

beginning, a thoroughly antiphlogistic line of treatment is indicated, while at a later stage this is to be avoided and stimulants are rather to be preferred. The exact period of this change in the character of the case, which seems to depend on the onset of suppuration, is certainly hard to determine, and it is more a matter to be settled by the practical skill of the physician than by theoretical rules.

For very young, badly-nourished, anæmic children, or those reduced by illness, we generally use dry cupping, at most 2—3 leeches, according to the age; but the bites must never be allowed to go on bleeding afterwards. For older children, on the other hand, especially such as are robust, 6—10 leeches are required, or a similar number of wet-cups on the neck, and perhaps also on the back. Under these circumstances I have even repeated the blood-letting when exacerbations set in, and the state of the strength permitted it. I warn you urgently once more against the lack of energy which is now so prevalent in the profession, and which induces the practitioner rather to stand with his hands in his pockets than to apply leeches. At the same time, as long as there are no symptoms of collapse, we should keep an ice-bag applied to the head, have mercurial ointment rubbed into the neck, back, arms, and thighs (grs. v.—x. every 3 hours), and give calomel internally, gr. $\frac{1}{4}$ to $\frac{1}{2}$ every 2 hours. The favourite antipyretics—quinine, salicylate of soda, antipyrin, cold baths and cold compresses—have in these cases no effect, and scarcely lower the temperature. When there is very great restlessness or violent convulsions, we may try injections of morphia (gr. $\frac{1}{32}$—$\frac{1}{13}$), or chloral (Form. 9), also luke-warm baths (88°—90·5° F.), with cold douche to the head. When the acute stage is over, I should recommend iodide of potash (Form. 13). During the continued use of this medicine I have frequently seen the children awake out of their comatose condition, the contractures disappear, and complete recovery take place at last. On the other hand, the derangement of the faculties which remains behind (deafness, loss of speech, amaurosis) almost always resists all treatment.

I have made use of this method of treatment in all forms of meningitis, including the infectious variety, and on the whole I am satisfied with it. We possess no specific remedy for this

any more than for many other infectious diseases, the treatment must therefore be symptomatic. But we must, in regard to this form, remember that it is an infectious disease, and carry out the anti-phlogistic measures with more caution than in those cases where an injury or other causes have given rise to it.

XIX. *Neuralgic Conditions.*

In childhood you will meet with striking sensory disturbances much less frequently than in adults. Anæsthesia, hyperæsthesia, and neuralgia are exceptional in children, and so greatly resemble in all particulars similar conditions met with in later life that it is unnecessary to discuss them at length here. Anæsthesia especially is extremely difficult to estimate, even in older children, because the result of the examination is rendered dubious by their terror when the needle is used—and this is the case even when the patient is blindfolded. Even in serious chronic diseases of the central organs (tumours, tubercle, sclerosis) I have never been able to ascertain the limits of the anæsthetic areas so exactly as in adults, and we must be content to base our diagnosis on observed intellectual and motor derangements. Among the forms of neuralgia in childhood, the only ones which call for special mention are colic—which either comes on with flatulence or along with diarrhœa (p. 126)—and hemicrania (migraine).

Migraine occurs in children—as only the inexperienced will deny—very nearly as often as in adults, and with pretty much the same symptoms. As the result of many years' experience, however, I am inclined to maintain that—especially during the last 20 years—the frequency of the disease has considerably increased. And the cause of this increase is to be found in the excessive demands which the education of the present day makes on the child's brain. The unceasing growth of our city which is always making the enjoyment of country air more difficult of attainment, the mental exertion in schoolrooms which are often overcrowded, and the very few hours left for recreation which are further encroached upon by home tasks and music lessons—all this, combined with nervousness which is

often inherited, and sometimes acquired through injudicious upbringing, appears to me the cause of those headaches which we so often meet with in boys and girls of about seven and upwards.

Besides this cause, there is also a hereditary tendency to be considered. I have not uncommonly had children under treatment for migraine in whom heredity, either from the father's or mother's side, was ascertained beyond doubt. The youngest of these children was 2½ years of age, and suffered about every 5 or 6 weeks from attacks of pain over the left eye, which lasted about half an hour, and ceased after vomiting and, less frequently, movement of the bowels had taken place. When such a tendency is present several children of the same family may suffer from this affection.

> Two children of the same family, aged respectively 10 and 8 years, had suffered for some years from well-marked attacks of migraine, frontal headache with nausea and vomiting, photophobia, a preference for dark quiet rooms. In the one case during the pains there was ecstatic excitement and great tenderness of the hair on combing, which disappeared during the intervals. Attacks set in every few months; duration 2—4 days. Father suffers severely from migraine.

Anæmia, also, which often occurs in children, even so young as 5 or 6, and is met with even more frequently after the age of the second dentition, favours the development of migraine, in these cases is generally combined with giddiness. In the same way in the hysterical conditions of which I have already given you illustrations (p. 220), nervous headache is often complained of. In rare cases, even after the disappearance of such conditions (attacks of hallucinations, spasmodic twitchings, &c.), headaches with the character of migraine persist for some time. On the other hand, the female genital system, the diseases of which so often occasion headache in later life, scarcely calls for any consideration in childhood. On this account the following case—which is certainly the only one I have met with—seems to me all the more remarkable.

> A girl of 7 years, brought to the polyclinic on 2nd January, 1873. Had suffered since May, 1872, from attacks of migraine. Violent pains on the forehead and temples, nausea, extreme prostration, photophobia. Duration of attack a few hours. Re-

currence irregular. At the same time restless sleep with frequent twitching of the body. Since May, 1872, there had existed fluor albus; the entrance to the vagina much reddened, hymen normal.

Treatment—Lead fomentations, injection of zinci sulph. (¼ per cent.) into the vagina. Internally quinine, later pot. brom. After various ups and downs all the symptoms disappeared until December, when the fluor albus, and with it the attacks of migraine again came on. Further course unknown.

In cases of this kind we must remember that both the vaginal catarrh and the headaches may have arisen from irritation of the genital organs by masturbation, and we must investigate this particular. A connection between migraine and the presence of worms is oftener assumed than the facts justify. However, we will do well to direct our attention to this point, as I have seen a few cases in which headaches disappeared for a length of time after several round worms had been passed.[1]

In general I have found migraine in children less frequently unilateral than in adults, and more usually in the middle of the forehead. The duration of the attacks varied between a few hours and two days. In the latter case the intervening nights were often disturbed by restlessness, sensation of heat and talking during sleep. Vomiting, dread of a bright light and of sound, sometimes also general trembling and rapid breathing as in the hysterical attacks described, were common. The intervals were quite irregular in duration, lasting in some cases only a few days and in others for several weeks. Among the determining causes none were more frequent than the close atmosphere and mental exertion of school, so that many children had to be kept at home. Emotional causes of every kind, fear of punishment, and scolding, I have also known to bring on an attack at once. When removed from their ordinary surroundings into the country or into health resorts, they generally remained quite exempt from the attacks, which usually recurred soon after their return home. Even after very careful examination and observation, the most conscientious physician often remains in uncertainty as to whether the case is one of migraine or of headache caused by cerebral disease (tubercle, tumour). I have already men-

[1] On the connection of migraine with visual disturbances (asthenopia and hypermetropia) also with nasal affections (swelling of the turbinated bones), I have no personal experience. *Cf.* Blache, *Revue mens.*, Avril, 1883; and Sommerbrodt, *Berl. klin. Wochenschr.*, 1885.

tioned (p. 275) that such affections may for a long time only indicate their presence by headaches which have all the characters of migraine, and the diagnosis can therefore only be established by observation during the intervals, carried on for some time, and by an exact investigation of the etiological conditions which I have already described.

According to the nature of these conditions the treatment must be directed. While we are powerless in regard to the hereditary tendency, we must combat all the more resolutely the influence of mental strain. I do not overlook the difficulties which meet us here. Only under very favourable circumstances can we take the children completely away from school and have them taught by private tutors, so that they may have more time for bodily exercise and be more in the open air. I have frequently also seen good results when I took the children away from town schools and let their further education be carried on in schools or boarding-houses in the country. The majority of the little patients, however, are unfortunately fixed where they are, and the treatment is then all the more difficult, as not only the teachers, but also many ambitious fathers put their veto to the doctor's advice. All that remains then, is to limit the home-tasks, to arrange for regular recreation and to prolong the holidays as much as possible. The government regulations which have very recently been issued, aiming at a limitation of the mental work of children, are therefore to be gratefully acknowledged. We may expect much more from the carrying out of these instructions by the teaching staff, than from any course of medical treatment. The rubbing down with cold water after getting up in the morning, which is much recommended for strengthening the nervous system, and which indeed is quite the recognised treatment, has in my hands done little or nothing in these cases. Cold baths and swimming have been more effectual. In anæmic patients, iron is to be recommended. I know of no specific remedy. The much extolled quinine and bromide of potash, which I have tried in numerous cases gave very various results (quin. sulph. or muriat., grs. ¾ thrice daily, pot. brom., grs. viiss—xv. also thrice daily). A visit to the sea-side, or to hills and woods, and mental rest are more efficacious than any medicine, although the good result is in general only temporary. The "holiday-colonies" which have been started in our time

are therefore an inestimable benefit for the poorer classes. We must always bear in mind, also, that there may be an element of simulation and that the pains may be considerably exaggerated in order to get away from school. When there is a suspicion or certainty of masturbation, a serious representation of the danger—which we may purposely exaggerate—has in my experience far more influence than punishment with older children.

SECTION IV.

Diseases of the Respiratory Organs.

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

The mucous membrane of the nasal cavity, larynx, and bronchi is extremely subject to catarrhal affections, especially in children of the lower classes; these being allowed to expose themselves to all sorts of weather. The symptoms are very similar to those in adults—swelling and obstruction of the nose followed by increased secretion of muco-purulent matter, sneezing, catarrhal affection of the conjunctivæ, hoarseness, rough or ringing hollow cough, with or without rise of temperature. Such a catarrh is one of the constant prodromata of measles especially, as well as being caused by atmospheric influences. And during a measles epidemic you may in fact, from such a catarrh appearing in a child who has not hitherto had the disease, predict with the greatest probability that the eruption is about to follow. Under all circumstances a catarrh of the upper part of the respiratory mucous membrane in very young children, though it may be slight in degree, is always to be regarded as much more serious than the same at a later period of life. For experience shows that even a simple cold in the head may in a very short time occasion symptoms of laryngeal obstruction, or may extend rapidly into the deeper ramifications of the bronchi. Infants with coryza, or slight catarrh of the larynx and trachea should not, therefore, be taken out of doors, and must be carefully protected from cold air.

Less frequently than measles, but still often enough, scarlet fever and diphtheria may cause severe inflammation of the nasal mucous membrane, which in both cases is usually secondary to an already existing "diphtheritic" affection of the pharynx.

[1] *Cf.* the description of coryza neonatorum and syphilitica, p. 142 and pp. 93, 142.

The nose is more or less swollen, and an offensive purulent secretion flows from it over the upper lip, which as well as the nostrils is reddened and excoriated by it. The parts round the nose, as far as the eyelids, are œdematous and swollen in severe cases, the conjunctiva is congested, and the eye waters much owing to obstruction of the nasal duct. It is but seldom, however, that one can see the diphtheritic membrane in the nose, owing to its always being situated so far up that even when the alæ nasi are held apart it still remains out of sight. Far less commonly, the membrane extends so far downwards that it comes into view—a fact to which I shall return later on. It is even more difficult—usually indeed, impossible—to examine the naso-pharynx at this age with a mirror. The swelling of the nasal mucous membrane in those cases is so great that breathing is more or less interfered with and a snoring noise is caused, especially during sleep. In general this rhinitis is a bad omen both in scarlet fever and in diphtheria; still, in both these diseases it often occurs in a less severe form without exerting any bad effect. We shall see later on that diphtheria may also begin with an affection of the nasal cavity; but only once—in the case of a daughter of our never-to-be-forgotten Traube—have I observed an independent rhinitis pseudomembranosa. The case which follows, acquires an additional interest from the careful observations made by her father.

> The girl, who was 8 years of age, and generally healthy, took ill with symptoms of coryza, accompanied by moderate fever. The marked snoring during sleep, and the complaints about something obstructing the breathing near the root of the nose, indicated a more considerable stenosis of the nasal canal than usually occurs with simple coryza. Traube himself made an examination with the mirror and found nothing but a catarrhal redness on the pharynx and on the epiglottis. After a few days the child expelled with great difficulty a tough, white mass of the length of a finger-joint, which swelled up when treated with acetic acid—thus showing its fibrinous nature. After a few days a much smaller mass was ejected, whereupon all difficulty of breathing at once disappeared. The treatment had been almost solely expectant (rest in bed, and a few doses of calomel).

Was this a case of true diphtheria confined to the nasal cavity, or was it only a non-specific croupous rhinitis?

A chronic form of rhinitis occurs very often in scrofulous

children, along with other more or less pronounced symptoms of this cachexia—eruptions on the head, ophthalmia, otorrhœa, eczema in the face and enlargement of the cervical glands. The commonest symptoms of this disease are external swelling of the nose, snuffling and snoring breathing, the trickling of a sero-purulent secretion out of the excoriated nostrils, and redness and swelling of the upper lip. Not uncommonly this chronic rhinitis gives rise to repeated attacks of erysipelas, which, extending from the nostrils, spreads over both cheeks or even still further, forming a wing-like outline (p. 48). But even where there is no tendency to scrofula, chronic rhinitis may be left as the result of measles, scarlet fever, or even of very severe coryza. In all such cases, besides using anti-scrofulous remedies (to which I shall return later), I have the nose painted daily with a solution of nitrate of silver (grs. xvi to ℥i) and this usually succeeds.

The application of iodoform, in powder or as an ointment, has proved useful. I must also mention in passing the rhinitis which may be caused by foreign bodies—peas, beans, &c.—in the nose, and which at first at least is usually one-sided.

In a large number of children there is a marked tendency to catarrhal affections of the entrance into the larynx, which develope very rapidly when the children get coryza. In such cases one must be prepared, when the slightest coryza begins, for one of the attacks which we are about to describe, and which on account of their resemblance to croup have been called "false croup."

II. *False Croup.*

When you find that a child has had "croup" 4 or 5 times, you may always be sure that the disease has been false and not true croup. Although usually not dangerous, false croup is a very alarming disease and one of the most inconvenient for the physician; for it is especially apt to cause him to be roused in the night time.

The disease always begins suddenly, usually following immediately on a slight coryza (snuffling, sneezing) and almost always in the night, often soon after entering on the first sleep. The children start up from sleep in a fit of coughing.

The cough is hoarse and hollow, quite resembling that of croup. The household is at once thrown into a state of alarm. Not only the cough, but—almost even more so—the deep inspirations which interrupt them, are accompanied by a distinctly croupy, sawing noise; and this is also heard between the whimpering and crying which little children are wont to set up in these circumstances. The cry itself may at the same time be either quite normal or a little hoarse. During this attack many children sit up in bed with an anxious expression and flushed cheeks, with laboured and noisy breathing, are extremely restless, and repeatedly catch at their throat. The child is hot, often covered with sweat, the pulse rapid. A fit of this kind usually lasts some minutes, but even after it is over the breathing often remains somewhat noisy and more frequent than in the normal condition. The physician is called in haste. By the time he arrives the child is usually comparatively quiet, or even asleep, the accessory muscles of respiration taking little or no part in the breathing—except for, perhaps, a slight movement of the alæ nasi. He may from these facts draw the reassuring conclusion that the obstruction to the breathing is not of a serious nature, and that as yet, at any rate, it is not a case of true croup. If one remains some time at the child's bedside, one is very likely to witness a repetition of the attack. At any rate, when the children wake out of sleep they generally begin to cough again with a croupy sound, and when they cry or sob their inspirations are harsh and prolonged. Pressure applied to the larynx and trachea at once brings about one of these attacks of coughing. The children are generally quite well next day, and there is nothing now except an occasional hoarse ringing cough to remind one of the violent symptoms of the night before. Sometimes the same scene is repeated on the following night, and I therefore always prepare the parents for this possibility. In most cases, however, the attacks do not occur after the second night,[1] and there remains only an ordinary loose cough, which may last 8—14 days. You see, then, that the course of the disease being such, there is no danger to be apprehended; but the troublesome thing is its frequent recurrence. Some

[1] Cases in which an attack occurs 12 nights in succession, as in one observed by Monti, are surely very exceptional (*Ueber Croup und Diphtheritis:* Wien und Leipzig, 1884, S. 18).

children are attacked by it repeatedly in the course of a single year, and its resemblance to croup inspires such terror that very few parents, in spite of their previous acquaintance with the disease, are considerate enough to leave the doctor undisturbed.

When we examine the fauces in a case of false croup, we find at most slight catarrh and redness. By means of a laryngeal mirror one may make out a swelling of the lower and inner portions of the vocal cords (inflammatory œdema below the cords), which rapidly spreads upward, but which may also subside in a few hours.[1] It appears, therefore, to be a catarrh spreading downwards from the nasal cavity into the larynx; and along with it, as in every coryza, the swelling increases, especially during sleep, and occasions a sudden awaking with want of breath, anxious feeling, and hoarse cough. The dryness of the cough, and of the breathing, is usually diminished by warm drinks (eau-sucré and milk); and with the commencement of a copious catarrhal secretion, all cause of anxiety completely disappears. The physician will therefore do well in such cases not to display too great energy at once; but rather to take an expectant line of treatment. I am in the habit of ordering frequent drinks of warm water or milk, with wet compresses, also, perhaps, hot poultices to the neck. But under all circumstances the children must be kept in bed for two or three days till the resulting catarrh has time to develope. The continuous application of a piece of bacon over the front of the neck is also to be recommended, as it generally causes a slight erythema or an eruption of small pustules. In the great majority of cases I have succeeded very well with the treatment I have mentioned, and I therefore consider the custom of giving an emetic at once in all such cases unwarrantable. In families where false croup is, so to speak, endemic—a not very uncommon occurrence—the mothers usually have emetics at hand so as to be able to give them before the doctor arrives. I must protest very strongly against this abuse, which weakens the children quite unnecessarily. There is no remedy against the recurrence of the attacks.[2] Inuring to cold is of no use; careful protection from chills is far better. Many children commence to suffer

[1] Rauchfuss and Delhio, *Jahrb. f. Kinderheilk.*, Bd. xx.

[2] I have no experience of the pot. iod. (1–2 p. c. solution) which Monti recommends.

from these attacks of "croup" in their 9th or 10th month. The attacks become less frequent or less severe, and usually disappear of themselves about the 6th or 7th years of life. Such children must be carefully protected from cold weather, and kept indoors, especially when they have a cold in the head. But even this does not always insure immunity from false croup.

The development of measles or whooping cough is sometimes ushered in by quite similar attacks. Both diseases—especially measles—may begin with such an attack; which then passes into an ordinary catarrh, manifesting its real nature in the case of measles after a few days, in that of whooping cough after one or two weeks.

From the description I have given you, you might be inclined to regard false croup as in every case a trivial affection, and one free from danger. But although this is true of the great majority of cases, you must not be misled into over confidence, or neglect keeping an eye on the child for some days after the first attack. Although very rarely, I have occasionally seen true croup (confirmed by the expulsion of false membrane, or by post-mortem examination) develope in 36—48 hours after such an attack of false croup. The possibility of this makes it incumbent upon you in every case to keep the children in their rooms till the catarrh is fully developed, *i.e.* so long as the cough has a slightly croupy character, or a hoarse sound is audible on forced inspiration.

III. *Atelectasis of the Lungs.*

In all the respiratory diseases of children, the tendency of the lungs to "collapse" is a fact of the very utmost importance. This peculiarity, which is known as "atelectasis" consists in the tendency which the pulmonary alveoli have to become empty of air and sink in in such a way that their walls touch one another. At the post-mortem examination of most children who have died of diseases of the respiratory organs and also of many exhausting diseases of other kinds, you come upon sharply defined, bluish-red, or steel-grey patches, varying greatly in size, situated on the surface of the lungs, especially along the anterior margin, and the lower and inner border of the lower lobe, likewise on the

"lingula," which overlaps the pericardium. These are somewhat depressed below the surrounding level, they are sometimes quite superficial in position, isolated, and of small size; at other times they are more extensive and run together so as to form elongated areas or rounded patches as big as a half-crown, or bigger. On section, these patches are tough and non-crepitant, no air-bubbles issue from them, but only a little bloody fluid; and they sink in water. The surface of the section is smooth, and on it we can easily see the fibrous septa of the lobules in the form of white streaks. The collapsed portions of lung were long held to be pneumonic patches; but with these they really have nothing in common, except the "consolidation" of the lung tissue. The nature of the pathological process was first recognised owing to the simple experiment suggested by Legendre and Bailly of blowing air through a tube into the communicating bronchus. For whereas inflation has no effect on pneumonic consolidation, parts which are only collapsed immediately become blown out, and assume a bright red colour.

Two factors in the causation of atelectasis may be indicated with certainty. In the first place, a lowering of the inspiratory power which is too weak to drive the air into the alveoli; and secondly, the filling of the bronchi with mucus, rendering it difficult for the air to pass through them. When the air can no longer obtain entrance into the alveoli, that which is already contained in them is absorbed by the circulating blood, and the alveoli collapse.[1] You will find the atelectasis most frequent and most extensive in those cases in which both these factors mentioned are at work, and therefore in all exhausting diseases which are accompanied by bronchial catarrh. For this reason we also meet with atelectasis under similar circumstances in adults, *e.g.* in typhus; but generally it is much less common and less extensive in them than in little children, whose inspiration even in health is comparatively much weaker. Rickety children with narrow chests are particularly liable to atelectasis; for in them a third factor is added to the already mentioned causes (weakness of inspiration and bronchial catarrh), namely, a narrowing of the capacity of the chest, which hinders the full expansion of the lungs. Also, in stenosis of the larynx, trachea, or large and small bronchi—whether due to inflammatory and

[1] Lichtheim, *Archiv f. exper. Path.*, x., S. 54.

cicatricial processes, the presence of foreign bodies, or compression of the air-passages—numerous patches of atelectasis of the lungs may occur, from interference with the entrance of air into the alveoli, along with the increasing weakness of inspiration present during the later course of the disease.

Although we so often find atelectasis of the lungs in children after death, one is rarely able to diagnose it during life. This difficulty of diagnosis is all the more to be regretted, as the addition of atelectasis to those diseases which it is wont to accompany, is by no means a matter of indifference. Although the assumption that slight hyperæmia of the lung-tissue resulting finally in broncho-pneumonia occurs in the collapsed areas as the result of deficient atmospheric pressure on the vessels, is not proved—and is indeed rendered doubtful by certain experimental facts[1]—still, we must always regard the increased insufficiency of the lung from patches of atelectasis as a factor which makes the prognosis very considerably less favourable. The difficulty of the diagnosis is due to the fact that the patches of collapse scattered through the lung-tissue occasion no visible signs whatever, as they are completely masked by the air-containing portions and by the bronchial sounds. Even extensive areas of collapse, *e.g.* when a large part of the lower lobe is affected, give rise to no physical signs beyond those of consolidation (dull note, bronchial breathing, &c.), which can in no way be distinguished from those of pneumonic consolidation. The only conclusive point for a diagnosis of atelectasis would be the absence of fever, did we not know that in little children in a state of extreme exhaustion even pneumonia occurs without rise of temperature; and that, on the other hand, atelectasis very frequently occurs as the result of febrile diseases (bronchitis, croup, typhus). For these reasons we can never, in my opinion, regard the diagnosis of atelectasis as certain; for it is at best only probable, although justified by the results of post-mortem experience, *i.e.* by the frequency with which this affection is found in certain diseases and in conditions of exhaustion in children.

The congenital form of atelectasis, which first became known through the work of Jörg,[2] is quite different from that

[1] Traube, *Beitr. zur experiment. Pathologie und Physiologie*, Heft 1, 1846, Experiment 63.

[2] *Die Fötuslunge im gebornen Kinde., u. s. w.:* Grimma, 1835.

which we have just been considering. In it we have to do with a persistence of the fœtal condition in a more or less extensive portion of the lungs. The parts affected have not yet been used in breathing, and therefore are tough, steel-blue, and sink in water, as is the case in the fœtal lung, being thus in the condition which we have already seen as acquired by the weakness of the inspiration or by the exclusion of air from the alveoli. For these reasons we usually speak of the latter form of atelectasis as a return of the lung-tissue to the "fœtal condition." In general, the causes active in congenital atelectasis are quite the same as those of the first form; especially a failing or very weak respiration such as occurs in asphyxia, or in premature and debilitated children. Obstetricians, therefore, have the most frequent opportunities of observing this affection, which is rarely met with by physicians even in children's hospitals. As a rule, congenital atelectasis is much more extensive than the acquired form; and not only presents distinct symptoms of consolidation on physical examination, but also causes engorgement of the pulmonary artery and of the general venous system with cyanotic discoloration, owing to material interference with the circulation. For the same reason, the closure of the channels of the fœtal circulation, especially of the foramen ovale, does not always occur in the normal way. Many such infants die very soon after birth from the atelectasis and the debility which has occasioned it. Still, in a certain proportion of the cases in which the consolidation does not affect both lungs to too great an extent, and the circumstances are otherwise favourable (sufficient care, and the choice of a good wet-nurse), one may succeed in increasing the general strength and rendering the collapsed portions of lung once more air-containing.

Thus, in May, 1880, a child of 3 weeks was brought to me, who had been born prematurely in a state of extreme debility, had become cyanotic in the first week, and had suffered from several violent attacks of dyspnœa. On the right side posteriorly there was dulness over almost the whole of the space between the spine and the scapula. The normal breath-sounds were absent there, and in their stead crepitations were heard. The left side appeared quite normal. There had never been any fever. A suitable nurse was procured, wine was given, and camomile-baths were used. The child throve well. When I examined it again, the percussion-note on the right differed but little from that on the left side; the

vesicular breathing was still weak, but distinctly audible. In October, the child (now well nourished) was found to have only a slight bronchial catarrh.

I believe that this case may be regarded as one of congenital atelectasis of a large part of the right lower lobe; since the condition existed from birth, there was no fever, and good nourishment was sufficient to remove gradually the threatening symptoms. In the following case, on the other hand, we see a fatal issue, happening under conditions that were extremely unfavourable.

A child of 6 weeks, left on a doorstep in severe winter weather by a mother unknown, and admitted into the ward on 8th January, 1873. Very small and wasted; cyanotic colour of the lips and eyelids, veins of the head and face distended, respiration extremely weak and superficial, instead of a cry only a plaintive whining. Percussion-note all over somewhat impaired, but nowhere distinctly dull; the breath-sound only heard very faintly; no râles. Heart sounds normal. Too weak to suck from the bottle, and had to be fed with a spoon. Thrush in the mouth and throat. Temperature subnormal (97·2° F.). Little improvement, in spite of good milk, wine, and the best nursing. As the inspiratory movements increased in strength, the cyanosis disappeared, but always returned when the respiratory movements got weak again. Death on 16th February in a state of collapse.

P.-M.—Heart normal. All channels of the fœtal circulation closed. Thrush of the œsophagus. Uric acid infarcts in the kidneys. Otherwise everything normal except in the lungs. The greater part of both lower lobes collapsed, but in such a manner that air-containing portions are visible between the consolidated areas. Also in the other lobes, scattered patches of atelectasis. Bronchi normal.

IV. *Inflammatory Affections of the Larynx and Trachea.*

Acute catarrh of the upper air-passages either arises suddenly with an attack of false croup, or gradually with increasing hoarseness and rough and ringing cough. There are children as well as adults in whom every cough, even when it lasts for weeks, has a hollow metallic sound, although they may have no other signs of the larynx being affected; in particular, no alteration of the voice. In considering each individual case, this peculiarity must be kept in mind, because it is apt to lead to

unnecessary anxiety. On the whole, a hollow metallic cough is much less ominous than a hoarse husky one; which, when combined with more or less thickness of the voice, is always a cause of anxiety. If in such cases you exert a moderate pressure with the finger on the trachea or larynx, the children not only make a face as if in pain, but also usually give a cough with the rough, hoarse character which we describe as "croupy." The inspiration, especially during crying or screaming—that is to say, when more air is required—is accompanied by a sawing sound, although the breathing may meanwhile be perfectly quiet, without a trace of dyspnœa. In the first few days after recovery from an attack of false croup I have often been hurriedly called back because violent laryngeal symptoms had re-commenced; and in these cases I have almost always found that a fit of bad temper in the child, with crying and screaming was to be blamed for it. Whenever the agitation ceased, the threatening symptoms at once subsided. It is therefore advisable to prepare the parents for these exacerbations, and to let them know that they are not dangerous. They are of importance only so far as they indicate that the catarrhal condition in the larynx still exists, although in process of resolution. To these local symptoms loss of appetite, coating of the tongue with mucus, and also often a moderate fever with evening exacerbations are added. Such cases always require the physician's utmost attention; for one can never foretell whether the disease may not become threatening within a few hours.

It is under such circumstances that emetics (Form. 6)—against the abuse of which in simple cases of false croup I have just warned you—have their proper use. When these have done their duty you may order an expectorant mixture (Form. 15) and wet compresses round the throat. The child must be kept in bed till the cough has lost every trace of its croupy character, and the inspiration has become absolutely noiseless. Under this treatment the catarrh usually improves within a few days; the cough becomes loose and rattling, the hoarseness disappears, and after 8—14 days, as a rule, recovery is complete. Still, one must always be prepared for the possibility of the disease getting worse, as it may do in spite of the most careful nursing. But usually this results from some want of care, and it is therefore particularly common in practice among the poor. Then, the

symptoms which hitherto have only appeared serious to the initiated, may within a few hours reach such a height as to considerably endanger life. This violent aggravation is due either to a rapidly increasing catarrhal swelling, or to a fibrinous exudation on the inflamed mucous membrane; or, finally, to an œdematous or sero-purulent infiltration of the arytœno-epiglottidean ligament and its neighbourhood. These different pathological conditions give rise to almost the same clinical symptoms—those of acute laryngeal obstruction, which we have next to consider.

To the symptoms already described—huskiness, hoarse cough, tenderness of the larynx and trachea on pressure, and noisy inspiration and expiration—are now suddenly added dyspnœa, working of the alæ nasi, movement of the head in breathing, and increasing retraction during inspiration of the episternal and epigastric regions, finally of the whole lower part of the thorax. At the same time, however, the frequency of the respiratory movements is scarcely increased; and even in severe cases it rarely exceeds 24—28 in the minute. The individual inspirations and expirations, which are accompanied by an uncomfortable sawing noise, are on the contrary unusually prolonged.[1] All this time, the child may feel almost quite well. A girl of 4 years took ill on 30th March with false croup. In spite of an emetic, the symptoms got worse; and when she was brought to the polyclinic on 1st April, there was the most extreme dyspnœa, croupy cough, sawing noise with breathing; but the child, all this notwithstanding, ran and played about the room. The expulsion of dichotomously branched portions of false membrane soon established the fact that it was a case of real croup. The hoarse stridor, which in all such cases accompanies the inspiration (also often the expiration), may be best compared with the to-and-fro noise of a saw in cutting wood. It is not always equally loud. It is less marked after vomiting, or may even disappear entirely for a short time; it is *most* marked during sleep, at which time it is so loud that it arrests the physician's attention as soon as he enters the room, and at once announces to him the nature of the malady.

During the further course of the disease, should the treatment

[1] On the significance of this symptom, *cf.* Cohnheim, *Vorlesungen über allgemeine Pathologie*, ii.: Berlin, 1880, S. 163.

be unsuccessful, the symptoms of obstruction increase almost hourly. The child often catches at his neck as if trying to remove the obstruction to the breathing, and bends its head forcibly backwards. The complexion, which has hitherto been natural, becomes pale and cyanotic, the eyes are anxiously directed to those around, as if imploring assistance, and on the forehead and cheeks clear drops of sweat are often to be seen, though the skin does not appear warmer than usual, and indeed is usually colder on the tip of the nose and on the cheeks. Along with the dyspnœa, the hoarseness of the voice rapidly becomes more marked and increases till there is complete aphonia; and at the same time the cough which was formerly hoarse and ringing, gradually becomes more toneless, and finally is almost quite extinguished—at any rate is more visible than audible. Fever is not an important feature in the course of this disease; for although it is never quite absent, yet the temperature but seldom reaches a very high degree. It usually varies between 101·3° F. and 104° F., with remissions in the morning hours; while the rate of the pulse is not unfrequently raised to 144 or more by the child's continual restlessness.

The group of symptoms described only permits (as I have already mentioned) the diagnosis of acute laryngeal obstruction. What the cause of this is, cannot at once be decided. First of all you must examine the pharynx carefully, to ascertain the presence or absence of diphtheritic patches on the mucous membrane. Should you find these, the diphtheritic nature of the obstruction is thereby rendered certain. Should you, however, find no patches, you must not on that account at once deny the possibility of the disease being diphtheritic; because, as we shall see afterwards, the patches in the pharynx may escape our observation during life, or may have already fallen off. When it is possible to use the laryngoscope successfully, we certainly gain a clearer insight into the nature of the disease. But, considering the difficulty of this examination in childhood (p. 10), you cannot expect to make much of it except in a small proportion of the cases. If you can with certainty exclude diphtheria, then it must be either simple or pseudo-membranous (fibrinous) laryngitis (croup). For it has been proved beyond doubt that the most violent dyspnœa—in fact, all the symptoms of croup—may also be caused by acute

laryngitis with swelling of the laryngeal mucous membrane only, and no croupous exudation. Such cases are naturally much easier to cure by anti-phlogistic treatment, than the pseudo-membranous form.

Marie F., 6 years old, healthy, took a violent attack of false croup on the night of 7th December (during an epidemic of measles). Next day she seemed well till 1 P.M., when suddenly such threatening symptoms came on that I was summoned in the greatest haste. Sawing noise with respiration, face cyanotic, covered with sweat. Head bent back, forced action of accessory muscles of respiration, eyeballs upturned between the half-opened lids; cough, excited at once by pressure on the larynx, was short, hoarse, and accompanied by a whistling sound. Voice also hoarse. Nothing abnormal in the throat; could drink without difficulty. The vesicular breathing completely masked by the laryngeal stridor. Sonorous rhonchus could be made out at the root of the lung only. Pulse 120; skin hot and perspiring. I ordered 6 leeches over the manubrium sterni, allowing no after-bleeding; and, internally, antim. tart. (gr. $\frac{1}{8}$ in aq. destill. every 2 hours). As there was no vomiting by 5 P.M., I gave as an emetic full doses of pulv. ipecac. and antim. tart., after which there was repeated vomiting. At 8 o'clock I found the child somewhat quieter, sitting on its mother's knee; the stridor less, the voice clearer, and the skin perspiring freely. I gave the solution of antimony again, and applied a blister to the larynx. After a quiet night, I found on the 9th that the stridor had almost quite disappeared, the breathing was quiet, and the cough lessened. After each spoonful of the medicine, vomiting followed, but no purging. The blister had raised a large bulla, which I opened, and ung. hydrarg. was then applied. About 2 P.M. a fresh exacerbation of the laryngeal symptoms took place, owing to the administration of an enema, against which the child struggled violently. But when the child was quieted, these symptoms soon subsided. From this time onward rapid improvement took place. The cough became loose, and disappeared about the 15th, under the use of an expectorant mixture.

You have here an example of a thing which I have already spoken of, namely, the development of serious laryngitis from what was at first false croup; and at the same time of the efficacy of energetic anti-phlogistic treatment, which in such violent cases I cannot too emphatically urge upon you. You should at once have 2—6 leeches (according to the age) applied over the front of the neck. The best position is just over the manubrium sterni, in order, on the one hand to keep the region of the larynx free for further external application,

and, on the other, to avail ourselves of the underlying bone for the compression of the leech-bites should the bleeding be excessive. The after-bleeding of the leech-bites, which it was formerly the custom to encourage, is inadvisable. When the leeches drop off the bleeding should be at once stopped. The use of cold compresses, or of an ice-bag over the larynx, I do not consider sufficient in these cases. I have frequently witnessed a marked alleviation of the most violent dyspnœa even during the blood-letting. The debility and temporary anæmia which may possibly result from very copious bleeding ought not to deter you; for the risk of such an occurrence is far less than that which the child is exposed to when death is imminent from inflammatory obstruction. After blood-letting, I give an emetic, or tartrate of antimony in divided doses (Form. 18) which, as we have seen in the above case, by no means always causes vomiting or purging. If the case is carefully watched, and the tartar emetic stopped at once whenever diarrhœa or excessive vomiting sets in, no bad results—as far as my experience goes—will ensue. But in practice among the poor, where the remedy has often to be left in careless hands, dangerous symptoms of collapse may certainly sometimes be occasioned. In such cases, therefore, it is always better, instead of continuing to use tartrate of antimony, to give a full dose of some emetic whose action can be more easily counted on and controlled. Inunctions of mercurial ointment (grs. x. twice or thrice daily) into the sides of the neck, and finally a blister over the larynx (to the sore which it leaves I generally order ung. hydrag to be applied), complete the list of remedies to be recommended for these severe cases of acute laryngeal catarrh. The remarkable rapidity with which the threatening symptoms disappear in cases like the one just given and that which follows, proves that it can really be nothing but a catarrhal swelling of the mucous membrane.

Paul B., 2 years old, admitted on the evening of 17th October with extreme dyspnœa. Face cyanotic, eyes prominent, with an anxious expression. Inspiration prolonged and sawing, all the accessory muscles of respiration in action; croupy cough, especially marked at night. Tonsils swollen, no patches on them, voice extremely hoarse. The epiglottis felt normal, pulse 160, temp. 102·5° F. Symptoms had lasted for 2 days. Emetics. Next day the cyanosis and difficulty of breathing were almost gone; the child sat in bed playing; the cough and inspiration still

croupy; temp. 101·8° F. Antim. tart.; inunction with ung. hydrarg., grs. xxx. in the day. Next day free from fever. A blister applied over the larynx on account of the persisting hoarseness and the harsh noise on forced inspiration. Discharged on 24th October.

If you only consider the troublesome narrowing of the nasal cavity which may suddenly take place in any ordinary severe cold in the head from increased swelling of the mucous membrane, you will easily understand how in catarrh of the larynx and trachea, very acute swelling may in like manner arise, only with very much more threatening symptoms; and, under suitable treatment, may almost as quickly subside again. This condition, however, may lead to a fatal termination—all care notwithstanding. For, an extreme serous or sero-purulent infiltration of the vocal cords and of the epiglottis and its folds (the so-called œdema glottidis; better, laryngitis submucosa) is very easily added to any inflammatory process in the neighbourhood of the glottis, thus causing sudden danger of suffocation. Therefore, not only in cases of acute laryngeal catarrh, croup, and laryngeal ulcer, does this danger threaten; but it is also to be apprehended in severe pharyngitis, in abscesses of the tonsils and in deep phlegmonous conditions of the cervical connective tissue. In England a scald of the gullet and of the entrance to the larynx with boiling water which the child has drawn in by sucking the spout of a tea-kettle, is a frequent cause of this laryngitis submucosa; but I have never myself met with any example of this. In all these cases, when "œdema glottidis" sets in, the symptoms of dyspnœa and obstruction, already described, reach such a height that suffocation is to be apprehended at any moment. Sometimes by introducing the finger deeply one may feel the greatly-swollen epiglottis, or one may even see it projecting upwards behind the tongue. The speedy performance of tracheotomy is now the only means left of saving life.

The danger in the acute laryngitis of children lies, however, not so often in the above-mentioned condition, as in the tendency to fibrinous exudation on the inflamed mucous membrane. While in the form we have hitherto been considering, the autopsy shows only more or less dark redness and swelling of the mucous membrane—at most, superficial erosions on it, and

a sero-purulent infiltration of the swollen epiglottis and its neighbourhood, especially of the arytæno-epiglottidean ligaments and of the vocal cords—here, we find on the mucous membrane of the larynx and trachea isolated patches, or larger pieces of false membrane of a greyish, or yellowish-white colour, either of gauze-like delicacy, or 1mm. or more thick, and in that case consisting of several layers—the outer of which (*i.e.* that next the mucous membrane) is usually the most recently formed, and the least tough. This membrane, which is seen microscopically to consist of an extremely fine fibrinous net-work and numerous young cells (epithelium, pus-corpuscles) often extends down the trachea, as far as the bifurcation, or even beyond that point into the large and middle-sized bronchi, there forming cylindrical casts of these tubes which can easily be drawn out of them, as they are not adherent but lie quite loosely on the surface. When the false membrane is removed we find the mucous membrane more or less reddened and swollen, but occasionally pale and without a trace of vascularity. Bronchitis and broncho-pneumonia are almost constant accompaniments, as are likewise emphysema of the upper, with numerous patches of collapse in the lower lobes.

In regarding croup as the highest development of acute laryngitis, I am directly at variance with those physicians who regard it as being invariably diphtheritic, and who absolutely deny to it any other mode of origin. I grant that since diphtheria became endemic and epidemic in Germany, croup has been much commoner. But I do not see in this any ground for denying the possibility of its originating in any other way. We know from experiments that the most typical tracheal croup can be produced in rabbits and dogs by various caustics applied to the mucous membrane, as well as by making them inhale hot steam through a canula introduced into the opened trachea. We may therefore readily assume that in human beings also, strong irritants—such as the inhalation of cold air, or the action of cold on the surface of the body—which when slight in degree only cause catarrh, may, when they act more strongly, produce croup. It is not yet settled whether Weigert and Cohnheim are right in thinking that if the epithelium, which in catarrh always remains intact, dies and is washed away by secretion, the fibrinous exudation secreted by the inflamed mucous mem-

brane coagulates, thus forming the croupous membrane. The irritation of the infective material of diphtheria—perhaps the inhalation of it from the pharynx—is certainly in this country the commonest, but by no means the only cause of croup. For any severe catarrh of the larynx may lead to it; and consequently in measles—a disease which from its very beginning always occasions a catarrh of the larynx and trachea—this condition may pass into croup at a very early stage, without there being any question at all of diphtheria.

> Boy of 3 years, admitted on 29th May, 1873, with measles in process of eruption. Rash upon the face; pulse, 150; temp., m., 103·1° F.; e., 104·9° F. Severe catarrh of the larynx. Hoarse, almost inaudible cough; voice also hoarse. On the most careful examination nothing could be discovered but a spotted redness of the palate and a simple sore throat. Treatment:—leeches over the manubrium sterni; antim. tart. Marked improvement on the following day:—pulse, 116; temp. 101·1° F.; resp. 32. Only the hoarseness was still unchanged, and the cough had still a laryngeal character. Thus 4 days passed without any fever, during which the above-mentioned laryngeal symptoms continued. Suddenly, on the evening of June 5th, the temp. again rose to 101·3° F., and on next morning to 103·1° F. About midnight well-marked croup set in, so that tracheotomy had to be performed next day at noon during the clinique. When the trachea was opened we drew out of it a long cast, which reached down to the bifurcation. Other fragments were also coughed up afterwards. The tracheotomy tube was removed on the 10th day. Complete recovery.

I have elsewhere published some cases tending to prove the existence of a primary inflammatory croup unconnected with diphtheria. The children were aged 7 and 15 months respectively; and at the post-mortem, croup of the larynx and trachea was found, without the slightest change in the pharynx. Since then I have had repeated opportunities of observing the same thing—not to mention the still more numerous cases in which no post-mortem could be made, and which I therefore cannot regard as completely satisfactory proofs; because there was certainly a possibility of the diphtheria having escaped our notice from being situated deep-down in the pharynx. On the other hand, it must be admitted that the following case is conclusive.

> Max R., 1½ years old, admitted 4th April, 1877, with rickets and slight bronchial catarrh. In the next few days a further

extension of the latter; mucous râles on both sides, both in front and behind. On the night of 9th—10th sudden croupy respiration and harsh cough. On the forenoon of the 11th, fully-developed croup. Over the lungs the croupy sound is heard, propagated from above—the breathing is harsh, and there is sibilant rhonchus behind. Temp. 102·2° F.; pulse, 144; resp. 42. In spite of strong emetics, the symptoms got worse on the following day. The temp. remained at 104·7°—105·6° F.; respiration, 48. Child extremely languid and drowsy. Death on 12th. *P.-M.*—Pharynx unaffected; croup of the larynx and trachea, œdema glottidis, double broncho-pneumonia; rachitis.

Such cases, beginning with bronchial catarrh and suddenly passing into fibrinous tracheo-laryngitis, are described under the name of "ascending croup." I have observed this manner of onset especially in children in the first years of life; also several times in the course of whooping cough and in diffuse bronchial catarrh occurring along with that disease. Tracheotomy under such circumstances is almost always unsuccessful, owing to the extensive bronchitis and multiple broncho-pneumonia.

Ernst G., 4 years old, admitted 21st March, 1877. Said to have taken ill 8 days before with an attack of false croup, and never to have been quite well since. Yesterday, at midday, sudden dyspnœa came on, rapidly getting worse. On admission he was cyanotic and collapsed. All the symptoms of croup were well-marked. Only redness and slight swelling in the pharynx. Tracheotomy at once, and lime-water inhalations. After some hours pieces of false membrane were coughed up. Among these was one cylinder which represented a complete cast of the trachea and commencement of both bronchi. Lessening of the dyspnœa followed, but increase of the collapse and continuance of the cyanosis. Evening:—pulse 168; resp. 54. Death during the night. *P.-M.*—Pharynx unaffected; croup of the larynx and trachea, extending into the large bronchi; double broncho-pneumonia; chronic fibrous endocarditis aortica; left ventricle hypertrophied.

Elise W., 3¼ years old, admitted 6th November, 1876, with a relapse of hereditary syphilis. Recovery under corrosive sublimate injections, about 1st December. On the 6th, huskiness; hoarse cough; redness of the pharynx; no fever. In spite of leeches, emetics, and mercurial inunctions, the symptoms got so rapidly worse that on the 7th tracheotomy had to be performed. After this, inhalations of lime-water spray. During the following days there was a remittent type of temp. (evening, up to 103·3° F.), and the frequency of the resp. rose to 60, finally to 72 in the minute; and a double broncho-pneumonia developed, with loud

râles and varying impairment of the percussion-note. Death on 18th—*i.e.*, 11 days after the tracheotomy. *P.-M.*—Pharynx perfectly normal; croup of the larynx and of the upper part of the trachea in process of recovery; extensive bronchitis and broncho-pneumonia.

Anna S., 2 years old, admitted 28th February, 1879, with laryngitis, which had lasted 2—3 days. Pharynx quite normal. Tracheotomy not performed, owing to presence of diffuse bronchitis. Death on 2nd March. *P.-M.*—Diffuse bronchitis and broncho-pneumonia. Pharynx but slightly reddened, completely smooth and healthy; croup of the larynx and of the trachea, reaching to the bifurcation.

Ella S., 6 months old, after suffering for some months from tracheal catarrh, was admitted on 15th March, 1879, with commencing croup. The symptoms got worse; tracheotomy was performed on the 19th. Fever (104° F.) and dyspnœa persisted after it. Death on following day. *P.-M.*—Pharynx quite normal. Croup of the larynx. Bronchitis, with numerous patches of broncho-pneumonia. Caseous degeneration of the bronchial glands and of a part of the left upper lobe.

In such cases as these—and I have met with many others since—is one justified in entrenching oneself behind the assumption that diphtheria has passed over the pharynx and has developed first of all in the larynx and trachea? Such an assumption I consider quite arbitrary. The unprejudiced observer who attentively follows the clinical development of the disease alongside of the pathological condition will be able in every one of these cases to assume a mere local inflammatory process which has nothing to do with infectious diphtheria. The commencement with symptoms of simple tracheal and bronchial catarrh, the absence of pharyngitis and of all premonitory symptoms of infectious disease, and also of glandular swellings under the jaw—are sufficiently characteristic. This view of mine is not rendered untenable even by the instances in which a case of simple croup is said to have given rise to diphtheritic affection in those near the patient,[1] because in these cases it is impossible with absolute certainty to exclude other sources of infection.

The clinical symptoms of croup present the most extreme degree of the acute obstruction of the larynx increasing hourly in severity, and in fatal cases having usually a duration of from 24 hours to 3 or 4 days. Even if short remissions occur during this time—generally as the result of artificially produced vomiting

[1] *e.g.* Demme's 24. *med. Bericht*, 1887, S. 14.

—still, these are almost always deceptive. The dangerous symptoms soon reappear and a steady progression from bad to worse becomes only too evident. In many cases the steadily advancing course is interrupted from time to time by attacks of extreme suffocation. The child throws itself violently back, panting; the breathing is quite arrested; the face is cyanotic; the little hands are convulsively clenched, and death appears imminent. But after a few seconds, and with difficulty the air once more begins to enter the larynx with a whistling sound, and the child returns to its former state until a similar attack again comes on. Perhaps we have really here to do with attacks of spasmus glottidis, excited reflexly by the inflamed mucous membrane. At this stage the sawing respiration is often audible even outside the door of the sick-room, while the aphonia increases and the croupy cough becomes less frequent and more toneless. The restlessness of the children increases enormously; they want out of bed into the nurse's arms; then they want back again into bed, looking imploringly for help to those round about. This distressing condition is only interrupted by short periods of sleep, in which the laryngeal stridor reaches its loudest. The examination of the lungs yields, usually, no result, owing to the sawing noise which drowns all other sounds. At most, dry or moist râles are heard at different places; and, rarely, impairment of the percussion note, indicating that the lung-tissue has become affected. When this is the case, the number of the respirations also—which, in uncomplicated croup, as we saw above, either remains normal or is scarcely increased—is now very much raised, reaching 50—70 or more in the minute. This symptom alone suffices for the diagnosis of a complication by diffuse bronchitis or broncho-pneumonia, even should the local examination be without result.

During this violent course, in many cases fragments and tubes of false membrane are expelled with much difficulty by coughing and retching; and this is to be regarded as the only reliable criterion in the diagnosis of true croup. All the other symptoms—as I have already said—may be brought about by an extreme degree of simple laryngitis, and especially by "œdema glottidis." The nature of the expectorated matters is best seen by letting them float in water. When this is done, one finds small or large white fragments—often

notched at the edges—or sometimes complete tubes, which often either divide dichotomously or even branch in a dendritic manner—thus showing that they represent not only a cast of the trachea, but also of the large and medium bronchi. The expectoration of those fragments or casts takes place, however, only in about half the cases. Not uncommonly the membrane is extracted by the fingers of the anxious mother from the child's mouth, when it is almost suffocated. Immediately after the expulsion, especially of the larger tubular pieces, great relief is always noticeable. One must not, however, trust these remissions; for it is just such cases that usually end fatally. The expectoration of dendritic casts, especially, indicates that the process has spread deeply into the bronchi; and little bifurcating tubes leave no doubt of the presence of a bronchial croup affecting even the medium and smaller branches. They have, therefore, under all circumstances an unfavourable prognostic significance; for the deeper the croup extends into the air-pasages, the more certainly fatal is its course. Besides, one must remember the very rapid re-formation of the expectorated membrane, which may take place even within a few hours, and which at once brings back the orthopnœa.

Anna B., 7 years old, on 6th November, 1872, suddenly became hoarse, and had coryza, slight cough, and some fever. On the following day, slight obstructive stridor with the breathing. Emetics had no effect. On the 8th, fully developed croup, with the pharynx quite normal. Leeches and antimony prescribed. On morning of the 9th, expectoration of a cast nearly 2 inches long, ending below in 2 small branches. After this, improvement took place; the stridor much less marked, cough and voice toneless; resp. 28; pulse, 132. Inunction of unguent. hydrarg. (grs. xx. every two hours), blister over the larynx. In spite of this, enormous increase of the croup-symptoms, dating from midday; cyanosis; symptoms of asphyxia. About 6 P.M.—that is, after scarcely 10 hours—expectoration of another cast of the whole length of the trachea, followed by great alleviation of the symptoms. Night quieter. On the following day apparent improvement; resp. 24; pulse, 132. In the afternoon a fresh exacerbation; death during the night. Tracheotomy had not been performed, on account of the length and character of the casts coughed up, which indicated the presence of bronchial croup.

The state of the temperature in croup is in no way characteristic. As a rule the fever remains moderate in degree,

rising in the evening to as much as 103·1°F.; while in the morning it is about 100·4°—101·5° F. Still, there are cases (*e.g.* that given on p. 372) with much higher temperature, reaching 104° F. and over. The addition of pneumonic complications has seemed to me to be the special cause of this. The pulse is at first strong, but as the disease progresses it becomes weaker, and in the last stages is often very irregular and intermittent, especially during inspiration; and at this stage the cyanosis becomes extreme, and the face, hands and feet are covered with cold sweat. At last the child sinks into a somnolent condition owing to the obstructed respiration and the resulting carbonic-acid poisoning. The eyelids are half-closed, the respiratory movements become shallower, the obstructive stridor becomes weaker, and the child dies in a state of collapse,—sometimes with convulsive contractions of the facial or other muscles. The anæsthesia, which Bouchut pointed out, is in my opinion nothing characteristic; it is to be explained simply by the coma which comes on towards the end.

The idea that croup is absolutely incurable save by tracheotomy, is by no means correct. Occasionally, although not very often, we meet with cases in which the most threatening symptoms of croup gradually improve and are recovered from under suitable treatment without any operative procedure—even where the expulsion of fragments of false membrane had removed all doubt of the really croupous nature of the complaint. But even after the disappearance of the threatening symptoms one must not at once become elated. For, by the long interference with the respiratory processes, and the oxidation of the blood, serious disturbances of the function of the brain may be left, even after recovery; either because the blood does not quickly enough recover the qualities necessary for nourishing the brain, or because an engorgement of the cerebral veins, followed by œdema of the pia mater or serous transudation into the ventricles, has resulted.

A boy of 8 years, who had recovered from a violent attack of croup, lasting for 5 days, during which fragments of false membrane had been coughed up, and who now suffered only from complete aphonia—remained deathly pale and extremely feeble, in spite of recovered appetite. On the 14th day of the disease, the patient, who was still very weak, became drowsy, gradually comatose; and 36 hours after, died in this condition. At the

P.-M. I found the larynx healthy, with the exception of slight congestion and swelling of the mucous membrane. The brain extremely anæmic, and much serum in the ventricles and in the meshes of the pia mater.

We had in this case, not a state of diphtheritic collapse—which condition we will become acquainted with later on—but a result of primary croup. We cannot deny that the energetic anti-phlogistic measures (leeches, repeated emetics, mercurials) with which, especially in former times, we attacked this dangerous disease, along with the anorexia and the insufficient nourishment due to it,—may occasionally have contributed their share in producing such weakness and anæmia.

I have myself witnessed in a boy of three years—who had been markedly improved by a very energetic line of treatment, but was exhausted to an extreme degree—a deep sleep come on, which was welcomed joyfully by the parents. It followed immediately on the use of an emetic, which had been given on the evening of the 4th day on account of a sudden suffocative seizure. On my visit I found the child, who shortly before had been very restless and breathing noisily, now lying motionless in his cot; the breathing almost inaudible and unusually slow. On feeling his pulse, however, I perceived that this was no healthy sleep, but a state of coma. The pulse was thready, scarcely perceptible, irregular and uneven; all extremities cold, and the eyelids half-shut. Even loud noises right at the child's ears were not sufficient to bring him to consciousness; and it was only after the continued use of stimulants from 7 P.M. to 11 P.M. that this dangerous state of inanition of the brain was removed. Mustard-plasters to the neck, back, and calves, fomentation to the hands and feet with the addition of mustard, ammon. carb. (grs. 2¼ every 2 hours), and wine; finally, the application of ice to the head, which I only allowed to remain on a few seconds at a time, but repeated often—succeeded at last beyond our expectations. And when the cerebral functions returned, strange to say, all the croup symptoms, except a slight hoarseness, had disappeared for good.

For the treatment of croup the same rules hold good at the beginning as I have already laid down in the case of acute laryngeal catarrh. If local blood-letting, emetics, tartrate of antimony in divided doses, the energetic use of mercurials, and the application of a blister over the region of the larynx do not bring about rapid improvement, the symptoms continue to get worse, and the commencement of dyspnœic attacks announces an extreme degree of the disease, we can then expect nothing

further from drugs. The more one is in the habit of relying on emetics in this disease, the more unpleasant is the fact that their action not uncommonly fails. Among others, I have given to a child with measles and croup a full dose of an emetic (ipecacuanh. ʒss., antimon. tart. gr. ½, aq. destillat. ℥i., oxymel. scillæ ℥ss.) during one day, morning and evening, without even once causing vomiting. In such cases, sulphate of copper (gr.ss—gr. iss every 10 minutes) occasionally succeeds; but apart from its nauseating effect it has no specific action on croup. I must, however, most decidedly dissuade you from frequently repeating emetics in a child who is already exhausted, merely because of the continual return of attacks of suffocation. For while they are of no use, they may increase the exhaustion to an extreme degree, and (as in the case given on p. 376) result in severe cerebral symptoms. I should also recommend you not to keep children with croup continually in bed; but to let them often be carried about, for this relieves them for a time. Also you should administer beef-tea, milk, or wine very frequently, in order to combat the increasing exhaustion as much as possible. But always be cautious; because children with croup are very apt to choke while drinking, and then at once have violent attacks of suffocation.

The onset of the first threatening attack of suffocation —in fact even the forcible indrawing of the lower part of the chest wall on inspiration—is to me the signal for tracheotomy. This latter symptom—which is due to the rarefaction of the air in the lungs, and the consequent disturbance of the equilibrium between the intra- and extra-thoracic pressure—I consider of especial importance. To delay the operation longer only increases the exhaustion, the danger of carbonic-acid poisoning, and the broncho-pneumonia which is in process of development. We have therefore operated not uncommonly even on the 2nd or 3rd day of the disease, according to circumstances. I shall return to this when considering diphtheria. According to my experience, the chance of recovery after tracheotomy is much greater in simple primary than in diphtheritic croup; because in the former we have only a local fibrinous inflammation, but in the latter a general infectious disease. Out of 22 cases of inflammatory (non-diphtheritic) croup which were operated on during the last few years in my

department of the hospital, 13 recovered; a fact which of itself proves that we had not to do with diphtheria. Neither the expectoration of false membrane, nor the evidence of bronchitis or pneumonia, do I regard as a contra-indication, for I have seen several children recover from the operation in spite of these complications. Since, however, the operation only serves the purpose of allowing air to gain access into the lungs, it is always well to go on with the mercurial treatment after it, in a moderate degree, and to favour the separation of any false membrane which may still be present in the air passages, by the inhalation of steam through the canula. Other methods of treatment, such as cauterising with concentrated solution of nitrate of silver (by means of a brush or a syringe), and the introduction of a tube into the larynx (intubation[1]) I have not tried. Tracheotomy is still the treatment which gives the greatest number of successes, and therefore I do not feel inclined to exchange it for any other.

V. *Bronchitis and Catarrhal Pneumonia (Broncho-Pneumonia).*

One of the commonest diseases of childhood is catarrh, spreading from the bifurcation of the trachea to the mucous membrane of the large and medium bronchi. It is not only common in practice among the poor, where cold and damp play an important part in its causation, but is equally so under more favourable circumstances. The period of the first dentition is that most frequently affected, and this process itself is regarded by many physicians as a cause of the catarrh. That this influence is over-estimated, I have already pointed out; but I cannot deny that in many children the eruption of each new group of teeth is accompanied by an attack of catarrh. Perhaps, also, the great frequency of rickets at this age has some influence; for rickety children show a very special tendency to bronchial

[1] "Tubage" of the larynx, which was first recommended by Bouchut and recently re-introduced by O'Dwyer, has many supporters in America; still its results are by no means so gratifying as to entitle it to take precedence of tracheotomy. *Cf.* "Intubation of larynx," *Medical Record:* New York, June and July, 1887.

catarrh, and should—for reasons which I shall enter into later—be protected from it with especial care.

In very young children, even within the first few months, we often meet with a peculiar form of tracheal and bronchial catarrh. In this condition they suffer either from a frequent hacking cough (which is at once started by pressure in the situation of the bifurcation of the trachea), or still oftener from a "stertor," which almost constantly accompanies the inspiration and expiration, and which the mothers call a "stuffiness" or "rattling in the chest." The noise is sometimes so loud that it makes the parents very anxious, and it depends on the quantity of secretion whether the stertorous breathing is accompanied by moist râles or is a dry noise like that of croup. It becomes weaker after each fit of coughing, and may entirely disappear, but soon returns. On physical examination we hear only hoarse mucous râles or sonorous rhonchi, especially between the shoulder-blades; but immediately after coughing there is usually only harsh breathing heard, which after a time again gives place to râles. All this time, the little patients may feel quite well, although most of those I have seen with this disease had rather a pale and flabby appearance. There is never any fever, the appetite is good; the only thing causing anxiety to the parents is the occasional cough. As regards the cause, I have sometimes found that the catarrh had been caused, to begin with, by a chill immediately or soon after birth—either from a too cold bath, or a cold room, or from the child being taken out-of-doors in bad weather. In all the cases which I have observed this disease was characterised by great obstinacy. It was many weeks, even months, before recovery took place, and this marked tendency to a chronic course is all the more serious because every fresh chill occasions an exacerbation, which may sometimes be accompanied by fever. With few exceptions, all my cases occurred in connection with the polyclinic, and the comparative want of care on the part of mothers in poor circumstances explains the obstinacy of the catarrh. In a few cases this disease reappeared with the cutting of each new group of teeth, lasted for weeks, and disappeared as soon as the teeth came through. As regards treatment, the chief matter is to protect the children from cold and damp, while at the same time letting them have pure air to breathe—conditions

which can only be fulfilled in well-to-do families. From drugs I have seen scarcely any result; perhaps a little from small blisters over the manubrium, frequently repeated and allowed to heal at once after the bulla had formed. Those who cannot do without giving medicine may try small doses of sulphurated antimony (gr. $\frac{1}{7}$, 4 or 5 times daily).

Catarrh of the trachea and bronchi in children, up to about the 5th year, differs from that in adults only in this,—that its tendency to a rapid and dangerous extension into the smaller bronchi is far greater; and, therefore, any catarrh at this age calls for much more careful nursing. The otherwise praiseworthy endeavours of many mothers to give their children as much fresh air as possible, very often lead them into the error of sending them out-of-doors in bad weather, even when they are suffering from a cough. We cannot too strongly oppose this custom. As a rule, the children in such cases present for days nothing beyond the symptoms of a simple catarrh, till a fresh chill either brings on the laryngeal condition just described, or—more frequently—occasions a regular bronchitis. We find then, usually, that the cough suddenly becomes worse, the breath shorter, the expiration noisy, the skin hot; and generally even before making a local examination we are able to diagnose bronchitis or broncho-pneumonia.

In all the very different degrees of these diseases, and the very numerous transitions from one to the other, coughing always forms one of the most striking symptoms. In many children it seems to be painful, and they show this by crying and making faces as if in pain when they cough. The cough is generally frequent, short, and dry, and is started or aggravated by crying. Children who are able to cry for a long time without coughing certainly have not got bronchitis. In bad cases violent attacks of coughing occasionally occur, with a livid redness of the face which reminds one of pertussis. Very young children almost never expectorate, but even in the stage of resolution, when the secretion is most copious, they swallow the sputa. Further, the character of the respiration attracts the physician's attention. The number of the respirations exceeds the normal in a varying degree, according as the inflammation has passed down more or less deeply into the bronchial ramifications. In young children 40—50 respirations is but a

moderate number, and indicates that the seat of the disease is the large or medium bronchi; while the implication of the small and finest branches at once produce a rate of 60—80, or more, in the minute. If, then, a child suffering from catarrh holds its breath while being auscultated—as so often happens—and makes the physician wait, this is always a favourable sign. The quicker the breathing, the shorter and shallower does it become; the accessory muscles of inspiration (those of the alæ nasi, scaleni) are seen acting. The head also moves with each breath; and there is distinct retraction with inspiration, both at the episternal notch and at the lower part of the chest. Each expiration is also accompanied by a "grunting" sound (*cf.* p. 9), which I always regard as one of the most valuable symptoms in the diagnosis of serious respiratory diseases. Not uncommonly we can hear, even at some distance from the chest, crowing noises with the breathing, and in nearly every case, on auscultation, sibilant and sonorous rhonchi or large, medium, and fine crepitations, which may be either confined to the back—especially about the bases—or extend over the anterior and lateral regions also. The distribution of these sounds is of less importance than their character. We may, *e.g.*, hear sibilant and sonorous rhonchi almost all over the thorax, without any great amount of dyspnœa being present, owing to the large or medium bronchi only being affected; while fine or even medium crepitations, heard over a considerable area in front as well as behind, give cause for great anxiety. Occasionally the crepitations are only with inspiration or expiration; while in other cases they accompany both. The percussion note remains normal at first. Along with the local symptoms there is always more or less fever, the temperature varying between 101° F. and 103° F., and in the evening reaching even 104° F. I have not uncommonly found the morning temperature approaching the normal (100°—100·4° F.), while in the evening it rose to 104° F. Even when exact thermometric examination is impossible—as in most cases in the polyclinic—the statements of the mothers may be worth something, as they are in the habit of noticing especially the children's "burning skin." I do not attach any special importance to the rate of the pulse, which varies between 120 and 180. Its quality is of much more importance; although,

when the disease runs a favourable course, this usually presents no abnormality. The altered ratio between the frequency of the pulse and that of the respiration, is always of the greatest significance. For we have no longer 3 or 4 beats of the pulse to one respiration, as in the normal condition, but the number of the latter increases disproportionately: *e.g.* 60—70 respirations to 144 pulse beats (p. 9). The other functions of the body may remain unaffected in slight cases; still I have often observed diarrhœa as a complication, especially during an epidemic of intestinal catarrh. As the disease gets worse, the appetite also naturally suffers; infants are prevented from sucking by the dyspnœa, because after a very short time they have to let go the nipple in order to get breath. This circumstance appears to me such a characteristic sign of the severity of the bronchitis that I advise you to let the child take the breast in your presence in order to ascertain how it can suck.

From the above symptoms and physical signs—especially the latter—you may always diagnose with certainty an acute or diffuse bronchitis. Whether there is also an affection of the lung tissue itself (broncho-pneumonia) we cannot diagnose with certainty; but just as little can we exclude it. The explanation of this is to be found in the pathological condition, of which the chief features are the following.

The mucous membrane of the bronchi is to a varying extent reddened, swollen, and thickened, and sometimes also eroded here and there. This condition often extends right into the smallest bronchioles, and may either be uniform or occur in patches. Their lumen, especially in the lower lobes, is blocked with a tough, yellowish-white, mucous secretion; and when the disease has lasted long, there is a moderate dilatation even of the peripheral ramifications. Owing to the marked tendency of this affection to spread deeply, there occurs in a number of cases a more or less extensive inflammation of the finest branches (bronchitis capillaris). In these cases, when a section is made through the affected lung, muco-pus exudes from many points, which indicate the sections of the finest bronchial tubes, as out of a sponge. Under these circumstances the inflammation passes, in many situations, to the extremities of the finest bronchioles and to the pulmonary alveoli, which are sometimes

visible under the pulmonary pleura as whitish-yellow, miliary granulations, resembling tubercles, and from which on section there exudes a drop of fluid (bronchite vesiculaire of the French). There also always occurs at the same time a development of broncho-pneumonic deposits, and these at first assume a lobular form corresponding to the area of distribution of the small bronchi. The number of these deposits varies according to the extent of the bronchitis, and they are most frequently situated in the lower lobes, and appear as hard thickenings of the size of a pea, bean, or hazel-nut, and of a reddish-brown colour, or sometimes with a tinge of grey. At first they are separated from one another by air-containing hyperæmic tissue, but as they increase in number they approach and finally run together into large masses. These usually have a wedge-shape, and extend upwards from the base of both lower lobes; but they also occur often enough in the upper lobes, and especially in the tongue-shaped process of the upper lobe which overlaps the pericardium. They may also in the end affect a whole lobe, or even the greater part of one lung. From the surface of a section made through one of these patches or extensive consolidations—which, when cut out, sinks in water—there only exudes an extremely small amount of fluid when squeezed, and on microscopical examination we find that the alveoli are filled with masses composed of fatty epithelium and numerous lymphoid cells of various sizes—which also may be becoming fatty, and then give a greyish-yellow colour to the consolidated area. According to recent researches (Charcot and Cadet[1]), a fibrinous exudation is almost always discoverable in them. There is always hyperæmia of the capillaries in the neighbourhood and cell-proliferation in the interstitial connective tissue. Emphysema of the borders of the lung, or of other unaffected portions, and patches of atelectasis are usually found; also not uncommonly a more or less extensive pleurisy and enlargement of the tracheal and bronchial glands.

From these facts we may gather that catarrhal pneumonia (broncho-pneumonia), developing from bronchitis, can only be diagnosed by physical signs, if the patches described are

[1] Cadet de Gassicourt, "Traité clinique des maladies de l'enfance," i., Paris, 1880, p. 152.—The bacteria in the alveoli described by Thaon (*Revue mens., Févr.*, 1886, p. 93), as the cause of infectious broncho-pneumonia, I consider as of no importance until their pathogenetic nature is established.

so numerous or run together to such an extent that the intermediate air-containing tissue is no longer sufficient to hide the symptoms of consolidation. As long as the patches are scattered at considerable intervals through the lung tissue, you will only find the signs of bronchitis—*i.e.* more or less widespread medium or fine crepitations which, in cases of capillary bronchitis, can be heard at almost every part of the chest to which you apply your ear. As soon, however, as the consolidation has extended over a larger area of the lung, you have a corresponding extent of dulness, fine sharp râles, bronchial breathing, and bronchophony. These physical signs usually appear first on both sides of the spine, from the base of the lung to near the spine of the scapula; not unfrequently, also, in the region of the apices, and especially in the tongue-shaped process of the left upper lobe. I have repeatedly discovered fine, sharp râles over the heart in the latter sooner than over any other part of the chest. It is noteworthy that sharp râles and diffuse bronchophony may be present in those cases even when there is no distinct dulness. The percussion may indeed remain quite normal, or may acquire a tympanitic character—which can only be explained by supposing that at the periphery of the lung there is still a sufficient amount of air-containing tissue—while auscultation is able to discover the signs of consolidation which is present at a greater depth. Such being the case, I would recommend you to percuss very lightly (p. 7), since a strong stroke may, by eliciting a loud sound from the air-containing tissue, obscure any slight impairment which may be present. Now, since it has been established by numerous post-mortems that in every case of extensive bronchitis in the first years of childhood, more or less numerous patches of broncho-pneumonia are also present—we must assume that even the absence of all physical signs of consolidation does not in these cases exclude the presence of broncho-pneumonia in the form of lobular patches. And in cases where such physical signs—even only those of auscultation—are discovered, we may always diagnose extensive confluent patches of consolidation.

In many cases, however, although there is very severe dyspnœa, we can discover either very few râles or none at all. The percussion is normal, and all over the chest we hear the breath-sound extremely harsh; or the breath-sound is absent, and one

hears nothing but sibilant rhonchi. These physical signs may gradually give place to moist râles, indicating a freer secretion; or they may last till death—which usually ensues a few days later.

> The most striking example of the first form that I have seen was in a child of 11 months, whose respirations were 72 and laboured, the pulse 160 and very small, and whose chest presented, all over, a normal percussion-note and very harsh breathing; only at the right posterior base there were a few fine crepitations. This condition lasted three full days, in spite of copious diaphoresis, caused by moist compresses round the chest; and then the respirations fell to 56, and the pulse to 130. The cough became more frequent and looser, and, soon after, noisy breathing and widespread mucous râles appeared.—I met with a rapidly fatal case of this kind in a child of 11 months. It took ill with a cough, and in 2 days showed all the symptoms of an advanced acute lung-disease; and over the whole thorax unusually harsh breathing was audible, with occasional crepitations here and there. After death, I found in both lungs numerous easily-inflated collapsed patches, and the small bronchi entering these were filled with muco-pus. All the other bronchi were completely free from secretion; but their mucous membrane, from the bifurcation down to the smallest branches, was much reddened and swollen.

Thus even without muco-purulent secretion, bronchitis may seriously threaten life, simply by the rapid hyperæmic swelling of the mucous membrane, and the consequent narrowing of the lumen of the bronchi.[1]

The deeper the inflammation spreads into the finer bronchial ramifications, the more numerous the lobular patches or larger consolidated areas of broncho-pneumonia—the more, of course, will the respiratory process and the oxidation of the blood which depends upon it be interfered with. No efforts of the inspiratory muscles are sufficient to force the air into the alveoli through the small bronchi which are filled with muco-purulent secretion; hence the pathological condition found in such cases of numerous collapsed areas in the lung. The efficiency of the lungs for respiration must thereby be considerably diminished, and also the increased number of shallow respirations (I have in some cases counted more than 100 in a minute) cannot make up for the loss of depth. The breathing is also often irregular; for example, 10—15 respirations may follow one another with

[1] *Cf.* Rilliet and Barthez, *l. c.*, i., p. 454.

extreme rapidity, and then a short pause take place, reminding one of Cheyne-Stokes breathing. The venous congestion, a natural result of the consolidation of the lung, and consequent engorgement of the right side of the heart, soon gives a cyanotic tinge to the pallid face and visible mucous membranes, and causes enlargement of the peripheral veins, and sometimes also slight œdema of the eyelids and of the backs of the hands and feet. The steady lowering of the heart's energy is indicated by the smallness of the pulse, which is exceedingly rapid and disappears under the finger, as well as by the coldness of the extremities. About this time also the power of coughing fails through weakness, and I always regard it as an extremely unfavourable symptom if the hitherto harassing attacks of coughing become weaker or cease entirely, while on auscultation we can still hear sharp crepitations all over. When this state of matters is found, it is usually soon followed by the carbonic-acid poisoning which necessarily results from insufficient action of the lungs. Drowsiness, half-closed lids, and up-turned eyeballs, sometimes also partial or general convulsions, terminate this distressing condition.

I now return to the fact that during the whole course of bronchitis and broncho-pneumonia the fever presents a remittent type, which is by no means characteristic, the temperature rising in the evening, and not uncommonly reaching 104° F., but presenting many variations; thus a considerable fall of temperature on certain days alternates with sudden, apparently inexplicable rises. These variations depend on the fact that the inflammatory process is always spreading from the bronchioles to other hitherto unaffected lobules, while in other places it may already be in process of resolution, and that each of these successive extensions is accompanied by an exacerbation of the fever. In very young children, especially when they are debilitated, the fever is often a very unimportant feature, or may even be entirely absent for days at a time, although the physical signs indicate a continuance of the inflammatory process. In one child of 10 days, with congenital syphilis, I found the temperature generally sub-normal (maximum 99·1° F.). In others it even went as low in the end as 95·9° F., a proof of the fact that under these circumstances there is a very great tendency to collapse, and even considerable inflammations may run

their course without fever, or even with a sub-normal temperature (p. 17). This state of things, however, is changed towards the middle of the first year. In a child of 5 months (admitted on 5th May, 1874, with double broncho-pneumonia) the temperature repeatedly rose to 104°—104·7° F., the pulse being 216.

Although the prognosis in extensive bronchitis and broncho-pneumonia is so bad, one not uncommonly sees resolution and recovery take place under apparently most unfavourable circumstances. The first hopeful sign is diminished frequency and increased depth of the respirations. The disease is always to be regarded as one which, even when ending favourably, is wont to be of long duration; in particular, it never ends with a regular crisis. Exceptional cases occur with a very rapidly fatal course. Even in such cases we almost always find that a bronchial catarrh has lasted for some considerable time before the sudden fatal onset of the capillary bronchitis and catarrhal pneumonia. On an average, the disease lasts 2—3 weeks, frequently much longer. There is an unmistakable tendency for its course to become sub-acute or even chronic, so that many weeks, even several months, may pass before a distinct change for the better sets in. The fever then falls considerably, or may entirely disappear, except for a slight elevation of temperature at midday and in the evening; the patches of dulness disappear to a greater or less degree, and the child seems almost quite well. But the cough, the wide-spread fine crepitations (which occasionally are still sharp in character), and the respiration (which continues to be rapid) indicate the persistence of the disease. In one such case—that of a boy of 7 years—which lasted for months, the muco-purulent sputum (which he had the sense to cough up) was not unfrequently spotted or streaked with blood, to the great alarm of the parents. Here also complete recovery nevertheless ensued. Still, the result is frequently fatal when the course is chronic, although the child's condition may have varied repeatedly during weeks and months. In many cases of this kind I observed, during such a course, intervals absolutely free from fever and lasting for weeks. In these the child which had already been despaired of, rallied considerably, got a better colour, coughed less, and seemed to be advancing towards recovery. But the persistence

of a quite abnormal rate of respiration (50—70 in the minute), which was out of keeping with the apparently satisfactory general condition, was always a bad sign in those cases. We must not allow ourselves to be misled by these intervals of improvement into giving a good prognosis. We are warned to be cautious by the persistent fine sharp râles, heard especially at the back, and also by the increasing emaciation of the children. In several of these cases with a chronic course finally ending in death after 2 or 3 months I have found at the post-mortem fatty degeneration of the heart with dilatation of its right side, along with the appearances of chronic bronchitis and broncho-pneumonia; and this especially in cases where the disease was complicated with whooping-cough. The great resistance which the right ventricle had to overcome in doing its work, from the long-continuing consolidation of the lung-tissue and the frequent attacks of whooping cough, must certainly be regarded as the cause of this degeneration, which has occasionally caused death from syncope.

In cases of broncho-pneumonia which have lasted for weeks or even for months, one not uncommonly finds thickening of the interstitial connective tissue surrounding the alveoli and separating the different lobules from one another. The small bronchi passing through the consolidated lung-tissue are dilated in many places, and sometimes also small abscesses of the lung are found, arising from the alveoli (which are over-distended with young cells and epithelium) having given way and coalesced to form large cavities filled with puriform fluid. This appearance (which is rare, on the whole) cannot be diagnosed during life, owing to the small size of the abscesses. Besides, in such cases there may be absolutely no fever. Thus in a boy who was admitted into the hospital on March 23rd, suffering from broncho-pneumonia of uncertain duration, only twice before death—which took place on 1st April—did I find the temperature at 100·4°—102° F. At other times it was always normal or even subnormal. At the post-mortem we found broncho-pneumonia of both lower lobes, especially extensive in the right one, which was almost entirely solid and empty of air. In both lower lobes there were several abscesses the size of a hazel-nut, filled with yellow pus. I believe that this condition is very apt to be caused by foreign bodies getting into the bronchi. At least

I found this in two cases in which, after all the symptoms of a chronic pneumonia had lasted for several months and death seemed inevitable, foreign bodies (a glass bead and a swollen bean) were suddenly expectorated with symptoms of great dyspnœa—in one case after hæmoptysis. In the first of these cases recovery rapidly followed. Under unfavourable circumstances chronic broncho-pneumonia not uncommonly ends in caseation and breaking down of the infiltrated material, but I shall return to this when speaking of chronic broncho-pneumonia.

Anything which can bring about a state of irritation of the respiratory mucous membrane, may also play an important part in the causation of bronchitis and broncho-pneumonia. First among these causes is the irritation of cold (a keen east or north wind, for example) which at times causes an epidemic prevalence of this disease along with colds in the head, laryngeal catarrhs, croup and sore throat. Next come several infectious diseases, of which this affection is often an after-result; especially measles and whooping cough, and next to them diphtheria particularly when it spreads into the larynx and trachea. Whether tracheotomy is performed or no, broncho-pneumonia always forms one of the worst complications in this disease, and it must always be had in mind if the rate of the respirations, which has hitherto been normal, suddenly rises to 50 or 60 in the minute. I believe that in such cases there is not only a simple spreading of the inflammation downwards from the trachea, but that the inspiration of diphtheritic matter from the upper air-passages plays an important part. In measles, broncho-pneumonia may begin even in the stage of eruption. More frequently, however, it developes after the disappearance of the rash and the fall of the temperature; it is then more severe, and forms always a very serious complication. The same is true of whooping cough, which it may complicate at any period of its course. Less frequently the disease comes on after scarlet fever or small-pox; while in typhoid—which is almost always accompanied by bronchial catarrh—the complication with broncho-pneumonia is found oftener. It is just those cases occurring along with the above-mentioned infectious diseases, that commonly have an unusually protracted course, and (by their accompanying weakness and wasting, as well as by the persistent remittent temperature) excite suspicion of a tuber-

cular or caseous condition of the lungs. Week after week, the rapid respiration, the harassing cough, and the sharp catarrhal crepitations defy all treatment; while dulness on percussion may either be quite absent or may disappear from the originally affected parts of the thorax and re-appear at other parts of the thorax hitherto normal. These changes, like the variations of the temperature (p. 387), are to be explained by the clearing up of former infiltrations and the implication of other hitherto normal areas. Thus the diagnosis, and with it the prognosis, varies with the daily change of the condition; till finally, after lasting many weeks or even months, either the fever ceases quite unexpectedly and all symptoms clear up, or death takes place at last with symptoms of phthisis, owing to the caseation and destruction of the infiltrated material.

Alice N., 12 years old, took ill during the first days of December, with severe typhoid. A bad cough and rapid breathing from the beginning. On the 24th day, threatening symptoms of collapse, with profuse perspiration (coldness of the extremities, disappearance of the pulse). After these symptoms had been removed by the use of stimulants for several hours, the typhoid condition seemed relieved, but the cough continued. On the right side behind, from the apex to below the spine of the scapula, dull percussion-note, bronchial breathing, bronchophony, and fine sharp râles. On the left side behind, mucous râles. Rise of temperature in the evening continued, pulse 120—132, hectic flush on the cheeks, emaciation. Under the use of simple expectorants (ammon. chlorid., antim. sulph.), afterwards of codliver oil and a strengthening diet, the threatening symptoms gradually disappeared. Percussion almost normal for first time on 19th January, 1874. In the middle of February complete recovery, which was permanent.

Pauline S., 6 years old, suffering from moderately severe typhoid fever, with broncho-pneumonia of the right lower lobe. In the 5th week of the disease when convalescence had already set in she became feverish again (evening temp. 103·1° F.) and developed diffuse catarrh on both lungs, and dulness with sharp râles for a second time over the area originally affected. There was also extreme emaciation, very sickly appearance, anorexia and brown tongue. The condition lasted three weeks. Then gradual resolution under the use of quinine, and finally complete recovery.

I have already published[1] three other cases in which the broncho-pneumonia had come on after measles and had lasted for months in a state resembling advancing phthisis, but at last

[1] *Beiträge zur Kinderheilk., N.F.*, S. 142.

was completely recovered from, so that when the children were brought to me again a long time after, they looked so thriving that I scarcely recognised them. In all these cases tonic measures (strengthening diet, wine and codliver oil) were of marked benefit.

Besides the infectious diseases I have named, other severe exhausting conditions must be pointed to as favouring the occurrence of broncho-pneumonia. Chronic intestinal catarrh, tuberculosis, basilar-meningitis, and gangrenous conditions—especially noma—are the most important. In my department in the hospital almost every child that dies, shows at the post-mortem more or less extensive broncho-pneumonia; wasted and weak rickety children especially have a tendency to this disease, and I often could not help thinking that some infection inspired along with the hospital air might have something to do with it. The course and termination of the disease has generally been more chronic and incurable in hospital than in private practice, or even in that of the polyclinic. The gradually progressive spreading of the process over large areas of the lung, the alternate improvements and exacerbations, the continually recurring relapses in spite of the best nursing—are all calculated (as the experience of other hospital physicians bears out) to indicate that the atmosphere of the wards may have an unfavourable influence. At the same time, one must not overlook the fact that the miserable state of nourishment in which most infants are brought to my department has a good deal to do with the failure of the treatment; because the weakness of their inspiratory muscles favours the occurrence of extensive atelectasis, and thereby considerably increases the insufficiency of the affected lung. We must also take into account the fact that the children are lying continually on their backs, as is necessary under such circumstances; and that this favours hypostatic congestion in the lower and posterior parts of the lung. Further, any tubercular tendency, or rickety malformation of the thorax diminishing its capacity is of especially bad prognostic significance. Bronchitis and broncho-pneumonia, and even apparently trifling colds, which would have had a favourable course in healthy children may under these circumstances end fatally.

In the matter of causes, we have finally to consider an irritation which directly affects the bronchi and alveoli; namely, the entrance of milk or other fluid into the respiratory organs. This

form of pneumonia not uncommonly occurs owing to the fluids being drawn into the air-passages from the bottle, and by their "going down the wrong way" in cerebral cases which are accompanied by coma; but it is specially apt to occur after tracheotomy. The fact of its occurrence in this way has been verified by experiments on animals (division of the vagus or recurrent laryngeal by Traube, Friedländer and others). One must not, however, be too hasty in assuming the presence of this cause; nothing but the actual discovery of food or other foreign bodies in the air-passages establishes it beyond doubt.

In many children there is a very marked predisposition to acute bronchial catarrh, so that they get it after every slight chill or cold in the head. We have therefore here a condition similar to that in false croup (p. 356). Such children have at least one, often several, attacks yearly, which as Rilliet and Barthez[1] have already remarked "by their short duration, their repeated occurrence and the severity of the dyspnœa, together with the slightness of the fever have some resemblance to asthmatic attacks in adults." I have myself repeatedly met with such cases even in very young children, and much oftener still in the second period of childhood; and in these cases I have usually found that the children had already suffered for years from attacks of this affection, which may be called "recurrent bronchitis." The causes of this predisposition are as little known to us as those which so often occasion false croup. I have sometimes found a persistent chronic bronchial catarrh from which the acute attacks arose. More commonly, when the children were examined during the intervals a perfectly normal and vesicular breath-sound was heard all over.

Boy of 2 years. Between the 8th month and the end of the 2nd year he had 6 violent attacks which began with coryza and reached their worst within 24 hours. Resp., 70 in the minute, stertorous, all the accessory muscles in action, loud mucous râles over the whole thorax, percussion normal, deathly pallor, suffocative attacks in the night. Fever and cough very moderate. Attacks occasionally commence with false croup. Emetics always acted exceedingly well. The attack passed into an ordinary catarrh, which lasted 1—2 weeks.

[1] *L. c.*, p. 451.

Child of 8 months. The attack began with coryza and coughing. Next morning the symptoms got rapidly worse; in the evening extreme pallor and orthopnœa; resp., 60—70 with harsh stridor. Cough slight; temp., scarcely raised; pulse, small, intermittent, extremely rapid. All over the thorax harsh breathing, no râles, percussion normal. Emetic, warm moist compress round the thorax, blister. Recovery within 4 days. Almost every 4 weeks a similar attack, but not always so violent. During the 4th compress, broncho-pneumonia developed with threatening cerebral symptoms, but was finally recovered from.

Boy of 4 years, brought on 8th April, 1878. Attacks of bronchitis from the 6th month, recurring every few months with severe dyspnœa and fever. Duration 3—4 days. Resp., in the attack observed, 80 and very superficial. Percussion normal, harsh breathing and sibilant rhonchus all over. Cured by tartar emetic.

Girl of 6 years. For the last 2 years bronchitic attacks almost every month, lasting 3 or 4 days. During the intervals simple chronic catarrh of the larger bronchi. Lungs normal.

Girl of 5 years. Healthy in other respects. Ever since the end of the first year bronchitic attacks, which during the last year had returned every 5 or 6 weeks and continued 8 days. Attacks began with fever; extreme dyspnœa, R. 56, P. 144. At the same time remarkably placid look and great cheerfulness. Cough violent; percussion normal, harsh sawing breathing all over. Expectorant mixture and wet compresses.

I have frequently observed as in the first case, an attack commencing as false croup and quickly passing into bronchitis. The croupy breathing in these cases soon becomes more whistling or accompanied with moist sounds, and on auscultation there is found harsh indeterminate breathing either alone or else with sibilant and mucous rhonchi. The dyspnœa is extreme, the rate of breathing 60—80, the pulse running, the colour pale or cyanotic, and the whole appearance so threatening that—especially to the inexperienced—the child seems to be lost. Although the fever is generally moderate, it may in many cases reach a high degree. Real alarm, however, is only justified when the physical examination proves with certainty the presence of extensive patches of broncho-pneumonia. This condition I have never found in such cases; and I am confirmed in my opinion that there is here (as in false croup) a rapid swelling of the mucous membrane reaching far into the medium-sized bronchi and diminishing their calibre,—by having observed that in spite

of the most threatening symptoms the attack usually ends very quickly within a few days, and passes into a simple loose catarrh.

Among other cases favouring this view, was that of a boy of 1 year and 3 months, in whom an attack of this kind rapidly developed. On the following day a slight attack of false croup which lasted 1½ days with threatening symptoms, then rapidly passed off leaving a slight catarrh. In a fortnight the child got coryza again and at once the stertorous breathing, the rapid respiration and wheezing in the chest also began again, and after 2 days disappeared just as quickly.

Still, I consider it possible that a spastic contraction of the bronchial muscles as in bronchial asthma may have something to do with this condition. I have repeatedly seen children who were never quite free from bronchial catarrh but always had sibilant rhonchi here and there, especially audible over the back. From time to time, especially following a cold in the head, there arose very suddenly a violent asthmatic attack with slight cyanosis of the face, without the larynx, however, being affected—*i.e.* without hoarseness or croupy inspiration. Unfortunately there was no sputum at all. All over the chest we heard sibilant rhonchi and very weak breathing. This alarming (but non-febrile) attack lasted occasionally scarcely half an hour or an hour and then disappeared as by magic, being replaced by the previously existing catarrh. The short duration as well as the sudden onset and equally rapid disappearance of the attack is in favour of a reflex spasm of the bronchi being present, which many specialists have recently described as depending on states of irritation of the nasal mucous membrane.

Treatment. Simple catarrh gets well spontaneously, as in later life, if the child is taken care of and kept in-doors. Still, it is nearly always 2 or 3 weeks before it quite disappears, especially when it has begun with fever at first. Among medicines, ipecacuanha (Form. 16) is especially recommended by many; and when the cough is violent it may be combined with cherry-laurel water (♏ xv—♏ xxx). I hardly think this remedy shortens the course of the catarrh, but I do not deny its soothing influence on the cough. It is most suitable when diarrhœa is present at the same time. When there is constipation and fever I prefer to give the ipecacuanha in combination with

calomel (Form. 17). I have found this successful in many cases of febrile catarrh and slight broncho-pneumonia.

Should the disease, however, begin more severely with great dyspnœa and high fever, more energetic treatment seems to be demanded. The application of leeches to the thorax and to the epiphyses of the bones of the forearm—which was once the fashion—has been almost entirely abandoned in our time, because the loss of blood is considered too weakening and dangerous. This view is certainly right in regard to the great majority of cases—especially in the sickly children one meets with in hospital and among the poor. It is another matter, however, when one has to do with children who were previously healthy and plethoric. Former experiences[1] have shown me that local blood-letting in moderation has by no means the bad results (anæmia, collapse) which the modern timorous school of practice imputes to it: and I cannot maintain that my results in broncho-pneumonia have become more successful since I banished blood-letting entirely from my practice. During the last few years I have again cautiously attempted an antiphlogistic line of treatment, and repeatedly with suprising success. This was done, of course, only in the case of children who were vigorous and formerly healthy, and at the commencement of the disease, whether it arose from an ordinary catarrh or came on during the eruptive stage of measles. I now however use, instead of leeches, wet, or preferably, dry cupping (4—8 cups, according to the age); because the latter withdraws blood equally well and no after-bleeding is to be feared from it. Since blood-letting is only to be undertaken in strong children, the sub-cutaneous fat is always sufficient for the application of the cupping-glasses. I repeat, nevertheless, that these methods must only be used with caution. The great majority of these patients are sickly, rickety and debilitated by other diseases, and in them any blood-letting would be pernicious and only dry cups can be used.

I should much rather recommend you to have wet compresses applied to the chest from the beginning, reaching from the neck to about the umbilicus. A napkin or towel is to be dipped in water at the temperature of the room, rung out and gently applied round the thorax without compressing it at all, and so as to leave the arms free. Over this a sheet of wadding is placed,

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

and the whole is covered with oil-silk, or gutta-percha tissue. When the temperature is high, I have these compresses changed at least every half-hour, but afterwards let them remain 1--3 hours, and carry this on continuously for several days and nights. I have sometimes even continued this treatment for a whole week, and in these cases, generally, the water which was used cold at first, was afterwards used at a temperature of 100—103° F. The compresses appear to have a favourable action in three ways: Firstly, by the deep inspiration which takes place immediately on the application of the cold, driving the air forcibly into the alveoli and possibly preventing atelectasis; secondly, by the counter-irritation of the skin which finally manifests itself in redness, papules and desquamation of the epidermis; thirdly, by the process of evaporation keeping the atmosphere round the child moist, and this may be aided by having steam coming from a tea-kettle or spray-apparatus close to the bed. The compresses also sometimes cause a favourable perspiration, but this must not be excessive. In one child of 11 months, I saw, as the result of such profuse sweating having lasted too long, an onset of threatening symptoms of collapse (extreme pallor, disappearance of the pulse, slight cyanosis); and these rapidly disappeared when the compresses were removed, and the sweating ceased under the use of wine. During the whole course of the disease it is moreover advisable not to allow a child to lie continually on its back, but to have it carried about in the arms from time to time, in order if possible to avoid hypostatic congestion.

As to medicines, emetics have always been held in highest estimation; and I must subscribe to this opinion as far as it applies to otherwise healthy children. In these the disease is always best treated by an emetic, and where careful nursing and observation is possible, I recommend tartar emetic in divided doses (Form. 18) as really the best, in spite of all that has been said against it. I give a dessert-spoonful of the mixture every hour until vomiting commences; and then every two hours. Should vomiting or even diarrhœa set in after each dose, the medicine must at once be stopped. Also, if no vomiting should follow after the first three spoonfuls, I lengthen the intervals to 2 hours in order to avoid a cumulative action, which when it has once set in is difficult to control. This line of treatment, how-

ever, is quite unsuitable in delicate children when there is diarrhœa, and in an advanced state of the disease; especially in practice among the poor, and in the polyclinic where the mothers, being left to themselves, might readily by giving this medicine carelessly or for too long, occasion exhausting diarrhœa and collapse. When, under these circumstances, the chief matter is to empty the bronchi which are choked with mucus, and to make the breathing freer, it is better to give a full emetic of ipecacuanha (Form. 6), and to avoid antimony entirely. In strong infants I have often at the commencement of the disease given an emetic of vinum antimoniale and oxymel scillæ (Form. 19) with good results.[1] But we must of course avoid the use of all emetics if the symptoms of carbonic acid poisoning and prostration are already present. At that stage the medicines not only fail to act, but may most seriously increase the weakness by exciting diarrhœa, and depressing the heart's action. Both of the principal modes of action of the emetics—the expulsion of mucus from the bronchi and the production of sufficiently full inspirations—are then entirely prevented by its debilitating action.

As soon as numerous râles indicate a copious secretion in the bronchi, and the sinking of the strength forbids a full dose of an emetic, you should give ipecacuanha, infusion of senega, or polygala amara, and to increase the coughing (and thereby the expiration) you may add aromatic spirit of ammonia. Mustard plasters over the sternum or the back, and small fly-blisters on the thorax are to be recommended at the same time. Milk, beef-tea, wine (sherry, tokay and port) must be given alternately, in order to sustain the strength as much as possible. Should these remedies have no effect and the strength continue to sink, a combination of camphor and benzoic acid (Form. 21) may be tried, and is often successful. Under these circumstances, also, warm baths (95—97·2° F.) with cold affusion repeated several times a day, have a surprisingly good effect, and should therefore never be neglected.

Finally, a few words more on the treatment of recurrent bronchitis (p. 393). During the attack, this is no ways different from that just discussed, and it is just in those

[1] My experience of Apomorphia, which is recommended by some, is not sufficient to enable me to give a decided judgment. The experiments which I instituted, and which did not end satisfactorily, were indeed confined to severe cases of broncho-pneumonia. I have now quite given up the drug.

cases that the action of emetics is usually most striking. In order, however, to prevent the frequent recurrence of the attacks, the best thing, according to my experience, is the use of brine-baths in some watering-place such as Reichenhall or Soden. Of course this treatment must be repeated two or three times, and a visit to the sea-side—epecially on the North Sea (Norderney, Ostend, Blankenberghe, Scheveningen, Heligoland), is to be recommended as after-treatment. I do not think it right to order sea-air from the very beginning, because owing to the irritability of the mucous membrane it not uncommonly occasions a fresh attack. Instead of the sea-air, we may also recommend a visit to one of the lower Alpine resorts (Kreuth, Aussee, Engelberg, Beatenberg, Heiden, &c.). From the use of compressed air, which is recommended by many,[1] I have observed no good results in the few cases in which I have tried it. Still, my experience in this matter is not sufficient to warrant a decided judgment.

VI. *Croupous Pneumonia.*

Although catarrhal or broncho-pneumonia is the commonest inflammatory affection of the lungs in childhood, yet the view which formerly obtained as to the rareness of the croupous form has long been done away with, and rightly too. Between the third and the twelfth year this disease is indeed very common, and also in the first two years of life it is by no means rare. The following description is founded on 124 of my own cases; in these the age could be determined exactly in 88 only. Of these there were:

19	between	1½	and	3	years.
32	„	3	„	6	„
37	„	6	„	12	„

Out of 74 cases there were 18 in the months between May and August 18th, inclusive; from October to April, inclusive, 56 cases.

But in its clinical and pathological aspects the disease corresponds so entirely with the pneumonia of adults, that I only need here to enter more closely into a few peculiarities caused by the patients' youth. You are aware that in croupous pneumonia

[1] v. Laszewski, *Zur pneumatischen Therapie des Kindesalters, Dissertation* Halle, 1886.

the alveoli of the lung are filled with a coherent exudation consisting for the most part of coagulated fibrin, while in catarrhal pneumonia the contents of the air-cells consist of a mixture of young cells and of epithelium, which is mostly fatty. You further know that the latter disease always occurs at first in lobular-patches, corresponding to the ramification of the inflamed bronchus, and only becomes diffuse gradually by the continual addition of new patches, while the croupous form affects, so to speak, all at once from the very beginning, a large part of the lung—even a whole lobe, filling it with solid exudation. The clinical symptoms also vary with the different pathological conditions. Instead of being ushered in by bronchial catarrh, and gradually increasing in severity and extent, like broncho-pneumonia, we find in the croupous form a rapid, almost sudden development, with violent fever, after the manner in which many acute infectious diseases are wont to set in. Also the double-sidedness of the former, depending on the diffuse bronchitis, separates it from croupous pneumonia, which generally occurs only on one side. As to the localisation of the latter in the upper or lower lobes, in 124 of my cases it affected:

In	3	the whole right lung,
„	3	both lower lobes,
„	5	the left upper lobe,
„	26	„ right upper lobe,
„	47	„ left lower lobe,
„	40	„ right lower lobe.
	124	

and from this we see the preference of the disease for the lower lobes in childhood also.

The above-mentioned differences can only claim, however, to hold good as a general rule. Even pathologically we meet with mixed forms. Steffen[1] maintains, in opposition to Bartels and Ziemssen, the possibility of the product of a lobular pneumonia being sometimes of a croupous nature. Steiner and also Damaschino[2] described patches of a croupous nature which were found along with broncho-pneumonic patches in one and the same lung; and Virchow formerly stated that besides

[1] *Klinik. der Kinderkrankheiten*, i., S. 146.

[2] "Des différentes formes de la pneumonie aigue chez les enfants". Paris, 1867, p. 29.

the cell-proliferation, fibrinous exudation may also occur in the alveoli owing to an extreme degree of irritation. I have myself also met with analogous cases; among which I may especially allude to that of a boy with pleuro-pneumonia of the whole left lobe, who had at the same time bronchitis and in the right lung a small patch of broncho-pneumonia (*cf.* p. 384). The clinical symptoms, however, are not always so typical as one might fancy from the descriptions in the books. Especially in the hospital and polyclinic, where the children are not brought for treatment till the disease is fully developed so that its early development remains unobserved, we may be in doubt as to what form of pneumonia we have really to do with. Suppose, for example, a case in which on physical examination we discover an extensive pneumonic consolidation of the right lower lobe and a catarrh of the left lung at the same time. In such a case you must bear in mind that the patches in broncho-pneumonia may not uncommonly run together into a large mass causing actual symptoms of consolidation in one lung only; while in the other they may remain isolated, so that we can only make out catarrhal sounds. On the other hand, the accompanying catarrh is not altogether characteristic of broncho-pneumonia; for, especially in children, I have not unfrequently had occasion to observe croupous pneumonia complicated by bronchial catarrh.

In these doubtful cases, the character of the fever is always a very valuable symptom. I have no hesitation in subscribing to the conclusions which Ziemssen[1] drew from his investigations as to the regular course of the fever in croupous pneumonia and its relation to the critical days; and I also agree with him that the characteristics of the catarrhal form are "its protracted course, with marked variations in the temperature during the later stages; the continually-recurring exacerbations of the fever, each of them corresponding to an advance of the local process; the slow fall of the temperature, delayed by small rises; and the protracted resolution of the regular consolidation." All this is certainly true in the majority of cases; but by no means in all. Not every croupous pneumonia ends with a crisis; for we may also have a more "spun-out" course, approaching to a chronic condition; and on the other hand I have occasionally observed cases of

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

pneumonia which had the entire appearance of being catarrhal but nevertheless had an unexpectedly rapid and favourable course, so that recovery took place within 5 or 8 days. Those who are interested in this matter may compare cases of this kind which I have elsewhere published.[1] Repeated observations since then have only confirmed me in the view I then took. Between well-marked cases of croupous lobar pneumonia on the one hand and broncho-pneumonia on the other, there lies an intermediate form which cannot be diagnosed with certainty clinically;[2] and the question whether it is possible to differentiate the two forms of pneumonia from one another during life in every single case must therefore in my opinion be answered in the negative. Also, the circumstances under which a case of pneumonia developes do not determine the matter; for both primary—*i.e.* idiopathic—pneumonia and secondary—which comes on in the course of some other acute or chronic disease—may present a croupous character. Thus I have sometimes found croupous pneumonia in children with tuberculosis of the lungs and caseation of the internal glands or other organs; also in acute infectious diseases, especially measles, although broncho-pneumonia is very much more frequent in that disease. The most astonishing case, however, that I have had was that of a girl of 12 years suffering from severe typhoid, whose temperature could not be brought down by any antipyretic measures, but kept continuously at 104° F. and higher. After death we found the whole left lung hepatised, almost from top to bottom; and in the middle of it, at the lower border of the upper lobe, two isolated patches of the size of a bean and a hazel-nut respectively, separated from the neighbouring tissue and surrounded by a line of demarcation (dissecting pneumonia).

I have already remarked that croupous pneumonia may also develope occasionally from a catarrh, either acute or chronic; in which case catarrhal sounds are heard during the whole course in the affected lung or in both. In the great majority of cases, however, pneumonia begins quite suddenly, as in adults. I have even observed occasionally, in children over 5 years, the rigor which ushers in violent fever, but more frequently repeated vomiting. This commencement, together with the

[1] *Beitr. zur Kinderheilk., N.F.*, S. 161.

[2] *Cf.* also Steiner, *Prager Vierteljahrschr.*, 1862, ii., S. 12.

rapid rise of the temperature to 104° F. and over (in one case I observed it 106·1° F. on the first evening), is all the more likely to lead to error because the respiratory symptoms at this stage may as yet be completely latent, and instead of them symptoms often appear which seem to point to the brain being affected—especially drowsiness, delirium, a dark flush on the face, and glistening eyes. Slight pains in the neck also, with congestion of the pharynx and of the gums are often present at first, and a slight redness of the skin usually occurring in certain places only (Rilliet and Barthez had previously noticed the same), make it all the more perplexing to the physician. We first think scarlet fever is about to appear, or gastric fever, or that meningitis is in course of development. Under these circumstances you should take particular notice of the character of the breathing. Even at this stage a careful observer is struck by the short breathing, which is very rapid in proportion to the pulse-rate, and by the "grunting" expiration; although there is not as yet any cough or real dyspnœa. The cough especially may be quite absent at first, and even in the later stages it may be very slight—probably owing to the bronchi not being affected. The examination of the thorax either yields no abnormality at all, or at most, if one auscultates very attentively, a weakening of the vesicular breathing at the affected parts or occasional crepitations on deep inspiration—for example, over the right lower base; while the percussion-note over the apex in front is somewhat tympanitic.

Emil A., 5 years old, brought to the polyclinic 10th June; a very strong child. Four days previously sudden onset of high fever, complaints of pains in all the limbs, apathy, loss of appetite, thickly-coated tongue. Pulse, 132; resp. 144, short. On examination nothing found, but rather weak breathing over the right base and the percussion-note higher and somewhat tympanitic over the right apex in front. The suspicion which I expressed to my class that pneumonia was in process of development, was confirmed within the next few days. On 12th, fever less; severe cough; over the right apex in front the percussion as on 10th; but behind—from the spine of the scapula downwards and in the axillary region—extreme dulness and bronchial breathing. On 16th, the fever having fallen critically, all symptoms were rapidly disappearing.

This latency of the physical signs, which may last 4—6 days,

may, along with the prominence of cerebral and gastric symptoms, readily lead to a mistaken diagnosis of meningitis or of typhoid, or even of an intermittent fever as I experienced in one case. Perhaps in such cases the pneumonia gradually spreads from the centre of the lung to the periphery; and only when it has reached this situation, do the signs of consolidation appear distinctly. Whenever this takes place, the gastric or cerebral symptoms which have hitherto been prominent usually now become less so, and the diagnosis at once becomes clear; but in some cases not until the fever is distinctly on the decline, or may even have already ended critically. I do not share the opinion of many writers that pneumonia of the upper lobes is especially apt to lead to these mistakes, since I have also frequently seen inflammation of the lower lobes follow such a course. The brain symptoms, which usher in such cases (pneumonie cérébrale), according to my experience, most frequently take a typhoid form, as apathy, drowsiness, giddiness, delirium, dry tongue, and, much less commonly, epileptic convulsions.

Pauline S., 4 years old, brought on 7th July. For the last few days continual fever and cough. On the morning of the 6th, repeated convulsions; pulse, 152; resp. 64; percussion normal all over; a few crepitations on the right side in front; violent pain in the head. On the 8th, for the first time (*i.e.* on the 4th day of the disease), dulness at the upper part of the right side behind, with distant breathing; and later, bronchial breathing. Crisis on the 7th day.

Auguste H., 4 years old, admitted 11th May; unwell for last two days, with headache and loss of appetite. On the afternoon of the previous day, sudden general convulsions, so violent that the child had been thrown out of bed. Delirium during the night; great apathy; half-closed eyes, with somewhat dilated pupils. Temp. 104·2° F.; pulse, 152; resp. 42, very superficial and somewhat difficult. Cough scarcely noticeable. A large patch of dulness over the right posterior base, with fine sharp râles, was discovered for first time on 13th, while the brain was becoming clearer. On the 15th, a complete crisis. On the 18th, dulness already considerably diminished, medium crepitations and sonorous rhonchi; loose cough. On 23rd, everything normal.

Helene S., 6 years old, admitted 4th February, on account of fluor albus. On 23rd, sore throat suddenly commenced; temp. 102·2° F.; ev., 106·1° F. Pulse, 150. At the same time the child became comatose, with violent twitchings of the muscles of

the eyes, face, and extremities, lasting 20 minutes. On morning of 24th, temp. 105·6° F.; sore throat still present; resp. 60, rapid and superficial; on the left side catarrh, on the right dulness, indeterminate breathing and sharp râles; later on, bronchial breathing in supraspinous fossa. Persistent high temperature, the brain, however, remaining quite clear; no return of the convulsions. Crisis between 6th and 7th day.

Otto S., 7 years old, suddenly took ill with high fever and vomiting in the night of 16th January. On 17th persistent drowsiness and apathy, from which, however, the child can be easily roused, and then answers correctly. Temp. always 104° F. and over; at noon reached as much as 106·7° F. Quinine, gr. ½, and two baths of 84° F. had no effect. On 19th—when I first saw the patient—persistent fever (107·2° F.), apathy, flushing of the face, injection of the conjunctiva, scabs on the lips, dry tongue. Resp. 40, not difficult, but slightly noisy; some cough. On the left side behind, especially from the spine downwards, and laterally, dulness, bronchial breathing, and bronchophony. On the 20th—*i.e.* between the 3rd and 4th day—fall of the temp. to 101·3° F., with general improvement. On 21st, it rose again to 104° F., with severe dyspnœa; resp. 60. Crisis on 7th day.

Opinions differ as to the causes of these initial cerebral symptoms. I believe that the more typhoid symptoms (vomiting, giddiness, headache, apathy, drowsiness, delirium, involuntary action of the bowels, and dry brown tongue) are due to the rapid rise and continued elevation of the temperature; and that it is possible, although not proved, that convulsions may also arise in this way in children who are predisposed to them. Likewise so long as we have no experimental proofs of the action of the recently discovered "pneumococci" and their "ptomaines," we cannot make them answerable for these symptoms. I have never been able to find any connection with a complicating otitis, such as Steiner has drawn attention to; and meningitis can only be assumed when the cerebral symptoms not only come on at the beginning, but also persist during the further course of the disease until death. It is well known that cerebro-spinal meningitis may occur along with pneumonia. The symptoms of the former, however, are in those cases the most prominent, and the pneumonia appears as a chance complication. It always seems to me that many of the cases of "recovery from meningitis," especially from "tubercular meningitis," have been nothing but cases of pneumonia with cerebral symptoms, which were wrongly diagnosed.

In connection with the gradual development of pneumonia from the centre of the lung to its periphery, which causes the latency of the physical signs for several days, we may consider the form described under the name "pneumonia migrans," which I have repeatedly observed in children. The fibrinous consolidation in these cases spreads onwards by small advances, like the rash in erysipelas, from the portion of lung originally affected; and may in this way gradually affect the entire lobe.

Anna S., 7 years old, admitted on 8th February with pneumonia of the left lower lobe and very high temperature (104·9°—105·6° F.). Next day dulness and sharp râles were observed over the left lateral region, and continued unchanged; the temp. remained high. Resp. 76—84; pulse, 144—150, and a marked gastric complication present (thickly coated tongue, vomiting, fœtor oris, diarrhœa). On 13th (the 10th day of the disease), the symptoms of consolidation behind already clearing up (temp. 101·8°—102·9° F.), while in front the dulness, bronchial breathing, and sharp râles extended up to the clavicle. Crisis on the 11th day. On 19th February complete recovery.

Elise B., 5 years old, admitted on 29th December. Took ill some days before, with fever, vomiting, drowsiness; no cough. Resp. 36; pulse, 144; temp. m. 103·1° F.; ev. 104·7° F. Looks like typhus. On 3rd January I discovered pneumonia in the left lower lobe. Resp. 52; dyspnœa. Bath of 90·5° F.; quin. sulph., grs. xv. On the 4th, spreading of the pneumonia upwards and laterally. On 5th, consolidation of left upper lobe, also observable in front. Temp. always 103·1°—104° F. On 7th, disappearance of fever; temp. 99·1° F. Gradual resolution. On 12th, dulness and indeterminate breathing still to be made out; everything otherwise normal. On 20th, discharged cured.

In these, and in several similar cases, we could by physical examination follow the separate advances of the wandering pneumonia, from the base of the lower lobe round the side to the apex in front. This process took 4—6 days. In the first case the temperature, which was already falling, rose suddenly on the evening of the 12th to 104·2° F., evidently owing to the last advance of the pneumonia to the apex of the lung, and the disease then terminated. In a boy of six years I have seen pneumonia—hitherto limited to the posterior portion of the right lobe, but spreading upwards—pass beyond the axillary line for the first time on the 10th day, and affect the anterior part of the lung; while the signs of consolidation near

the vertebral column again disappeared. The crisis did not occur till the 13th day. In such cases one may, therefore—especially when resolution is delayed at the lower part—readily be misled into assuming the presence of considerable pleural exudation, till the clearing up of the percussion-note over the lower part of the back, or else the appearance of the crisis, establishes the diagnosis of pneumonia migrans.

I must take this opportunity to remind you that in pneumonia of the upper lobe dulness may appear at the base, not due to a sudden spreading of the pneumonia, but to a pleural exudation which has come from the apex (Traube). The complication with pleurisy occurs in children just as frequently as in adults, and in older children it is revealed by complaints of pain on coughing, by their lying on the affected side, and by percussion and palpation of the intercostal spaces. As a rule, the accompanying pleurisy and the exudation which it causes do not reach any considerable amount, although the dulness and weak breathing at the lower part of the back, due to the latter, continues far into the period of convalescence. I have also seen (but in far fewer cases) purulent pleurisy gradually develope from pneumonia, which—on account of the persistent hectic fever—required puncture, or finally the radical operation for empyema; after which recovery took place. In the case of a girl of 11 years the pleuro-pneumonia was primary, and in a boy of 9 it came on in the course of scarlatinal nephritis. The differential diagnosis between pneumonia and pleuritic exudation is especially difficult in children who are too young to speak, because two important symptoms—the vocal fremitus and the rust-coloured sputum—are wanting in them. The former can almost never be obtained distinctly, and for it one has to avail oneself of the moments when the child is crying loudly. I have never been able before the third year of life to make out the increase or diminution of the vocal fremitus with sufficient distinctness to warrant conclusions from it as to the diagnosis. Rust-coloured sputum I have observed almost only in older children, of 8—12 years. Only on one occasion have I seen the expectoration streaked with blood in a boy of 4½ years.

The course and termination of this form of pneumonia in children resemble those of the disease in adults just as much as do the symptoms. The great majority of cases end favourably

with a regular crisis (72 times in 82 cases). Fewer end gradually and with symptoms of lysis (10 times). The crisis took place in most of the cases (53 times) between the 6th and 8th day; the remainder varied in this respect, it being in some on 9th—11th, less commonly on the 5th, and most rarely of all on the 3rd or 4th day. Quite similar in this respect were the 39 cases which I formerly collected.[1] Only in one case did the crisis take place on the 17th day, which was to be explained by the fact that in this case the pneumonia appeared in two attacks, separated from one another by an interval of 24 hours almost free from fever; and, what was very remarkable, both attacks affected the left lower lobe. The sudden fall of the temperature was sometimes, but not always, accompanied by a copious perspiration, and in these cases there were also symptoms of collapse, at least of great weakness—continued restlessness, cold extremities, pale sunken features, and very small rapid pulse—so that I was obliged to administer stimulants (wine in large quantities). In a boy of 3 years old, who was admitted during the crisis, I found the pulse (124) so small, the brain so much affected, and the temperature so low (94·6° F.) that we had to administer injections of ether and large doses of camphor and benzoin. The temperature rose again in 24 hours to 99·7° F. I have often observed during the crisis a similar fall of temperature to 95° or 94·5° F. Moreover, in almost all my cases the exact time when the crisis occurred could not be determined with absolute certainty, because it often took place during the night, at a time when the thermometric observations were only exceptionally made. It was therefore often uncertain whether the sudden fall of temperature took place at the end of an even or at the beginning of an odd day. I have also repeatedly observed that in the course of pneumonia, the continuous very high temperature fell temporarily between the 3rd and 5th day; *e.g.* from 104° F. to 101·9° F.; then after 12—24 hours again reached its high level, the real crisis not occurring for some days. It remained undecided whether the appearance of this "dies index" and the fresh exacerbation which followed it, depended on a fresh advance of the pneumonia. By physical examination, at any rate, it was not possible to discover any such advance.

[1] *Beitr. zur Kinderheilk., N.F.*, S. 169.

The crisis was not always complete at once—the temperature, which in the evening was 104° F. or more, falling on the following morning to 98·6°—99·5° F., and remaining permanently normal or sub-normal (97·7° F.); for I have repeatedly seen cases in which the crisis occupied a longer time, about 24 hours; *e.g.* as follows:—

Anne B., 7 years old, admitted on 8th March, with pneumonia of the left lower lobe.

	M.	E.
On the 8th March	—	104·9° F.
„ 9th „	104·9°	105·6°
„ 10th „	104·5°	105·1°
„ 11th „	102·9°	103·3°
„ 12th „	102·7°	104·2°
„ 13th „	101·8°	102·9°
„ 14th „	96·8°	97·0°

There also happened occasionally, in the first few days after the crisis, another sudden ephemeral rise of temperature (100·4°—104° F.), the cause of which could not be discovered. It did not recur, nor in any way influence, the further favourable course of the disease. Thus in one boy the crisis took place between the 6th and 7th days, and the temperature fell in the morning to 97·7° F., at noon it was 99·1°, but rose in the evening again to 104·3°, and it was only on the 8th day that the fever permanently disappeared. During convalescence (especially when it is beginning), and in the erect posture, I have frequently, like most other writers, observed irregularity of the pulse. Perhaps the molecular changes in the heart-muscle which occur in diseases where the fever is very high, and which are afterwards again recovered from, account for this symptom.

One case which ended fatally was distinguished by an extremely acute course. It lasted scarcely 9 hours.

Boy of 4 years who had been treated successfully in the ward towards the end of 1873 for diphtheritic nephritis. He had been convalescent for 14 days, and on 9th December at midday the temp. was still 98·4° F. In the evening he felt suddenly unwell; temp. 102·4° F., pulse 158. Persistent severe cough, increasing dyspnœa. After a few hours, a dull note, indeterminate breathing and sharp râles below the spine of the scapula on the right side. At 3 A.M. death, with extreme dyspnœa. At the *P.-M.*, hepatisation of the whole right lower lobe was found; catarrh of left lung; kidneys normal; heart somewhat enlarged and

pale. Unfortunately its microscopic examination was omitted, since from other observations it seems to me probable that there must have been a degeneration of the heart-muscle, due to the diphtheria which had caused the rapid and fatal course of the pneumonia.

I have already[1] published a case which ended favourably with a regular crisis after lasting only three days.

The pneumonia began in this case on 8th April, at 5 A.M., with high fever; the boy who was 10 years old and was suffering from catarrh, having exposed himself on the previous day to a keen east wind. On the evening of the 9th the hepatisation of the right lower lobe could be distinctly made out. On the evening of the 10th the temp. was still 104° F., but at 8 P.M. a warm perspiration set in, which lasted all through the night. On the 11th the fever was gone, and all the symptoms disappeared so rapidly that on the 12th very little dulness could be found.

The following case had a similar course, but the crisis in it took place in the night between the 2nd and 3rd days.

Max S., $11\frac{3}{4}$ years old, admitted on 27th June, 1876, with non-febrile catarrh. On morning of 30th, sudden high temperature (104·9° F.), resp. 40, pulse 136. Cough and pain on left side on breathing. Indeterminate breathing below the scapula. On 1st July dulness, bronchial breathing and sharp râles. Temp. 104°—104·7° F., resp. 48. In the evening great weakness. Typically rust-coloured sputum. Next morning (beginning of 3rd day of disease) after copious perspiration and a good sleep, the child felt quite well. Temp. 98·6° F., resp. 25, pulse 80. On the 4th, dulness had quite disappeared, and only harsh breathing with occasional râles were audible.

A few examples of a still shorter course have also been published, some even of only one day (Leube, Weil), observed in adults. These cases of so-called "abortive pneumonia" have caused the French physicians—especially Cadet[2]—to describe acute pulmonary congestion as a special disease. I cannot regard this assumption as justifiable, attractive though it be, because as yet there are no pathological grounds for it. The short course, the rapid change of the physical signs and the speedy resolution are not in my opinion sufficient points of distinction; for how rapidly within a few days after the crisis the physical signs of true pneumonia can clear up, is shown by the last case. This does

[1] *Beitr. zur Kinderheilk., N.F.*, S. 167.

[2] *Loc. cit.*, p. 1, and Revilliod ("Notes clin. sur quelques maladies des enfants": Paris, 1886, p. 90).

not always happen, indeed; but apart from those rare cases which take on a chronic course, I have seen in the majority the clear percussion note and normal breathing return after a week, or at latest after ten or fourteen days—unless there was some considerable pleuritic exudation keeping up the dulness at the base for a longer time. I have only seen three cases that formed an exception to this rule in the remarkable fact that the physical signs were in process of resolution even before the crisis commenced.

> Heinrich S., 9 years old, admitted 11th May, 1877, healthy. In the night, 7th—8th, had complained much of pain in the head and belly, and of thirst and fever; repeated vomiting. After that anorexia, thirst, fever delirium at night, and slight cough. Temp. on admission 104·7° F.; pained expression of the face, cheeks flushed, eyes generally closed, drowsiness. Pulse 120, of high tension. Respirations 60, superficial. Abdomen tender on pressure. Percussion over the left side behind, dull from apex to base; in this situation fine sharp râles. Nothing else abnormal. Evening temp. 105·1° F. The following day brought no change; but on 13th—while the high temp. still lasted (105·1° F.), the pulse was 120, and the resp. 60—we found the dulness almost entirely gone, and instead of the fine sharp crepitations there was nothing to be heard but mucous râles. The high temp. (varying between 104° and 104·9° F.) continued till morning of 16th, when the crisis suddenly took place and the child felt quite well; temp. 97·7° F.; and rapid recovery followed.

The statement of Grisolle[1] that in 26 patients with pneumonia the auscultatory signs had remarkably improved even during the height of the fever, was criticised unfavourably by other writers (*e.g.* Fox) on the ground that Grisolle had estimated the fever by the unreliable indications of the pulse and not by the thermometer. The case just given, as well as the remark of Sidlo[2]—that in 37·5 per cent. of the cases the local process, as far as it could be made out by physical examination, came to an end 41 hours on an average before the onset of the crisis—goes to support Grisolle's assertion.

Croupous pneumonia, in children as in adults, is a disease in which the prognosis is exceptionally favourable, unless it comes on under very unfavourable circumstances (nephritis, typhoid, tuberculosis). Out of 64 of my cases only 7 died, and

[1] "Traité de la pneumonie," p. 307.

[2] *Deutsches Archiv f. klin. Med.*, Bd. xiv., S. 348.

of these one was found at the post-mortem to have the whole right lung hepatised, another had double pleuro-pneumonia and purulent pericarditis, a third had diffuse peritonitis, and a fourth tuberculosis in many organs. The greater the extent of the disease, the greater is the danger of insufficiency of breathing-power, and this explains the far more favourable prognosis of fibrinous pneumonia, which usually affects only a portion of the lung, as contrasted with the diffuse catarrhal form. For this reason there is always less cause for anxiety if the pneumonia is limited to one side, and the symptoms of hepatisation are confined to the back or front, and do not affect the whole thickness of the lobe. The prognosis is worse if catarrh is present at the same time, or copious pleuritic effusion; but the trifling pleurisy which is almost always present need cause no anxiety. Nor yet need the less common termination of the fever by lysis, which may take days (in one case as many as 12 days), although with it we cannot exclude the possibility of the consolidation lasting for a considerable time, or of its passing into a chronic condition. I have only twice had an opportunity of observing the termination by the formation of an abscess, and it was followed by recovery.

In April, 1875, I was asked to see a girl 7 years old, who had been formerly quite healthy and was now suffering from double croupous pneumonia. Besides hepatisation of the whole right lower lobe there was also dulness and bronchial breathing at the lower part of the left posterior base. The crisis took place on the 7th day; the temp. fell to 99° F. with copious perspiration and threatening symptoms of collapse, but only for a few days. While the signs of consolidation at the left base rapidly disappeared and the fever returned and soon assumed a hectic character with intermittent elevations of temp. There was also persistent cough with scanty mucous expectoration, increasing exhaustion and extreme emaciation causing the most serious apprehensions. At the same time we were unable to make out any cavity on physical examination; dulness and bronchial breathing continued unchanged on the right side posteriorly from the spine of the scapula downwards, while over the apex in front only prolonged expiration could be heard. On 26th May—*i.e.*, about 5 or 6 weeks after the commencement of the pneumonia—an enormous amount of pure pus was suddenly discharged by coughing, with symptoms of suffocation; but the amount, unfortunately, was not measured. From this time all the morbid symptoms improved very gradually; so that on the 14th July the child was

quite well and no longer required treatment. Only indeterminate breathing and a slight dulness at the right posterior base bore witness to the disease which had existed. From the commencement of the hectic fever the child only received decoction of bark, wine and strengthening nourishment; after the abscess burst into the bronchi, she spent the greater part of the day in the garden. I found out that the child subsequently enjoyed uninterrupted good health. The second case had a quite similar course.

The mainly expectant treatment which has recently been recommended in pneumonia, is applicable in children as in adults. I never use wet-cupping except where the dyspnœa is extreme and the disease very extensive or complicated with severe pleurisy, and the violent pain on breathing and coughing seems to call for it. In less robust children, however, dry-cupping is quite sufficient. Where the pneumonia is localised and the pleuritic complication is either absent or at least not a prominent feature, one may dispense with blood-letting entirely and use instead the cold wet pack or compresses to the thorax (p. 397), renewed every half-hour as long as the high temperature continues, and afterwards every two hours. An ice-bag applied to the head is to be recommended; but on the thorax it is too heavy to be borne. The use of luke-warm or cold baths, recommended by Jürgensen especially, I do not approve of in children, because, for one thing, I fear their depressing action on the heart, which is particularly to be avoided in pneumonia; and especially because I regard them as unnecessary. The maxim "ne quid nimis" is here fully applicable. The vast majority of cases run their course, according to my experience, without any active treatment. There is therefore no reason whatever to expose the children to the risk of collapse, which I have seen result from the cold-water treatment of typhoid in children. It may also be added that I have seen just as little permanent effect from cold baths (77°—81·5° F.) during the acme of the fever as from the use of large doses of quinine (grs. viiss—xv.), antipyrin (grs. iv.—viiss), or antifebrin (grs. iss—iii.). Although the temperature is brought down considerably for the next few hours, still this fall is always only temporary, and in order to keep up the action one must repeat the bath or the antipyrin every 2 or 3 hours—a method of treatment which, in children, must be strongly con-

demned. I could lay before you a number of curves such as the following :—

			Temperature.	
			M.	E.
On the 11th May		...	—	... 105·1°
				Bath of 77° F.
„ 12th	„ 9 A.M.	...	103·6°	
	12 „	...	104·6°	
	5 P.M.	...	105° quinine grs. viiss.	
„ 13th	„	...	103·2°	... 105·1° quinine grs. viiss.
„ 14th	„	...	103·2°	... 105·0° quinine grs. xv.
„ 15th	„	...	104°	... 104·2°
		Bath of 81·5° F.		
„ 16th	„ Crisis.			

I have therefore gradually abandoned the use of quinine and other antipyretics also, and confine myself to the local use of cold—especially of compresses, first lukewarm then cold to the chest and abdomen. If you will or must order medicine internally, the best thing to give is infusion of digitalis with nitrate of potash (Form. 22), which, however, is contra-indicated by gastric complications (repeated bilious vomiting, thick coated tongue, nausea). In that case you had better order hydrochloric acid (Form. 3), or ipecacuanha (Form. 16). I only use antimony as I have mentioned (p. 397) when the gastro-hepatic symptoms are very prominent (constant frontal pain, retching, fœtor oris), but in these cases it is very successful. You must at the same time take care that the diet is moderately nourishing (milk, beef-tea, and a little wine). The collapse which sometimes comes on at the crisis is most effectively warded off by large quantities of wine and injections of camphor and ether. Still the occurrence of such collapse is not common.

VII. *Chronic Pneumonia.*

Acute pneumonia, whether it ends with crisis or lysis does not always undergo such rapid resolution. The physical signs of consolidation of the lung may continue for weeks, even for months; and in that case there is always apprehension lest changes in the lung may occur—such as caseation, gangrenous disintegration and the formation of phthisical cavities—which seriously endanger life. This result follows broncho-pneu-

monia far more commonly than it does the croupous form, whenever the conditions (hereditary tendency, unfavourable circumstances) favour such a change in the exudation. You will however remember (p. 390) that even when broncho-pneumonia has a very insidious course, with unfavourable symptoms (emaciation, fever, diarrhœa) a favourable termination, though not expected, is always possible; and I think I may conclude from certain of my cases that croupous pneumonia also may take a similar course.

Max K., 6 years old, admitted into the ward on 17th March, 1873, with eczema capitis and bronchial catarrh. On the 19th sudden development of croupous pneumonia of the right lower lobe. Temp. 105·1° F., pulse 160, resp. 44. During the following days the temperature varied between 103·6° and 105·8° F. Dulness, sharp râles and bronchial breathing over the right back reaching to above the spine of the scapula, and limited by the axillary line. There was also drowsiness, delirium and restlessness. Wet-cupping (on account of pleuritic pain), cold baths, quinine—without apparent effect. On 25th—*i.e.*, on 9th day of the disease—the temp. fell to 100°—100·8° F., which lasted for 4 days (lysis) accompanied by copious perspiration and an eruption of herpes labialis. On 31st—the 14th day—complete disappearance of the fever, while the cough still lasted, and the physical signs in the right lower lobe gradually improved, the dulness cleared up somewhat and the breath sounds remained indeterminate and were accompanied by fine râles. Within a few days, however, a slight evening rise began, being sometimes also observable in the morning; so that the temp. for a fortnight, up to April 21st, varied between 100° and 101·1° F. While the rate of the resp. was but slightly increased (26 to 30, rarely 40), the cough severe, and there was a great tendency to perspiration, the dulness disappeared entirely for the first time towards the end of April, indeterminate breathing and râles being left behind. About the same time a remittent rise of temp. was observed for a few days (from 26th to 28th April). The same took place from 4th to 27th May (temp. always 100·8°—101·3° F.). Increasing pallor and emaciation in spite of a tolerable appetite, and the indeterminate breathing with prolonged expiration and râles, still audible at the affected part, were all the more suspicious because the scanty mucous expectoration was now frequently streaked with blood, and was gradually becoming purulent. Under the microscope, however, nothing could be made out but pus corpuscles and epithelial cells, never fragments of any other tissue. On 27th May, —*i.e.*, 2 months after the beginning of the pneumonia—everything had returned to its normal condition, and the child could be discharged as cured.

It seems to me that this slow course of croupous pneumonia tending to become chronic, but ending finally in recovery, occurs more frequently in children than in adults. In the case just given there can be no doubt that the croupous form was present from the first. In others, in which you see the disease only after it has lasted for some time—and therefore have not observed its first development—it is often impossible to say whether it originated in the croupous or catarrhal form. Some of the cases which I formerly published[1] as examples of "chronic pneumonia" were made doubtful in this way; while in others the broncho-pneumonic character was clearly established.

These children were between $1\frac{1}{2}$ and 4 years of age, but I do not doubt that older people may also be similarly affected. They were pale, more or less emaciated and flabby, with an expression of suffering; so that their whole aspect indicated the presence of a serious disease. Some weeks or months before—according to the usual history—the illness had commenced with an attack of "inflammation of the lungs," which was either primary or the result of measles, whooping-cough, or typhoid. Ever since, there had been an obstinate cough, shortness of breathing, noisy expiration, and a certain amount of fever. Very often to these were added anorexia, coated tongue, and diarrhœa; and then the emaciation appeared more rapidly. In most cases I have found signs of consolidation in an upper, more rarely in a lower lobe; dulness on percussion, weak or indeterminate breath-sounds, bronchial breathing and bronchophony, a greater or smaller number of sharp râles. The accompanying fever has almost always the remittent type, but may also be misleading from its resemblance to intermittent fever. I shall never forget the child of a country gentleman, sent to me with the diagnosis of intermittent fever, but whose emaciation, shortness of breath and cough, at the very first glance gave one the impression of a case of lung disease. On examination I found consolidation of the left upper lobe, the result of a pneumonia some months before. He was completely restored by spending two winters in the south. In cases such as this I have repeatedly seen bloody sputum, although only in the form of specks or streaks in the muco-purulent matter, which during the chronic course of the disease the children learned to expectorate.

[1] *Beitr. zur Kinderheilk., N. F.*, S. 189.

Examination sometimes reveals catarrhal sounds in the other lung also, and from time to time an acute catarrh is added to the chronic affection, and must be treated very carefully. Under such circumstances we are always justified in suspecting phthisis; and a certain proportion of the cases do in fact end in this unfortunate way, owing to caseous metamorphosis and breaking down of the inflammatory products. But experience has taught me that cases of this kind even when they seem quite desperate may yet be completely cured. It may, of course, take a long time; for instance, I have been able to make out the remains of consolidation after a full year, often after 6—9 months; while the other respiratory symptoms had quite disappeared, and the general health and nutrition had been completely restored.

That broncho-pneumonic consolidation in particular may last for many weeks—and even months—without becoming caseous, I have repeatedly satisfied myself by post-mortems on children who had presented the clinical signs of broncho-pneumonia during that length of time. We must therefore admit the possibility of the complete absorption of the fattily degenerated contents of the alveoli, even after such a long period. On the other hand the lung may become indurated owing to hypertrophy of the interstitial connective tissue; and here the process generally stops.[1] The proliferating interstitial connective tissue gradually contracts, and the lung becomes indurated and greyish-white or bluish in colour. In young children in particular, the whole lung or lobe of the lung—especially the upper lobe—may be changed in this way into a firm mass which creaks on being cut, and in which the obliterated bronchi are distinctly recognisable as white bands. When the disease takes this termination, the physical signs of consolidation last, of course, during the patient's life, unless masked by emphysematous distension in the neighbourhood. You therefore generally find when the upper lobe is the seat of the contraction that the subclavicular region on the affected side is flattened or retracted and less movable on inspiration than that of the unaffected side.

Occasionally, however, there occur in children as in adults bronchiectases in the contracted portion of lung at

[1] Steffen (*Klinik der Kinderkrankh.*, i., S. 422) describes these processes under the name of "interstitial pneumonia" and is of opinion that they may occur both in the catarrhal and in the "diffuse croupous" form, if the course is protracted.

the same time. The cases of this kind which I have seen presented exactly the same features as one sees in adults :—dulness on percussion, numerous coarse and occasionally sharp râles, flattening of the front of the chest on the side affected, high level of the diaphragm, &c.; and especially a severe spasmodic cough with copious purulent sputum which was usually fœtid and often mixed with, or even composed alone of blood.[1]

As to the treatment of chronic pneumonia, I have but little to tell you. Our main object is to favour the absorption of the inflammatory products, and to protect the little patient from all injurious influences which might cause fresh catarrh or inflammation, and might disturb the process of contraction just mentioned, should such contraction be inevitable. Protection from chills and tonic treatment (by bark and cod-liver oil) are the most important means. Although I have effected little or nothing with quinine—even in regard to the evening rise of temperature—I have seen good results from the use of decoction of cinchona (Form. 23) continued for months; or from extract of cinchona (Form. 24). I do not order more than two dessert-spoonfuls of codliver oil in the day, to avoid causing dyspepsia. It is a necessary condition for the use of both these medicines that the digestive organs be unaffected. For well-to-do people, the thing which ought to be most strongly recommended is residence in a calm, pure, mild atmosphere; and several of my cases in private practice, which seemed at first to justify a very gloomy prognosis, were completely restored by spending a number of winters at Montreux, Meran, or on the Riviera. Nourishing diet is likewise a matter of the first importance; and also the careful attention to any attack of dyspepsia or diarrhœa which may tend to interfere with the successful treatment.

In cases of extensive shrinking of the lung with bronchiectasis, I have frequently used the much recommended inhalations of turpentine; but I have only found them yield slight and quite temporary benefit, or even cause positive harm owing to

[1] One case of this kind (with copious hæmoptysis) observed in my wards has been described by H. Braun ("Beitrag zur Casuistik der Bronchiectasien im Kindesalter." *Inaug.-Diss.:* Berlin, 1887). The cause of the hæmorrhages was the formation of an immense number of new blood-vessels in the dilated bronchi, which were denuded of their epithelium and, in places, of their mucous membrane also.

their setting up a fresh catarrh, which may even be accompanied by fever. I have seen just as little lasting result from other inhalations, or from the pneumatic chamber.

VIII. *Pleurisy.*

Pleurisy in children differs in no essential particular from the same disease in later life. It is by no means rare. I have found chronic latent pleurisy, leaving behind more or less extensive adhesions of the pleural surfaces, in a surprisingly large number of the post-mortem examinations I have made in children in the first year of life who were not at all tubercular. I have also often enough discovered pleurisy with effusion in children even of 5—9 months with unmistakable symptoms and more frequently after the end of the first year. Acute pleurisy with its sharp pains, short cough, quick shallow breathing and more or less high fever, is in children in every respect similar to that in adults. Older patients localise their pains very exactly, while younger children mistake the real seat of the pleuritic pain and frequently complain of the "belly," although on physical examination we distinctly find all the signs of pleurisy. Under these circumstances percussion also helps in the diagnosis because it generally, like palpation of the intercostal spaces, excites pain and draws the attention of the physician to the true seat of the disease. Little children who are too young to complain of pain cry when they cough and make faces as if in pain; but this symptom is unreliable, and at this age only physical examination can furnish us with reliable criteria. Moreover, I have occasionally seen older children in whom the pain was quite absent; *e.g.*, in a girl of seven who was suffering from severe febrile pleurisy with effusion (the whole left side of the chest being filled with fluid), and had not complained once of pain.

Acute pleurisy in children is sometimes ushered in by "cerebral" symptoms (vomiting, epileptiform convulsions); but far more rarely so than croupous pneumonia. This manner of onset draws away the physician's attention from the real seat of the disease. We only find this symptom in children of 1—5 years.[1]

[1] *Journ. f. Kinderkrank.*, Bd., xiii., S. 2, 1849.

Otto N., 3¼ years old. In the end of October, 1846, a fall on the forehead followed by ecchymosis. On the evening of 30th October, sudden high fever, which persisted through the night, and at 10 A.M. on the 31st an epileptiform attack took place. After half an hour he awoke from his drowsiness; headache, inability to sit upright, or to hold the head erect. Fever persisting, pulse 160, drowsiness. About 2 o'clock, a second epileptiform attack. About 6 o'clock seemed all right, at play. During the night continuous fever, vomited once. Remained about the same till 15th November; forenoon remission, evening exacerbation of the fever with circumscribed redness of the left cheek, occasional slight cough. On the 15th for the first time I determined to examine the thorax; for I had hitherto neglected to do so, being then a very young and inexperienced practitioner and full of the idea of meningitis. I at once discovered a considerable pleuritic effusion on the right side of the thorax. Percussion dull, laterally and posteriorly over the lower two-thirds, breath-sounds and vocal fremitus quite absent in this area, the intercostal spaces distended; R. 60, scarcely noticeable on the right side; P. 124. Cough trifling, generally only in the evening, complaints of pain "in the belly." Liver displaced downwards. Child always lies on the affected side. Urine abundant, clear. From 15th to 27th hectic character of fever, emaciation, much perspiration during the night. Gradual improvement under strengthening diet and tonic treatment (decoct. cinchon.). On 22nd December lateral percussion almost normal, posterior still quite dull; breath-sounds audible, increase of strength and bulk, better colour, fever subsiding. After 25th December no more night-sweats. Besides the cinchona, ol. morrhuæ, 2 dessert-spoonfuls daily. On 10th January, 1847, seemed quite well, position of liver normal, still some impairment below the scapula behind. On 14th February discharged without any deformity of the thorax of importance.

This case, which occurred in the second year of my practice, impressed me so much that from that time forward I never neglected the examination of the thorax in any febrile disease, even when no symptoms seemed to call for it. I cannot sufficiently urge this upon you, for it was only thus that I managed to avoid the same error in some similar cases.[1] The mother's statement —that the symptoms arose immediately after a fall on the head —is the very last thing you should rely upon; for this explanation is one of the commonest simply because little children are always falling.

Boy of 4 years. Fall on the head 14 days before. For some

[1] *Beitr. zur Kinderheilk., N. F.*, S. 199.

days drowsiness, high fever with evening exacerbations. P. regular, rapid. Frequent spontaneous vomiting, constipation; inability to hold the head erect. First examined on 10th January, 1875; thoracic organs normal. Cessation of the suspicious symptoms after 5 days; slight cough. Pleural effusion on the left side behind and below. Re-absorption after 2 weeks.

Otto R., 9 years old, brought to the polyclinic on 17th March. On the previous afternoon a fall on the head. Headache and vomiting ever since, especially on changing the position. Apathy, screaming during sleep. Pupils normal. Fever; P. 156, regular. Beneath the left scapula slight impairment on percussion with vesicular breathing, reaching round to the axillary line. Complained greatly of pain at this spot, especially on coughing and on deep inspiration. Tender on percussion. Digitalis with pot. nitrat., 5 wet cups. On the 18th pain considerably abated. On the 24th dulness still continuing, distinct friction-sound. On 21st April everything normal.

The preliminary brain-symptoms appear therefore, in such cases, either in the form of headache, vomiting and obstruction, drowsiness and delirium; or else, in little children, as epileptiform convulsions similar to those in croupous pneumonia. Here also the high temperature seems to be the cause of these symptoms, since we find that when it falls and the signs of exudation become more distinct, the brain usually becomes clear. More frequently the disease begins with gastric symptoms which may mislead the physician for days—nausea, anorexia, thickly coated tongue and complaint at night of pain in the body; and to these jaundice was added in the case of two of my patients (of whom one was suffering from pleurisy on the left side). A boy of three years, who had been ill for a week past, complained of pain in the left inguinal region, while the left half of the thorax was completely filled with effusion. In all these cases there were however at least some morbid phenomena which caused anxiety to the parents and led them to seek medical aid. Those cases are more difficult to recognise which develope subacutely or quite gradually, and run their course without any striking symptom of a serious respiratory affection. Cases of latent pleurisy are, as far as my experience goes, more frequent in children than in adults, probably because when the latter feel ill they get themselves examined; while in the former, the symptoms, being apparently trifling, are overlooked by parents who are not over-careful.

Elise B., 7 years old, had measles in autumn, running a perfectly normal course. In the middle of January the child, who had hitherto been perfectly healthy, began to get feverish every evening, and during the night she was very hot, thirsty, restless, and short of breath, while during the day she seemed pretty well. The appetite also was gradually lost, and the child became pale. I was called in for the first time on 5th February. On the left side from the fifth rib downwards, especially laterally and posteriorly, percussion-note quite dull, absence of breath-sound, and vocal fremitus; higher up, puerile breathing. Respiratory movements normal, no cough, no pain; still, when I asked her the child remembered that she had several times felt a slight stitch in January. Treatment: rest in bed, warm poultices to the affected side, infus. digital. with pot. acetat. for the scanty secretion of urine. On the 10th, profuse diuresis, no more fever, percussion clearer. On 1st March, everything normal and the child seemed quite well.

In this and similar cases, the parents' neglect was to blame. Especially in young children the inoffensive "teeth" are made answerable for the illness, until after weeks increasing emaciation, shortness of breath and cough at last occasion anxiety and the physician is consulted. I must, however, unfortunately add that in spite of all warning examples—of which I have published several[1]—inexplicable mistakes are always occurring in this insidious form of pleurisy, even on the part of medical men. It is not ignorance that we have to find fault with, but rather indolence, the shrinking from a thorough examination, and the idea that with such trifling respiratory symptoms no serious disease can exist in the thorax. The "latency" of the pleurisy is owing, not to the nature of the disease, but to the carelessness of the physician. Especially often I have met with such cases in practice among the poor and in children who had attended a polyclinic—where the large number of the patients is apt to lead to off-hand prescribing without careful examination. But even physicians in private practice are guilty of such sins of omission.

On 6th November, 1873, for example, a pale little boy of 3 was brought to my polyclinic, who had taken ill with fever about 8 days before, and who had been referred to the hospital by his doctor who was well known to me as conscientious ("because he could not make out what was the matter with him"). This prac-

[1] *Journ. f. Kinderkrank.*, Bd. xiii., S. 1, 1849.—*Beitr. zur Kinderheilk., N. F.*, S. 197.

titioner acknowledged to me afterwards that he had not examined the thorax even once, because no symptom seemed to point to it. There was certainly no pain at all, and only a quite trivial cough; but the respiration was somewhat quickened and a rise of temp. took place twice daily, between 9—10 A.M., and between 5—6 P.M. On examination we found the whole left pleural cavity filled with effusion, pushing the heart to the right. On 27th February, 1874, there was still some effusion to be made out at the base behind. Still more blame attached to the physician of a boy of 4, who had given an entirely false explanation of the bulging forward of the chest which was filled with pleuritic exudation, and had declared that the child must undergo a course of orthopædic treatment.

So much for the peculiarities of the general course of the disease. In the matter of physical signs, which correspond to those in adults, I would only point out the frequent occurrence of bronchial breathing in the pleurisy of children; which, as we learn from post-mortems, occurs without pneumonic complication, and is simply caused by the effusion compressing the lung. Those who are interested in explanations, will find them given by Rilliet and Barthez,[1] and Ziemssen.[2] I shall here only insist upon the fact that especially in recent cases, bronchial breathing is almost invariably heard over those parts of the thorax which are dull, and that only gradually, as the effusion increases, is it replaced by weakening and finally by complete disappearance of the breath-sounds. In little children, therefore, the absence of sputa and the difficulty in making use of the vocal fremitus for diagnostic purposes, always renders it doubtful whether pleurisy or pneumonia is the principal disease; while in older patients the above-named points usually enable us to arrive at a diagnosis. Should bronchial catarrh happen to be present in such a recent case of pleurisy, the mucous râles sometimes assume a sharp character from compression of the lung tissue, and may—specially in exhausted feverish children—excite a suspicion of phthisical cavities, which turns out later to have been unjustified. In purulent pleurisy in children especially, the first and second intercostal spaces (in front and close to the sternum) where they are widest and most yielding—often appear abnormally bulged forward, as has been borne out by the recent experiments of Rivet[3] (injection of

[1] *Loc. cit.*, i., p. 555. [2] *Loc. cit.*, S. 71.

[3] "De la voussure sous-claviculaire dans les épanchements pleuraux chez l'enfant," *Thèse:* Paris, 1880.

water into the thorax). The fact that this region is the spot where rupture most frequently occurs agrees with this observation.

Most children with pleuritic effusion lie, as adults do, on the affected side. This is seen even in little children in the first year of life; and this accounts for the fact that infants with pleurisy prefer to take whichever breast allows them to lie on the affected side when sucking. I have observed that children with effusion in the right pleural cavity would only take the left breast, and *vice versâ*; otherwise their sucking was interrupted by violent dyspnœa. In one case of this kind the mother even made the mistake of supposing that the infant's preference for the left breast indicated something wrong with the right.

Among the complications of pleurisy, pericarditis seems to me commoner especially in very young children than in adults. In one child of 5 months, I found, besides double fibrino-purulent pleurisy, a considerable effusion of a similar character in the pericardium. In another child of 8 months who had broncho-pneumonia, especially in the right lung, I found considerable purulent effusion in the left pleura and in the pericardium, the visceral layer of which was covered with villous deposits of fibrin, especially on the front of the heart. That there had been in this case an extension of the inflammation from the left pleura to the pericardium was proved by the firm adhesion of the outer surface of the latter to the left lung. In the following case, however, there was an old loculated effusion in the right pleural cavity, complicated by chronic pericarditis and endo-carditis.

Eleonore P., 3 years old, admitted into the ward on 18th September. Poorly nourished, pale. No history whatever obtained. The right side of the thorax dull on percussion over almost its whole extent, with the exception of the upper part in front, which gave a somewhat clearer note. Sternum and left half of the chest normal. The breath-sound quite absent on the right, behind and at the side; indeterminate over the upper front with bronchial expiration. Right half of the chest ⅔ in. narrower than the left, scarcely elevated on breathing. On the left side behind, some sonorous rhonchi. Cardiac dulness somewhat enlarged towards the right, apex-beat in the fifth intercostal space in the mammillary line, loud systolic murmur at the apex. No fever. Diagnosis—incompetence of the mitral valve, dilatation of the right ventricle, old fibrinous pleurisy of the right side with contraction of the thoracic cavity. In the course of the next few

months the child's condition became steadily more wretched owing to repeated attacks of intestinal catarrh. The bronchial catarrh also underwent exacerbations from time to time with slight rises of temperature. After 25th January, 1873, mucous sputum mixed with bright red blood was sometimes expectorated, and at the same time very distinct sharp râles, loud bronchial breathing and bronchophony were heard on the right side above, near the sternum, and also above the clavicles where the percussion-note was somewhat clearer. In the last days of January she developed typhoid, which ended fatally on 7th February.

P.-M.—Firm adhesion between the pericardium and left lung, the former thickened, both layers firmly adherent to one another. Mitral valve thickened, stiff and incompetent, both ventricles hypertrophied, the right also dilated. Old fibrous patch under the endocardium $\frac{2}{5}$ in. below the aortic orifice. Almost the whole of the left lung vascular, brownish-red. Right lung much diminished in size, pressed upward and forward and at this point adherent to the pericardium. At its lateral and posterior margin an enormous sac with extremely thick and tough walls which was so firmly adherent, on the inner side to the lung, and on the outer to the thorax—that it had to be separated by the knife. In its interior it contained a quantity of creamy, greyish-red matter. The left lung was densely carnified. Catarrh of the large bronchi. Typhoid.

How the pleurisy, in this case, came to be complicated with pericarditis, it is impossible to say. What we found was only the result of these chronic diseases—the firm adhesion of the pericardium to the heart, incompetence of the mitral valve with dilatation of the right ventricle, and a considerable loculated effusion enclosed by a thick membrane. The retraction of the whole right lung forwards and upwards which seemed to be caused by old adhesions between it and the pericardium, in this case gave rise at the time to an error in diagnosis. For I thought that the bronchial breathing and sharp râles heard after the 26th January over the upper part of the lung anteriorly, taken along with the bloody expectoration, must be due to the presence of a cavity in the upper lobe, while we found on post-mortem examination that these phenomena were simply caused by catarrh of the right principal bronchus and by the dense carnified lung which rested immediately upon it.

Caries of the ribs is more frequently a cause of pleurisy in children than in adults. Of this the following case is an interesting example [1]:—

[1] *Charité-Annalen*, 1. Jahrg., S. 586.

Margarethe M., 5 years old, admitted on 15th April. She had had numerous abscesses of the connective tissue ever since birth, anæmia and atrophy. On admission enormous ulcer on the scalp, abscess the size of an apple just under the sacrum, numerous enlarged glands in the neck and in the inguinal region. Incision of the abscess, which healed by 3rd May. The ulcer on the head gradually cicatrised. The child was free from fever, very pale and weak. Fresh abscesses formed in the neck up till 6th June and were opened. On the 7th June, close to the right breast a roundish swelling, rather more than an inch in diameter, not reddened but fluctuating, which gradually grew to the size of an apple and was opened under the spray on the 20th. From this time high temperature (evening 101·3°—102·9° F.), which, however, was absent for days at a time. Close to the right shoulder-blade a new abscess of considerable size formed; opened 11th July, and a carious rib was felt by the probe. About the same time we found on examination, so far as this was practicable on account of the swelling and painfulness of the affected part, dulness over the right side of the thorax both in front and behind increasing towards the base, abundant crepitations, some of which had a sharp character, and indeterminate breathing. On the 10th we observed for the first time that on deep expiration, especially on crying, a quantity of pus bubbled out on to the chest from the abscess wound, mixed with a large quantity of air-bubbles. This condition continued till her death on 18th August.

P.-M.—The 5th, 6th and 7th ribs on the right side carious; between them (*i.e.*, within the intercostal spaces) there were a few openings the size of a pea through the costal pleura into a cavity. Pericardium completely adherent to the heart, and right lung to the pericardium. The right lung felt very tough and was adherent over its whole surface to the chest-wall. The pleura costalis and pulmonalis form thick indurated masses of fibrous tissue. In the immediate neighbourhood of the abscess-wound on the thorax, there was the already-mentioned cavity situated between the the two layers of the pleura and filled with about 8 tablespoonfuls of purulent pleuritic effusion. The pulmonary pleura in the neighbourhood of the cavity was wanting, so that a probe could be passed directly into the small bronchi. Almost the whole right lung carnified.

The extensive caries of the ribs in this case evidently formed the starting-point of the abscesses near the mamma and shoulder blade as well as of the chronic pleurisy. Besides the adhesions and fibrous membranes, it gave rise to the cavity filled with pus which communicated on the outside with the abscess in the chest-wall, and finally also it had penetrated the pulmonary

pleura inwards by a process of necrosis. In this manner air was enabled to find its way out of the lung into the cavity and then outside along with the pus of the abscess. The firm adhesions which surrounded it prevented the occurrence of pneumothorax. Here also the inflammation spread from the pleura to the pericardium and caused complete adhesion of the two layers of the latter to one another and to the right lung.[1]

In children tuberculosis and pneumonia (croupous more often than catarrhal) are also important factors in the causation of pleurisy. When the two diseases are combined, as is so commonly the case, the pleurisy as a rule is least important, and indicates its presence only by pain and by slight effusion at the base (p. 407). Still cases do also occur in which pneumonia, which at first was the more prominent condition, yields place to the pleurisy, and it developes further and leads to a more or less considerable effusion (pleuro-pneumonia). How rapid the pus-formation under these circumstances may be is shown by the case of a boy of 5 from whose right pleural cavity more than 35 oz. of pus were evacuated by puncture on the 6th day of the disease. In broncho-pneumonia we find when both sides are affected the pleurisy also is not uncommonly bilateral, both lungs being covered with fibrino-purulent deposit, also perhaps purulent exudation being present in both pleural cavities. Putrid pleurisy I have only observed exceptionally in children (apart from cases where the discharge became offensive after operation), *e.g.* in the following case:—

> Anna O., 11 years old, treated in the ward in May for pleuro-pneumonia of the left side, discharged 26th May. Re-admitted on 4th June. Rigor 5 days before, since then persistent fever, cough, pain in the left side in which a considerable effusion could be made out. T. 103·1° F., R. 44, P. 124. The left side of the thorax scarcely rose during breathing. The intercostal spaces filled out, dulness on percussion almost all over, bronchial breathing, no vocal fremitus, dulness over the sternum, heart sounds audible most distinctly near the right border of the sternum. Urine scanty, but otherwise normal. Wet-cupping, wet compresses round the thorax and digitalis were practically

[1] We must not confound with these cases those in which purulent pleurisy forms the primary disease and caries of the ribs only arises secondarily and may then lead to abcesses in the chest wall and communication with the pleural cavity. *Cf.*, *e.g.*, a case of diaphragmatic pleurisy from my ward, described by Jacubasch in the *Berl. klin. Wochenschr.*, 1883, No. 41.

useless. On the 10th owing to the increasing dyspnœa, the thorax was punctured with Potain's syringe and 11½ oz. of greenish-yellow offensive pus evacuated containing numerous putrefactive bacteria. Although there now occurred a partial re-expansion of the lungs especially of their upper part, and the resp. sank to 32, the fever still persisted unchanged and therefore on the 13th the radical operation for empyema was performed, a silver canula was inserted after evacuation of 17¼ oz. of offensive pus and the thoracic cavity was syringed out with carbolic lotion. Fever now disappeared at once (T. 98·6°—99·5° F.), and after 2 days the discharge from the pleura was odourless. On the other hand the cough increased considerably and the copious greyish-yellow, tough, somewhat sweetly-smelling sputum contained distinct elastic fibres. On account of the blackish colour of the urine a solution of salicylic acid (3:1000) was used for washing out after the 15th instead of the carbolic lotion, and the thoracic wound treated with strict antiseptic precautions. During the next few weeks a rise of temperature was observed on several occasions without any evident cause; for example, on 9th July 104·9° F., but after this attack the child remained quite free from fever until her discharge on 1st May, 1879—that is, about a year after her admission. The wound on the thorax, from which there was always a slight discharge, closed in August, the general nutrition and health were restored gradually, and the rate of breathing was soon only 20 in the minute, the pulse 108. While on the front and on the upper part of the side and back the physical signs had become normal, the lower part of the axillary region and the back from the spine of the scapula downwards still remained much impaired, and bronchial breathing, sharp râles and friction were heard there. The cough also continued with varying severity and the expectoration, which varied in quantity, contained blood from time to time. On every occasion when this occurred the child was kept in bed for a few days. Elastic fibres, however, were no longer found, and on the 1st May, 1879, the patient was discharged in very good health, free from cough, but still with dulness and bronchial breathing in the region of the left lower lobe. The treatment during the last months consisted of inhalations of carbolic lotion (1 per cent.), ol. morrhuæ; and plumb. acet., whenever hæmoptysis occurred.

This case was, in fact, one of a circumscribed patch of gangrene at the periphery of the pneumonic portion of lung, from which the germs of putrefaction had found their way into the pleural effusion, and had caused it to become putrid. The fact that neither on physical examination nor when the puncture was made could pneumothorax be made out, is against the existence of a large communication between the pleural cavity

and the gangrenous patch. On the other hand the hypothesis of fine openings in the pleura of the affected lung, which had later on become closed by adhesions, is more probable.[1] After the cure of the putrid pleurisy by puncture and incision, the necrotic patch in the lung lasted for many months, and indicated its presence by repeated relapses of fever, and by purulent sputa mixed with blood and elastic tissue. At last recovery took place, and nothing remained but physical signs, which were to be attributed to an area of much-thickened pleura at the lower part of the left side of the chest. As I learned later, the child died a year afterwards from an inflammatory chest affection. On the other hand I have in private practice seen a boy of 9 years with a copious right pleuritic effusion following pneumonia of the right upper lobe, who became very feverish for some time, and began suddenly to expectorate putrid purulent sputum; an incision was at once made into the thorax, and the pleural cavity was treated antiseptically, and complete recovery finally took place. The characteristic expectoration proved that the putrid character of the effusion had resulted from the entrance of septic germs through an opening into the upper lobe of the lung.

I have repeatedly also observed pleurisy in children resulting from acute articular rheumatism, scarlet fever especially scarlatinal nephritis, and measles. One of these cases, in which a diagnosis was made only four weeks after recovery from measles, was distinguished by complete absence of fever (temp. never above 99·5° F.), although on two occasions more than 15 oz. of greenish-yellow pus were evacuated by puncture. Only once, in a girl of 5, have I seen a purulent pleuritic effusion in the course of whooping cough, as the result of a concomitant broncho-pneumonia.

On the various terminations of the disease—re-absorption, suppuration, bursting of the empyema externally or internally—and on the resulting deformity of the thorax, I have nothing new to tell you. The former belief, that deformity of the thorax occurs less frequently in children than in older people, is a mistake. On the contrary, we observe considerable retraction occurring on the affected side after insidious purulent effusions which finally burst externally and form suppurating fistulæ lasting for years, as well as in cases where there is a formation of

[1] *Cf.* A. Fränkel, "Ueber putride Pleuritis," *Charité-Annalen*, iv., 1879, S. 256.

thick masses of fibrous tissue between the lung and the chest-wall. In a boy of 14 who had suffered from pleurisy in his 5th year, I could fill up the whole right pleural cavity with my fist.

Finally, a few words on treatment. At the beginning of the disease when there is violent pain, I consider wet-cupping necessary (the number of cups varying according to age), and in weak children, dry-cupping. Next to cupping, wet compresses, such as I recommended for pneumonia, are to be used continuously, while we give internally digitalis (Form. 22) with nitre. Also calomel along with digitalis (Form. 25) is useful, especially when there is constipation. When the effusion increases, diuretic treatment becomes important, infusion of digitalis with acetate of potash and Bilin or Wildung water (3—4 wine-glasses daily) to drink. In the very chronic cases, I would recommend decoction of bark (Form. 23) with acetate of potash (grs. xxx.), codliver oil, whey, fresh country or mountain air and, during the winter, residence in the South, especially on the Riviera.

The greater activity of tissue-change in children favours the reabsorption of serous pleuritic effusion generally, more than is the case in adults. I have, indeed, reports of a very considerable number of cases which recovered perfectly well without surgical assistance, under diuretic and tonic treatment, within some weeks or months. We should not, therefore, be in too great a hurry to operate. For my own part, I recognise only two indications as urgently calling for the evacuation of the fluid.

(1) A rapid increase of it, with acute displacement of the mediastinum and considerable aggravation of the dyspnœa, so that the children are no longer able to maintain the horizontal position for any time, but are obliged often to assume a sitting posture. Under these circumstances, especially when the effusion is on both sides or when there is a complication with bronchitis or pneumonia, early puncture is indicated in order to relieve the lung from the pressure of the exudation. As a rule, the fluid rapidly re-accumulates, but we can in that case repeat the operation if need be; or, if the symptoms are not severe, we may quietly await the re-absorption of the fluid.

Girl of 7 years, examined for first time on 6th July. For about 1½ weeks acute pleurisy of the left side, which had run its course from the beginning without any pain. The left side of the

thorax filled with fluid, and dull note over the sternum. The heart displaced to the right, the left lung backwards and upwards. In front, bronchial breathing; at the side and at the base behind no breathing audible at all. Fever remittent, M. 101·7°, E. 103·5° F. and over. In the beginning of the third week of the illness, increase of the dyspnœa, frequent sitting-up to get breath, pulse small. On the 11th, puncture under antiseptic precautions and evacuation with an aspirating syringe, which was four times filled with clear, greenish serum. During the next few days, until the 17th, the temperature remained high (100·4°—102·6° F.), while the effusion again increased considerably. Then rapid re-absorption, improvement of general health, disappearance of fever. After the 22nd, free from fever. Recovery. The deficient diuresis was considerably improved by infus. digital. and Wildung water.

In this case, therefore, one puncture and aspiration sufficed for the cure, and in serous pleurisy I have frequently observed this. It is also worthy of notice that, although the serous effusion rapidly re-accumulates after puncture, the dyspnœic symptoms do not reach the same degree as formerly, and the respiration usually get rapidly into its ordinary way of working after a few days, as if the removal of the pressure from the pleura by the single puncture had restored its power of absorption.

(2) The purulent nature of the effusion (empyema). The points which were formerly regarded as decisive in the diagnosis of this condition, *e.g.*, the so-called "œdema laterale" of the thorax, are almost all valueless. The latter, especially, is very often absent, and is not observed until the pus has already begun to burrow its way outwards, and forms a localised bulging of the thorax, which is often surrounded by blue distended veins (empyema necessitatis). When this external rupture does not take place, we must attach importance to the character of the fever. A persistence of the fever for weeks with afternoon and evening exacerbations, with emaciation and loss of strength, is in favour of the purulent character of the effusion. But even this symptom is not constant; for, as is shown distinctly by the case just given (p. 430), the fever may last for at least 2½ weeks, with afternoon and evening rise of temperature, and yet the effusion be entirely serous. On the other hand, however, the fever may be quite absent in purulent effusion as in the case of empyema after measles given on p. 429. I have records of a whole series of cases of empyema in children between 4 and 9 years of age where there

was absolutely no fever. In a few, indeed, the temperature varied between 97·7° and 98·8° F. The only certain means of recognising the character of the effusion is therefore the exploratory puncture, which may be made without any danger under antiseptic precautions, either with a hypodermic syringe, or better, with Dieulafoy's aspirator or Fraentzel's trocar. As soon as the aspirated fluid is found to be purulent we must give up expectant treatment and undertake artificial evacuation. Further delay might result in rupture of the empyema through the chest-wall or into the lung, and exhaust the patient by continuous hectic fever, or, in the most favourable case, lead to the drying-up of the pus and to caseous matter being left in the thoracic cavity which might later act as the starting-point of miliary tuberculosis. The method of evacuation is still a matter of dispute. Every year increases the number of examples of complete recovery after one or more simple punctures. Thus, in the case given above, a single puncture was sufficient for the cure of a serous effusion; and in the same way I have also seen in three cases of purulent effusion (one of which was after scarlet fever) the same good result from this procedure without the much recommended washing-out of the thorax. The quantity of pus removed in these cases varied from 21 to 52 oz. We should, therefore, always in children begin by trying this mode of treatment. I always use Potain's aspirator, and I can recommend it highly, especially for use with children. Usually the effusion increases again a few days after the aspiration, but afterwards it remains stationary and at last gradually retrocedes. It is, however, only in a very few cases of empyema that this proceeding will suffice, and after repeating it once or twice we see ourselves at last obliged to have recourse to the radical operation, that is, to opening the thorax by incision, with resection of a portion of rib. As I have already remarked, I have only in three cases seen a lasting result from one or two punctures. In all the other cases I was obliged to incise; and any one who has once seen the masses of coagulated fibrin saturated with pus which are removed from the thoracic cavity by this operation will readily understand why simple puncture is almost never sufficient. We will best obtain outlet for the pus by making the incision over the base at the back or in the axilla, and by introducing a drainage-tube or wide silver canula.

A counter-opening in front is also of great use, especially in those cases where we have to remove a large quantity of coagulated lymph. We endeavour as far as possible to prevent the entrance of infectious elements into the thoracic cavity by applying an antiseptic dressing, and changing it as seldom as possible. On the other hand, the washing-out of the thorax with carbolic lotion which was sometime in favour has fallen into disrepute, owing to carbolic acid poisoning having been observed, and for this injections of thymol, boracic and salicylic acids and chloride of zinc have been substituted. These also, however, are to be used as little as possible, unless there is an offensive odour which calls for them. The success of the operation—especially in children—has been proved by many cases, and I regard it as unnecessary for me to give in detail my own experience which is in favour of the operation being performed even in apparently desperate cases. I cannot impress upon you too urgently the importance of performing the operation without delay, as soon as the purulent nature of the effusion has been established and simple puncture, on two occasions at most, has proved insufficient. Should the exploratory puncture reveal a putrid effusion, the radical operation must be undertaken on the spot.

IX. *Tuberculosis of the Lungs.*

The difference of opinion among anatomists as to how tuberculosis is to be regarded, especially as to its connection with caseous processes, is not yet fully settled. While one party, supported by Virchow, sharply accentuate the differences between the two conditions, the other—especially the recent French writers (Charcot, Grancher, &c.)—take a more intermediate position which, as I believe, is borne out by the clinical facts. Unprejudiced observers, and especially practitioners, cannot overlook the fact that a clinical proof of the essential connection of the two processes with one another is furnished by the frequent association of miliary tubercle and caseous degeneration, as well as by the fact (also proved experimentally) that the former develope from caseous deposits elsewhere; and such clinical proof has greater weight than all the results of microscopic examination. This proof is far oftener afforded by children in the first years than at a later age. When I recall the

numberless cases in which I have found miliary tubercles in the lung or pleura close beside caseous patches in the lung tissue, or those in which there were miliary tubercles of the pia mater in the immediate neighbourhood of caseous nodules in the brain, while at the same time both conditions were met with together in many other organs also—I cannot believe that there is any essential difference between them. Since R. Koch, the discoverer of the tubercle-bacillus, has proved the occurrence of this pathogenic element in both morbid products, I feel myself more than ever justified in including them both under a common description in the following account.

The symptoms of tuberculosis of the lungs in children who are past 6 or 7 years of age correspond so entirely with those of later life, that they call for no description here. We shall concern ourselves, therefore, mainly with the occurrence of the disease in the first years of life, during which we very often have an opportunity of observing it, especially in practice among the poor and in hospital. The younger the children are, the less as a rule does the clinical picture of the disease correspond to that of phthisis pulmonum in older people. For, the local affection remains more or less insignificant in comparison with the general disturbance of nutrition which presents the symptoms of atrophy already described (p. 73). On examining the bodies of little children who have died in a state of atrophy, I have very often found a large number of tubercles and caseous deposits in the lungs which had remained entirely latent during life. I have also found large cavities occupying the greater part of a lobe in a few children who were only some months old, and who had presented nothing during life but a progressive emaciation and debility and a slight cough; so that it was only the examination of the thorax that revealed the advanced destruction of tissue. The fact that the disturbance of the general nutrition is so much more prominent than the symptoms of local disease, is especially due to the fact that in very early childhood tuberculosis is generally much more widely distributed than is the case in later life. Caseous deposits and miliary tubercles are almost always present at the same time in a large number of organs—in the lymphatic glands, the spleen, the serous membranes, the liver, the kidneys, the bones, &c. Indeed cases occur in which scarcely a single

organ is found free from tubercular deposits. All these changes may have a more or less latent course. The main symptom is atrophy, steadily increasing from week to week, and this in many cases is combined with otorrhœa, eczematous eruptions on the head and other parts of the body, enlargement of the cervical, occipital and inguinal glands, often also with multiple (so-called cold) abscesses in the subcutaneous tissue. Since, however, these concomitant conditions occur by no means exclusively in tubercular atrophy, a careful examination of the thorax, even when the cough is entirely absent, is indispensable to establish a diagnosis.

This examination presents far greater difficulties in the phthisis of infants than in that of older children or adults. Sometimes we find nothing abnormal, except harsh breathing or catarrhal râles. All signs of consolidation may be absent, and we should not therefore be justified in diagnosing anything beyond a chronic bronchial catarrh, if it were not that atrophy, hereditary tendency, or enlarged glands, made us suspect that this catarrh was tubercular. In many cases, however, more extensive broncho-pneumonic patches occur, which under the influence of unfavourable conditions (*i.e.*, the presence of the tubercle-bacillus in the lung) caseate, and then present the ordinary physical signs of consolidation (dulness on percussion, indeterminate or weak breathing, prolonged and harsh expiration, bronchial breathing, bronchophony and sharp râles). In later life the development of phthisical processes in the lungs generally takes place from above downwards, and hence the limitation of the physical signs to the upper lobes and their apices gives us valuable criteria for the diagnosis of the early stages. In little children, however, we not uncommonly find an irregular distribution of the tubercles and caseous nodules through the whole of the lung tissue; and on examination of the supra-spinous and subclavicular regions we find but little, while the lower lobes on the other hand show signs of consolidation; or if these are absent, only catarrhal signs are found throughout. Irregular variations of temperature (which become less extensive as the child becomes more collapsed) and dyspeptic symptoms, anorexia and especially diarrhœa, are frequent complications, and are therefore all the more likely to mislead the physican. For since—as we have already seen—extensive tuberculosis of the lung and even cavities may exist

without any cough or marked dyspnœa, the diarrhœa is thus all the more likely to draw our attention away from the respiratory organs, and we are astonished to find at the post-mortem that the principal changes are in the lungs, while we had expected to find them in the intestinal canal. A few examples from very early childhood will illustrate to you what I have been saying.

Otto F., 4 months old, hand-fed. Since the 6th week of life, multiple abscesses over the whole body. For the last 9 weeks increasing atrophy, and flabbiness, little appetite, cough and short breathing. Percussion-note over the upper part of the chest on both sides, both in front and behind, less clear than in other regions. On the right side above, indeterminate breathing and bronchophony. Râles on both sides behind. P. 150, T. not elevated. At the beginning of the disease, fever was said to have been present. Father died from phthisis. Death after 8 days.

P.-M.—Extreme emaciation. Cervical and inguinal glands enlarged, some of them caseous. Partial adhesions of the pericardium to the heart and to the mediastinum; miliary tubercles on the visceral layers of the former. Left lung freely movable, containing numerous grey nodules the size of a pea. Right lung firmly adherent all over. In the upper lobe a cavity the size of a pigeon's egg, communicating with one still larger which ran backwards. Large and small tubercle-nodules scattered through the whole lung-tissue. A large caseous deposit in the lower lobe. Swelling and caseation of the tracheal and bronchial glands, one of which contained a cavity. Miliary tuberculosis of the liver and its serous covering. Spleen firmly adherent all over to the neighbouring parts, very large, tubercular both inside and out. A few small nodules under the capsules of the kidneys. Mesenteric glands partially caseous. In the ileum a few flat ulcers with small grey nodules in their edges.

Helene D., 8 months old. Increasing atrophy for 6 months, diarrhœa and coughing. For the last 8 days fever, especially in the morning hours. P. 144, R. 68. Noisy expiration, dyspnœa. Percussion note higher on the right side above both in front and behind, breathing very harsh all over, here and there mucous râles. Gradual increase of the dulness in the places mentioned, bronchial breathing and bronchophony. Œdema of the face and feet; collapse. Death after 3 weeks.

P.-M.—The right upper lobe firmly adherent to the chest wall, caseous almost throughout, and containing pretty large cavities communicating with one another, one of which reaches almost to the pleura. The middle and lower lobes, as well as the left lung, have miliary tubercles scattered through them. Bronchial glands

caseous, one of them softened in the centre. Extreme miliary tuberculosis of the spleen and peritoneum. Fatty degeneration of the liver.

The latency of widely-spread tuberculosis is especially noticeable in little children who finally die of tubercular meningitis. Without any marked prodromata, in the midst of apparent good health, or at most ushered in by some flabbiness of the skin or muscles which is easily overlooked and with some degree of emaciation—the meningitis suddenly appears. At the post-mortem the beginner is then surprised to find miliary tubercles and caseous deposits in many of the organs, although these had given rise to no symptoms whatever during life.

In older children—from 3 years old until about the time of the second dentition—we find tuberculosis not uncommonly beginning with dyspeptic symptoms. The children lose their appetite, the tongue is always more or less furred, they suffer often from diarrhœa, become emaciated, and complain of vague pains in the chest or abdomen long before the cough excites attention. At the same time they are ill-tempered, become feverish towards the evening, have dry lips and are restless during sleep. In the morning and forenoon, however, there is a remission, and nothing indicates the latent disease but a slight elevation of temperature and an unusually rapid pulse. Such cases are very apt to be treated as those of latent pleurisy are (p. 421), and the obscure symptoms—the gradual "falling off" of the children, as the mothers say—is referred to a protracted dyspeptic condition. Under these circumstances a careful examination of the chest cannot be too urgently recommended. The suspicion of incipient tuberculosis becomes more surely established if a hereditary tendency can be ascertained, if cough sets in, or if we can at the same time discover caseous or scrofulous deposits—*e.g.*, bone- and joint-suppuration, spinal caries, glandular enlargement and abscesses in the neck or in other parts of the body, chronic inflammation of the eyes, eruptions on the head, and otorrhœa. In any case, after a few months local lung symptoms also, cough, rapid breathing, &c., are sure to develope so distinctly, that one is forced to examine the lungs. His having hitherto neglected this examination may however have misled the physician into giving a favourable prognosis, for which the afflicted parents will be slow to forgive him. Even

although an early examination may reveal nothing very definite, still we may often make out chronic catarrh, and in such circumstances this may justify us in forewarning the family of the probability of danger. At this age (from 3 years upwards) we almost always find remittent fever (hectic) developing sooner or later, while in very young children we do not always find it, and it may be quite absent; as for example in the following cases.

Paul K., 1½ years old, treated in the hospital from 5th to 30th May. Extreme flabbiness and wasting, moderate cough, R. 50—60. Dulness on both sides at the base behind with sharp râles and indeterminate breathing; diarrhœa. During the whole time that the child was under observation, the temperature only rose once (on the evening of 10th May) to 100° F.; at other times it was always below this, and, in fact, generally sub-normal. At the post-mortem we found in both lungs many caseous deposits, a few cavities from the size of an almond to that of a plum, caseation of the bronchial and mesenteric glands and a few tubercular ulcers in the intestine.

Marie M., 7 months old, treated in the hospital from 16th January to 16th February. Continually increasing flabbiness and emaciation, constant cough and dyspnœa. On the right side very harsh indeterminate breathing and numerous large and medium crepitations not sharp in character. Dulness nowhere discoverable. Diarrhœa. During the whole time the temperature was seldom over 100·4° F., and was generally normal or sub-normal. On 14th February, fever began for first time (101·1° F., ev. 104·2). On the 15th the temp. was 102·7° F., and on the day of death only 100° F., the resp. 72; the limbs cold and covered with a bluish mottling. At the post-mortem we found the left lung quite healthy, while the right lung had a number of caseous nodules of different sizes scattered through almost its whole extent and contained in its apex one very large ragged cavity. Bronchial glands and spleen partly caseous.

This absence of fever scarcely ever occurs in older children. Even without using the thermometer we can at once recognise an exacerbation of the fever from the heat of the head and hands, the thirst and the increased feeling of malaise. The temperature rises to 102·2° F., and the remission is often ushered in by a slight perspiration which, however, is never so copious and regular as in the hectic fever of older patients. In many cases I have observed quite irregular temperature curves in which the morning temperature was often higher than the

evening. In a girl of 2 years at whose post-mortem we found miliary tubercles and extensive caseous processes in both lower lobes, we had the following temperature chart:—

	M.	E.
22nd August	100·0	103·1
*23rd ,,	104·7	99·7
*24th ,,	101·8	100·2
25th ,,	100·0	100·9
26th ,,	100·2	101·1
*27th ,,	103·3	101·1
28th ,,	98·6	105·3
*29th ,,	103·6	103·1
30th ,,	101·1	104·0
*31st ,,	103·1	101·3 &c.

On the days marked * the morning temperature was the higher.

The diagnosis of this disease in children is further rendered difficult up to a certain age by the absence of sputa, which in adults furnish a valuable point for the diagnosis owing to the discovery of elastic fibres, and especially tubercle-bacilli. The cases in which there really is some expectoration are all the more worthy of note. This takes place more by a process of retching or by the help of the mother, who draws out the expectorated matter with her fingers. Among others I have seen one boy, only seven months old, with extensive caseous degeneration and cavity-formation in the left upper lobe, who for months brought up a very large amount of greyish-yellow fœtid sputum, which occasionally contained elastic fibres but never blood. Hæmoptysis in children (apart from that which occurs as the result of tracheotomy) is on the whole a very rare phenomenon before the age of the second dentition, although I cannot confirm the statement of Rilliet and Barthez that they have never observed blood-spitting before the 6th year. I have met with at least a dozen phthisical children under 5, who on violent coughing brought up small quantities of blood, and occasionally even as much as a teaspoonful, either pure or mixed with mucus and pus. I have only on two occasions seen a copious hæmoptysis at this age. In one of the cases this was explained by the post-mortem:—

> On 29th December, 1884, a pale, wasted little girl of 10 months was admitted into the hospital. Said to have had measles and inflammation of the lung a few months before and to have wasted ever since, but to have coughed but little. The relatives

say that during the last few weeks she has vomited blood on two occasions, once a small quantity, the second time a large amount (filling a small bowl). The motions were still of a tarry black colour. There was slight impairment under the left clavicle; here and at other places on the thorax numerous crepitations were heard. Very marked anæmia and incipient rickets. In the night between the 5th and 6th January, 1885, there was a fresh discharge of blood from the mouth and nose, during which death took place.

P.-M.—Left lung firmly adherent to the costal pleura. In the middle of the upper lobe, which was much consolidated and partly caseous, there was a cavity about the size of a walnut, which communicated with a bronchus, and, besides some bloody caseous pulp, contained a roundish tumour (¾ inches in diameter). This proved to be a thin-walled aneurism, filled with parietal thrombi, and connected with a branch of the pulmonary artery.[1]

There are in pædiatric literature a few quite similar cases of aneurism of a branch of the pulmonary artery in the middle of a cavity, ending in rupture and very copious hæmoptysis.[2] On the other hand I have never myself met with a case in which the compression or perforation of a branch of the pulmonary artery or vein by caseous bronchial glands at the same time communicating with a bronchus had occasioned a copious hæmoptysis, although such an occurrence has occasionally been observed by other writers.

I shall take this opportunity of saying a few words about the great tendency of the tracheal and bronchial glands, especially the latter, to become enlarged and to caseate. If tubercle or caseous processes occur anywhere in a child's body, we may almost certainly count upon finding the above-mentioned glands similarly affected. In fact, out of innumerable post-mortems of tubercular children, I can recollect only a few exceptions to this rule; and this proves that the tendency of these glands to enlargement and caseation in children is even greater than that of the lungs. While Louis has seen the lungs remain unaffected only once in a series of 123 tubercular adults, Rilliet and Barthez on the other hand have found them perfectly unaffected in 47 out of 312 tubercular children. I think that the extreme frequency of glandular

[1] *Cf.* the Dissertation of my pupil Dr. Hoffnung, *Ueber Hämoptoë bei Kindern*: Berlin, 1885.

[2] Wyss, *Gerhardt's Handb. der Kinderkrankh.*, Th. iii., 2, S. 807.—Rasmussen. *Hirsch-Virchow's Bericht*, 1869, ii., 101.—West, *Lectures*, &c., vii. edition, p. 530.

enlargement may be referred to two circumstances: firstly, to the peculiar general predisposition which many children have to glandular enlargement, which we are accustomed to designate the "scrofulous" diathesis; and secondly, to the fact that bronchial catarrh and whooping cough are so very common. The irritation of the mucous membrane is transmitted by the lymphatics to the neighbouring bronchial glands just as in intestinal catarrh, typhoid fever, &c., it is carried to the mesenteric glands. The glandular affection very often forms the chief disease in children, while the lungs themselves may contain but few tubercles and deposits. We find the bifurcation of the trachea and the large bronchi surrounded by glands either separate or conglomerated, sometimes gathered into masses of the size of a hen's egg. Some of these are simply hypertrophied, vascular, greyish-red, but generally either some or all are tubercular or transformed into a whitish-yellow mass. Also, on cutting into the lungs we frequently find little caseous glands at the bifurcations of the medium-sized bronchi. A few of the glands show on section a cavity filled with softened debris, situated either centrally or towards the periphery, which, after they become adherent to the pulmonary pleura or to the bronchi, ruptures into an adjacent lung-cavity or even into one of the large bronchi. When this occurs, fatal suffocation may result from fragments of caseous matter finding their way into the upper air passages.[1] Even the rupture of such a gland-cavity into the pericardium causing fatal pericarditis, has been observed in a few cases. Large bunches of glands at the root of the lungs may even compress the adjacent vessels more or less, especially the pulmonary artery and vein, and their branches, the superior vena cava, and the common jugular vein, the vagus and its branches. The latter, especially, we occasionally find so surrounded and flattened by the glands that it is scarcely possible to follow its course through the mass. Adhesion of some of the glands to the œsophagus, to the pulmonary artery or a branch of it has likewise been observed, by which these parts are not only displaced, but, owing to the pressure, may be gradually thinned and eventually perforated.

[1] Frühwald, *Jahrb. f. Kinderheilk.*, Bd. xxiii., S. 423.—Petersen, *Deutsche med. Wochenschr.*, 10, 1885. Successful treatment of such a case by tracheotomy. —Loeb, *Jahrb. f. Kinderheilk.*, Bd. xxiv., 1886, S. 353.

Can we, then, diagnose this condition of the bronchial glands during life by any definite symptoms? As far as my experience goes, I must answer this question in the negative for the great majority of cases. Certainly we will scarcely ever be mistaken if in a tubercular child we diagnose caseation of the bronchial glands before the post-mortem; but this is only because this condition is almost never absent in these cases. The clinical descriptions which authors give of glandular enlargement have the look of having originated in the study and not at the bedside. It is said that the compression exerted by the glands on the neighbouring parts might readily give rise to pressure-symptoms; and in fact cases do occur in which œdema of the face and dilatation of one or both jugular veins in the neck take place, and likewise hæmoptysis and hæmorrhagic infarction of the lung from pressure on the pulmonary veins. I have myself, in a little girl of $1\frac{1}{2}$ years, observed compression of the right bronchus by a mass of tubercular glands the size of a hen's egg, whereby the entrance of air into the right lung was considerably interfered with, and the breath-sounds on this side could only be heard extremely faintly. The compression of the vagus and recurrens also may, as I have frequently noticed,[1] cause certain nervous symptoms, especially alteration of the voice (hoarseness), fits of spasmodic cough with inspirations like those of whooping-cough, also asthmatic attacks with whistling breathing and cyanotic discolouration of the face. According to my experience I must, however, regard such cases as extremely rare. We have often at post-mortems found large masses of caseous bronchial glands, the presence of which had not been revealed during life by a single symptom, the children having presented nothing beyond the well-known features of tubercular meningitis or phthisis. Even the distension of the external jugular veins, to which so much importance has been attached, and the œdema of the face, may occur merely as the result of engorgement of the right side of the heart from extensive consolidation of the lungs, without there necessarily being any compression of the large venous trunks within the thorax. On this account I regard the diagnosis of enlarged glands during life as very problematical. I would, however, attach least value of all to the dulness of the percussion-note over the inter-scapular region, which many

[1] Romberg and Henoch, *Klinische Ergebnisse:* Berlin, 1846, S. 165.

insist upon. I, at least, have never yet seen a glandular tumour so large that it could have caused a well-marked dulness in this locality. Rilliet and Barthez point out also that large masses of glands in the posterior mediastinum act as good conductors of sound, and intensify to the ear of one who is auscultating the back any sound heard from the lungs, and that on this account we may hear loud bronchial breathing and sharp râles without the lung itself being consolidated or containing cavities. I have not myself as yet met with an error of this kind due to the presence of masses of glands. At any rate percussion would soon clear this up; for where these sounds were really caused by consolidation of the lungs and by cavities, distinct impairment of the note at the back would scarcely fail to be present. Therefore I cannot admit that there is a quite definite independent series of symptoms indicating enlargement and caseation of the bronchial glands. In most cases the condition can only be suspected, and is therefore merely of pathological interest. Only in exceptional cases can we make a diagnosis with any degree of probability when there are distinct symptoms of pressure on the veins or on the vagus nerve.

The tuberculosis of children up to the beginning of the second dentition is distinguished from that in later life by its acute course. Cases which are very chronic and protracted, lasting for years, are extremely rare, and the fatal termination almost always occurs within some months or at most within about a year. This of course is to be accounted for by the wide distribution of the tuberculosis throughout many organs in childhood. In children, also, much oftener than in adults, we have tubercular meningitis, broncho-pneumonia or pleurisy developing, which bring life to an end sooner than would otherwise have been the case. The pleura is affected, indeed, in tuberculosis almost as often as the pia mater, the disease either taking the form of numerous miliary nodules scattered over the costal and pulmonary pleura, or of large caseous patches on the free surface of the membrane, or in the subserous connective tissue under the costal layer. In the latter case we occasionally see little extra-pleural cavities resulting from the breaking down of these patches, which may either rupture into the pleural cavity or after previous adhesion of the pleura to the lung may empty themselves into cavities in the latter or into the bronchi. More or less

extensive adhesions of the two layers of the pleura to one another likewise occur very often, while in other cases we have the development of sub-acute or chronic pleurisy with copious purulent effusion often blood-stained. The same may be said of the pericardium, the partial or complete adhesion of the two layers of which I have met with, not uncommonly, in tubercular children. I shall enter into this more fully later on. The fatal course of the disease is accelerated in many cases by the rapid development of acute miliary tuberculosis, the symptoms of which are here pretty much the same as in older patients. The acute eruption of miliary tubercles in a more or less large number of tissues may, however, take place not only during the course of pulmonary tuberculosis which has hitherto been chronic and constitute its fatal termination, but it may also occur in children who are apparently perfectly healthy, and are not at all suspected of a tubercular tendency. In both cases great and sudden variations of temperature with irregular exacerbations (occurring sometimes in the morning, sometimes at noon, and sometimes in the evening), very rapid superficial breathing and harsh breath sounds, to which, usually, widely-distributed fine crepitations are by-and-by added—form the chief symptoms; and in the further course of the disease we may also have enlargement of the spleen, roseola, and cerebral symptoms. The fever, however, does not always reach a very high degree. Thus in a child of 2 years who had hitherto been quite healthy I found during two weeks a temperature of only 100·8°—102·2° F. while the rate of respiration was from 60 to 80, although nothing abnormal could be discovered on examining the lungs. It was only in the beginning of the 3rd week, when convulsions, hemiparesis, and coma suddenly set in, that the temperature rose to 104° F.; and death took place 2 days after. In the diagnosis we may easily be misled by the cerebral symptoms into thinking either of typhoid fever or of tubercular meningitis; the former especially if there is enlarged spleen and roseola.

Wilhelm K., 3 years old, brought to my polyclinic on 15th March with traces of scarlatinal desquamation, presenting the symptoms of pleuro-pneumonia of the right lower lobe. During the next few days the pleurisy became more prominent. By the 30th April, however, it was quite gone, so that there was nothing left but an impaired note laterally and a very slight dulness

behind; vesicular breathing was heard all over. On the 6th August—that is, 3 months after—the child, who had during the interval remained well, was brought again to the hospital. During the last 5 days, headache, vomiting, and constipation. P. 92; T. somewhat elevated. The physical signs unchanged. Persistent constipation, in spite of repeated doses of calomel and syrupus rhamni and enemata. On the 8th, frequent vomiting; pulse, 132. On 15th, the spleen not enlarged. Nothing new to be discovered in the chest. Pupils reacted sluggishly; drowsiness, out of which the boy could not be wakened. Abdomen somewhat retracted. Yesterday afternoon an epileptiform fit, lasting 3 hours. During the next few days, increasing coma, frequent perspiration, left pupil wider than right. R. 48 unequal; P. 128. On 21st, permanent convulsions and contractures. Death in the course of the following night.

The nature of the symptoms and their succession during the 3 weeks' course of the disease were here so characteristic that the diagnosis of tubercular meningitis seemed to me beyond a doubt; and what did we find at the post-mortem?

Pia mater hyperæmic, otherwise quite normal; no trace of inflammation or tubercles in it; much serum in the dilated ventricles, central parts generally macerated (post-mortem appearance). Bronchial glands enlarged and caseous, right lung completely adherent, pleura costalis much thickened, scattered over with grey intra-pleural nodules lying together like stones on a causeway. The anterior lower border of the lung caseous, the posterior portion brown and carnified. Left lung sprinkled throughout with innumerable miliary nodules. Liver fatty. Spleen full of miliary tubercles. In the intestine a few small tubercular ulcers.

Max R., 1½ years old, admitted into the ward on 31st March. Coma, dry crusted lips, both pupils contracted, R. irregular, interrupted by pauses. Percussion normal, harsh breathing all over the chest, with coarse crepitations. Abdomen distended and apparently tender on pressure. Constipation. Pulse very small, 144. T. 85·5°; towards evening, 101·3° F. The same condition on the 2 following days. On 3rd April, the day of death, T. suddenly rose to 105·4, R. 76, P. imperceptible. Cyanosis, trismus, rigidity of the neck and of all the limbs. Death at 8 P.M.

P.-M.—Pia mater in a state of venous hyperæmia and œdema. No exudation or tubercle anywhere. Brain very vascular, ventricles (especially the 4th) filled with a moderate amount of clear serum. Very abundant miliary tuberculosis of the pleura and of both lungs, of the spleen and liver. Bronchial and mesenteric glands enlarged and caseous, likewise the intestinal follicles.

In both these cases, then, we found neither tubercle nor

exudation in the pia mater, and yet during life the characteristic symptoms of tubercular meningitis were present; and in the second case there was also the rise of temperature immediately before death of which we have spoken (p. 325). At the post-mortem we only found hyperæmia, and, in the second case, also œdema of the pia mater and accumulation of fluid in the ventricles, that is, hydrocephalus acutus (p. 309), to which we could ascribe the cerebral symptoms. I have seen one other quite similar case, that of a child of 9 months, who, during the last few days, presented a tetanic muscular rigidity, so that one could raise the child either by the head or feet and hold it almost horizontally. In this case we found at the post-mortem only œdema of the pia mater, and extreme internal hydrocephalus, although there was miliary tuberculosis of the pleura, lungs, spleen, and liver, and caseation of the bronchial glands. I have found the same appearances in two other cases of miliary tuberculosis which had assumed a typhoid form at their onset—in the case of one child of 3 especially the temperature-curve corresponded so exactly to that of typhoid fever that I adhered to this diagnosis until the post-mortem, at which we found extensive acute miliary tuberculosis instead of the expected appearance of typhoid. In both cases the cranial cavity was entirely free from tuberculosis, and there was nothing found but hyperæmia of the pia mater with serous distension of the ventricles.

I have only exceptionally seen a hæmorrhagic diathesis resulting from acute miliary tuberculosis.[1]

Otto K., 4 years old, admitted 8th December, 1879. History obscure. Took ill on 26th November with violent fever and hæmorrhages from the mouth and nose, which, with short intermissions, had lasted ever since. A hæmorrhagic diathesis had never been observed before. Pale, emaciated child, much collapsed. Sclerotic and skin slightly jaundiced. Cutaneous veins markedly distended. Slight branny desquamation of the epidermis. Scrotum œdematous. T. 101·7° F.; R. 40, superficial, costo-abdominal. On examination nothing found but coarse crepitations at the back; P. 156, small. Flatulent distension of abdomen, liver extending about 2 inches below the margin of the ribs, spleen not to be made out. Motions thin, very black, passed involuntarily. Urine removed with the catheter (7 oz.), brownish-red, acid, containing some albumen, no tube-casts, no whole blood corpuscles

[1] Jacubasch, *Jahrb. f. Kinderheilk.*, xv., S. 167.

(hæmoglobinuria). Death in a state of collapse on 10th December. After a few injections of camphor, the punctures bled long and severely.

P.-M.—Pericardium presented a few submiliary nodules, heart-muscle slightly fatty, a few submiliary tubercles close under the aortic orifice. An enormous quantity of the same on both lungs, on the pleura, in the spleen (which was enlarged to three times its usual size), in the kidneys, on the capsule and in the substance of the liver, which was much enlarged and fatty. Some of the bronchial glands the size of a walnut and caseous, thoracic duct free from tubercle.

I must leave it undecided whether the hæmorrhages from the mouth and nose observed in this case as well as the hæmoglobinuria are really to be ascribed to the acute miliary tuberculosis. Further observations will decide this. With the exception of the above, I have never met with a case of this kind, and Jacubasch has searched medical literature in vain for another like it. I have indeed observed one case of a boy who died of general miliary tuberculosis and tubercular meningitis, and who during the last few weeks presented numerous purpuric spots, especially on the lower limbs. There were, however, no hæmorrhages from the mucous membranes at all.

Occasionally acute miliary tuberculosis develops in a succession of distinct attacks, each of which is accompanied by more or less high temperature, while the intervals between are entirely free from fever. The following case is a characteristic example of this rare form.

Hermann K., 6 years old, admitted to the hospital on 2nd February, 1878. Utterly neglected, and affected with chronic eczema. Some cough, without abnormal physical signs. Improvement after malt baths. From 13th—28th, diarrhœa, which was cured by bismuth. subnit. and argent. nit. After seeming quite well, he suddenly, on 6th March, had anorexia and fever (T. 105·6° F.; P. 134; R. 44, very superficial). In the lungs nothing but harsh breathing all over, percussion normal. The fever lasted unabated for 4 days, during which time there were twice rigors in the morning. T. only on the morning of the 8th, 98·6° F.; at other times always 106·2° F. R. rose to 64, without any other abnormal symptoms. From 11th March to 8th May—*i.e.* almost 2 months—free from fever (only on 4 days did the evening T. rise to 100·4°—101·5° F., at other times it was either normal, or even subnormal). Nothing found on examination. General health good, strength increasing. Suddenly, on 8th May, a fresh exacerbation

of fever (105·4° F.), lasting 2 days (never under 104° F.), with P. 144—160, and R. 60. From this time catarrhal sounds appeared in both lungs, and the rate of perspiration remained 40—50. From 10th—13th the T. fell again gradually, and remained quite normal till the 25th, while the catarrh and the rapid breathing persisted, and the abdomen became distended with flatulence. From the 25th there was again an exacerbation of fever (102·9°—104° F.), lasting 5 days. After a few days free from fever, there began on 1st June a remittent type of temperature (m. 100·8°; ev. 102·6°—103·8° F.), which lasted without interruption until the day of death (5th July), with continual quick P. and R., increasing emaciation and weakness, persistent bronchial catarrh, and continually-recurring diarrhœa. Finally collapse, œdema of the hands and feet, slight dulness on the right side over the base behind, bronchial breathing and sharp râles, extreme dyspnœa. Death on 5th July.

At the post-mortem we found extensive pleuritic adhesions, extreme miliary tuberculosis of the pleura, of both lungs, of the whole peritoneum, of the spleen, liver, and both kidneys. Caseous consolidation of the base of the right lower lobe, caseation of the bronchial and mesenteric glands.

This case, then, shows that apparently inexplicable feverish attacks lasting for several days with very high temperature, must arouse suspicion of incipient miliary tuberculosis, even although they are separated from one another by weeks of complete apyrexia, and although on examining the lungs we find nothing but harsh breathing and catarrhal sounds. We must, of course, assume in these cases that there is an invasion of tubercle-bacilli advancing in successive attacks—probably starting from caseous bronchial or mesenteric glands.

Unfortunately there is but little to say on the treatment of tuberculosis in the first years of life. I cannot record real success in the treatment of a single case presenting the signs of tuberculosis or of far-advanced pulmonary phthisis, although cases of the "chronic pneumonia," formerly mentioned, not uncommonly completely recover. I would refer you to the treatment I recommended for the latter (p. 418), which will here also fulfil all the indications. As regards prophylaxis I must especially draw your attention to milk from tubercular wet-nurses, or from cows with "perlsucht" (unless the milk has been boiled), as the identity of "perlsucht" with tuberculosis has been established by Koch's researches. There are plenty of cases published of infection as well as of artificial

production of tuberculosis by inoculation with the milk of cows with "perlsucht" (Bollinger, May, Demme[1]). The danger, however, is not so very great, because according to the investigations into the milk which have been made as yet, it is only infectious when the mammary glands themselves contain the "perlsucht" nodules, which does not often happen. Abelin[2] says he has observed a small epidemic in the Stockholm "Children's House" resulting from infection. Since we have become acquainted with the tubercle-bacillus, the fact of the disease being contagious has become conceivable, and several of the cases recorded in pædiatric literature of children being infected by tubercular wet-nurses or attendants, by tubercular operators having sucked the wound of circumcision, &c., deserve attention. I have not myself as yet met with a single certain case of this kind.

X. *Gangrene of the Lungs.*

Gangrene of the lungs in children differs clinically from the same disease in adults only in this, that the diagnosis is more difficult on account of the frequent absence of sputa. We must also add that less importance is to be attached to a gangrenous smell of the breath in children, because in them necrotic processes in the mouth and throat are often present at the same time, and these, quite as readily as pulmonary gangrene, may give rise to this symptom. This disease, moreover, seems to occur oftener in children than in adults. It appears as the termination of a croupous pneumonia only in rare cases when the disease ends with the formation of "sequestra," and these become infected by putrefactive germs which have entered along with the air-current. It is in this way that we must regard the cases given on p. 427, which ended in putrid pleurisy. Gangrene of the lung arises oftener as the result of embolism, septic matters which have been formed in different parts of the body finding their way into the lungs through the circulation, and there infecting already-existing

[1] *Jahresber. d. Jenner'schen Kinderspitals*, 1879, S. 27; 1882, S. 48; 1886, S. 21. See also Stein, *Experim. Beitr. zur Infectiosität der Milch perls. Kühen*: Berlin, 1884.—Absolutely negative results were obtained in the feeding experiments of Imbach, *Jahrb. f. Kinderheilk.*, xxiv., S. 292.

[2] *Archiv f. Kinderheilk.*, iv. 1.

broncho-pneumonic patches. Thus, *e.g.* I have observed patches of pulmonary gangrene resulting from caries of both petrous bones with offensive discharge, and often from gangrenous processes in the skin, such as are not uncommon among the ill-cared-for children of the poor, especially after infectious diseases (measles, scarlet fever, typhoid), also after gangrene of the vulva and of the cheeks. I found a patch of gangrene in the lung of a child of 2½, who had suffered for many weeks from extensive ecthyma cachecticum, resulting in gangrene of the skin.

> Chest and back so penetrated by deep, gangrenous ulcers, covered with black necrosed fragments, that physical examination of the thorax was out of the question. After death, which took place in a state of collapse (T. 96·1° F.), we found at the post-mortem (6th May, 1879) the following changes in the respiratory apparatus:—Chronic sero-fibrinous pleurisy, multiple broncho-pneumonia, especially on the left side; many offensive, embolic abscesses and hæmorrhagic infarcts in both lungs. Circumscribed patch of gangrene in the left lower lobe, partial thrombosis in the course of the pulmonary artery. On account of the impossibility of examining the thorax and the prominence of the cutaneous and general symptoms, all these affections had remained latent during life.

Gangrene of the lung also arises from direct inhalation of septic matters; for example, in pneumonia under the above-mentioned circumstances, likewise in cases of tubercular cavities and abscesses of the lungs, noma, and diphtheria of the pharynx. I have frequently observed putrid bronchitis both in scarlatinal necrosis of the pharynx and in true diphtheria, and in the latter I have several times seen a number of gangrenous cavities the size of a pigeon's egg in the centre of a patch of broncho-pneumonic consolidation. In these cases also the disease was not discovered until the post-mortem, for the gangrenous odour of the breath during life was of course referred to the necrosis of the pharynx. On the other hand, in the case of a phthisical boy of 4 years, at whose post-mortem we found several gangrenous cavities of various sizes in a consolidated left lung, and putrid pleurisy, I was able to make the diagnosis during life owing to the extremely offensive smell of his breath, especially when he coughed. At any rate, the extreme general weakness, which we find in all exhausting diseases, is an im-

portant factor in the causation of pulmonary gangrene, owing to the retardation of the blood-current and the tendency to thrombosis. In a sickly little boy of 2 years, who was treated in my ward for general eczema, an attack of broncho-pneumonia took place; at first it caused no anxiety, but after about a fortnight it ended suddenly in extreme collapse, with deathly pallor of the skin, and such a fœtid odour of the breath that the ward was regularly poisoned. At the post-mortem we found a gangrenous patch almost the size of a hen's egg in the right lower lobe surrounded by consolidated tissue. To this class also belong the cases of pulmonary gangrene which result from severe typhoid fever, and of which I shall by-and-by give you two examples. At the same time we must not forget that under these very circumstances the entrance of food into the air-passages may favour the occurrence of a septic destruction of the inflammatory patch.

The origin of the pulmonary gangrene in the following case remained unexplained.

Albert St., 11 years old, admitted 23rd June, 1881. Formerly healthy. Had taken ill suddenly 10 days before with a rigor, followed by fever; afterwards, frequent loose motions and delirium. When admitted, the boy, who was otherwise strong and of a good complexion, lay in a state of profound drowsiness, and could only be roused for a few moments at a time. On examination of the chest, we found dulness and occasional sharp râles below the spine of the scapula on the right side. No enlargement of the spleen nor roseola. T. 103·1° F.; P. 120; R. 40. In the evening the T. rose to 104·4° F., the pulse 148. During the following night active delirium and three attacks of rigor, with cyanosis, which (as we afterwards learned) had also occurred frequently previous to his admission. On 24th, general collapse, cyanosis of the extremities. Vomiting. T. 104° F.; P. 160; R. 52. Extreme dyspnœa in the course of the following night. Death towards morning.

P.-M.—The right side of the diaphragm arched downwards into the abdominal cavity. On opening the right pleural cavity, a quantity of foul-smelling gas escaped. The pleural cavity formed an empty sac, against the median wall of which the lung lay quite collapsed, and of a dirty greyish-green colour. The costal pleura covered with offensive discharge. In the pleural cavity about 7 oz. of greenish-grey offensive matter. In the right lower lobe there was a spot $1\frac{5}{8}$ inches long by $1\frac{3}{4}$ broad, which could be recognised even externally as a patch of gangrene, and which presents an elongated perforation through the extremely thin pleura. The lower lobe was hepatised to a slight extent, and at the

base contains a few other gangrenous patches the size of cherry-stones underneath a thin fluctuating pleura. At the apex of the left upper lobe likewise a patch of gangrene the size of a walnut, the rest of the lung tissue vascular and air-containing. Spleen considerably enlarged (3½ in. long, 1¾ in. broad, 1¼ in. thick), bluish-red, soft. In the intestine copious epithelial coating, Peyer's patches in places somewhat swollen, mesenteric glands slightly enlarged. All other organs normal.

Although there was neither enlargement of the spleen nor roseola, still the whole aspect of the case seemed to justify the diagnosis of typhoid fever with broncho-pneumonia of the right lower lobe. The very slight changes that were found at the post-mortem in the Peyer's patches and mesenteric glands are not, indeed, of much weight against this diagnosis; since, as we shall see later on, cases of this kind do occur in the typhoid of children, and in them the spleen is found much enlarged. Nevertheless, I would not in this case regard the gangrene of the lung as typhoid, because, for one thing, the disease had only lasted 10 days, but especially because the multiple form of the gangrene and the repeated rigors pointed to a septicæmic source, although, to be sure, no such source was found at the post-mortem. Who knows whether there may not have existed a septic patch somewhere in the osseous system, from which the embolic processes had started. The fatal issue in this case occurred through rupture of a superficial patch of gangrene of the lung, followed by putrid pyo-pneumo-thorax. I would especially point out that in this case there was not the slightest suspicious smell in the breath.

XI. *Whooping Cough.*

Although I close my account of the Respiratory Diseases with a description of whooping cough (tussis convulsiva, pertussis) I am perfectly well aware that it does not really belong to this section, but indubitably to that of the Infectious Diseases. Nevertheless I consider it expedient from a clinical point of view to treat of whooping cough immediately after the diseases of the respiratory organs, because its symptoms and its most serious complications belong mainly to this system.

There are certain signs which may enable the physician to

diagnose pertussis even before he has heard the child cough; especially the parents' statement that the child suffers from a cough coming on in fits and especially frequent during the night, and that it is accompanied by crowing and dark redness of the face, and ends with retching and vomiting of mucus. The suspicion that we have to do with whooping cough is strengthened if we find that the child's face looks puffy, especially about the lower eyelids, and if the veins of the latter are dilated.

We distinguished three stages in the course of the disease, which pass imperceptibly into one another. The first (catarrhal stage) differs as a rule in no way from ordinary tracheal or bronchial catarrh, and therefore does not excite any suspicion of its being the forerunner of whooping cough, unless there happen to be an epidemic of that disease or if children of the same family are already suffering from it. Less frequently, the cough has a peculiar character even at this period—a more paroxysmal onset with inclination to retch towards the end—which may lead us to suspect incipient whooping cough. The catarrhal stage is in these cases extremely short, being limited to a few days, and little children in the first years of life are, it seems to me, especially liable to this peculiarity. In general, however, the first stage lasts 10—12 days. During this time the cough which was at first purely catarrhal, gradually assumes a paroxysmal character. As many authors—*e.g.*, Lombard and West—say that they have seen this stage lasting 5—6 weeks, I shall not dispute it; but I cannot help thinking that in these cases there was more probably an ordinary catarrh and that the children became affected with whooping cough during its course. In children who have a tendency to false croup (p. 358) I have occasionally seen the first stage of whooping cough begin with an attack of that disease, followed by catarrh which passed into whooping cough.

The second stage (convulsive) presents the acme of the disease. The characteristic paroxysms now occur with more or less frequency, being most severe and frequent during the night. It is these that have given their name to the disease, from the intermittent crowing ("whooping") inspiration.

Often, but by no means always, the individual attack begins with a kind of aura, *i.e.*, with preliminary symptoms by which the child and those around it are made aware of the approach of

an attack. The child suddenly becomes restless, anxious, leaves off eating or playing, sits up quickly if it has been lying down, and clings to its mother or to any fixed object as if it were thus better able to meet the approaching attack. Even in an infant at the breast of 3 weeks I have observed an anxious beating about with the arms before every attack and sometimes also short whistling inspirations; in a boy of 14 weeks hasty evacuation of urine and fæces and in some older children vomiting ushered in the attack. The latter ran hurriedly into the corner of the room and emptied their stomachs, and then the paroxysm immediately followed. In a child of 2 years the attack began with restlessness and innumerable sneezes following rapidly on one another; and these also recurred towards the end. On the other hand a girl of 9 years had for aura much quickened breathing with dyspnœa and noisy expiration; which condition lasted over an hour before the paroxysm commenced. Immediately after the attack and in the intervals the breathing was perfectly quiet and only here and there was a slight râle audible. The attack itself consists of coughs following rapidly on one another and interrupted from time to time by crowing inspiration. The child bends forward while the attack lasts. The quicker the coughs follow on one another (*i.e.*, the fewer the inspirations) the more does the child present the aspect of suffocation—a dark somewhat bluish redness of the face and neck, great distension of the cutaneous veins, and cyanosis of the visible mucous membranes, especially of the tongue. Tears in the eyes, trickling of blood and mucus from the nose, ecchymoses under the conjunctiva and in the subcutaneous connective tissue of the face—are frequent accompaniments and results. The action of the muscles of respiration is considerably increased, especially that of the abdominal muscles and of the hard arched sterno-mastoids. It is only during the crowing inspiration that a momentary abatement of the above-named symptoms takes place, and this is followed at once by an aggravation when a new fit of coughing begins. Thus the succession of suffocative coughs and hurried inspirations is repeated 3—6 times, or even oftener, and after lasting 2—3 minutes the attack ends either without, or oftener with, the bringing up of pure or blood-stained bronchial mucus and fragments of food, which the mother tries to help out by putting her finger into the mouth. One almost always observes a second less severe

fit of coughing after quite a short pause; and this may even be followed by a third, so that the whole paroxysm really consists of 2 or 3 successive seizures. Then at last, for the first time, complete rest follows. While many, and especially little children, lie in a state of complete exhaustion after the attack, the older ones go on with their occupations almost immediately as if nothing had happened. The slight influence of the frequent nocturnal attacks is especially remarkable. The children jump up, go through the attack and then at once fall asleep again, without appearing much put out by the frequent interruption of their night's rest. If one examines the chest during the paroxysm one cannot hear the vesicular breathing even during the crowing inspiration, because this conceals all the rest, and the air cannot enter the bronchi in the usual way.

The number of the attacks in the 24 hours varies greatly. While many children during the whole course of the disease never have more than 10 or 12 in the day, others have as many as 30—60. But in these, generally, are included of course the various phases and component parts which as above mentioned form one complete paroxysm. You will understand that the danger of the disease must increase with the number of the paroxysms, partly from the exhaustion which becomes more and more marked, and partly from the repeated venous engorgement which accompanies each attack and may be of serious significance. Trousseau is therefore right in his advice to note down the number of attacks on a slate so as to be able to estimate their increase and decrease and, thereby, the danger of the disease. Although the attacks generally come on spontaneously, still they are easily excited by emotional causes (crying and screaming), by change from the prone to the upright position, and sometimes also by distension of the stomach. I can usually succeed in exciting a paroxysm for the purposes of clinical demonstration either by pressure on the larynx or by examining the pharynx. It is remarkable that if there are a number of such children together (*e.g.*, in the waiting-room of the polyclinic) the attack of one is very apt to be answered by the others, and a general coughing ensues.

The intervals between the attacks are in simple whooping cough entirely free from morbid symptoms. There is no cough whatever, the respiration is quiet, and on examination we find

either normal breath-sounds or at most a few catarrhal rhonchi. We may diagnose the disease from the already-mentioned slight œdematous swelling of the eyelids, and from the dilatation of the small veins round about the eyelids which, after the disease has lasted for some time, are apt to arise owing to the constantly recurring engorgement. From the same source arise the attacks of epistaxis which are sometimes exhausting, the bloody sputa (bronchial hæmorrhage), and the ecchymoses under the conjunctivæ. The latter usually occur only in spots, but they may attain a considerable size, and I have seen the whole cornea surrounded by an effusion of blood, quite covering the sclerotic, the conjunctiva palpebrarum suffused with blood, and both eyelids of a blackish-blue colour. The pressure of the venous engorgement which occurs during the attacks may, however, show itself in other ways also. Any erectile tumours enlarge. When stomatitis is present, hæmorrhage may occur from the inflamed gums. In one child who had eczema of the ear, I saw bleeding take place from the affected area of skin during every severe attack. Hæmorrhage also occasionally occurs from the outer ear, and this is accounted for by a rupture of the tympanum, which (especially when otitis externa is present) is apt to be caused by the force of the air—which is much compressed during the cough—being driven through the Eustachian tube into the tympanic cavity. These ruptures, however, almost always recover without leaving any traces, and cases of suppuration of the tympanum resulting from such an occurrence are very exceptional. Barrier observed a hæmorrhage between the dura mater and arachnoid as the result of a paroxysm; and I have already (p. 269) mentioned a case of hemiplegia which occurred during an attack of whooping cough, and which must without doubt be referred to hæmorrhage into the brain.[1] From the violence of the forcible expiration, hernia and prolapsus ani not unfrequently occur. Indeed Cadet[2] described a case of rupture of the rectus abdominis with the formation of a large tumour (hæmatoma) under the skin of the abdomen, which underwent gradual resolution.

In very many children who have suffered from whooping cough

[1] See a similar case of hemiplegia and aphasia in the *Jahrb. f. Kinderheilk.*, 1876, x. S. 400.—On blindness occurring after whooping cough—which I have never met with myself—see Alexander, *Deutsche med. Wochenschr.*, 1888, No. 11.

[2] *Loc. cit.*, ii., p. 306.

for some length of time we observe a whitish grey erosion or deeper ulceration of the frænulum linguæ, which may cause a partial or complete destruction of it. The fact that this ulcer, with very few exceptions, only occurs in those who already possess incisor teeth, proves that it is due to the constantly repeated friction which the frænum suffers during the attack owing to the tongue being rapidly shot out over the lower median incisors. From a like cause I have several times seen the ulceration also on the lower surface of the tip of the tongue in the neighbourhood of the frænum, and even on the dorsum of the tongue, in which case the lesion was to be referred to the lower lateral or to the upper incisors. The ulcer, however, is by no means always present, even in children who already have teeth, and its occurrence depends especially on the number and severity of the paroxysms, and likewise upon the condition of the frænulum, *i.e.*, whether it is long and loose or short and tight. For in the latter case the shooting out of the tongue during the attack, and the consequent friction on the teeth does not occur to an extent sufficient to denude the frænum of its epithelium. Since I have directed my attention more to this point I have also occasionally met with cases of quite similar ulceration of the frænum in children who either had no cough at all or were only suffering from an ordinary bronchial catarrh, but had unusually sharp teeth.

The duration of this severest stage is, on an average, about 4 weeks; by the end of this time the nocturnal paroxysms are already beginning to diminish in severity and frequency. Gradually the spasmodic and suffocative character of the paroxysm disappears, the crowing inspirations become shorter and weaker, the final retching ceases, and thus the disease passes almost imperceptibly into the third stage ("stadium decrementi"), which we may regard as another catarrhal stage. Nothing remains now but a loose cough, which still recalls pertussis by many of its features, especially by its tendency to come on in paroxysms and by the unusual redness of the face which it causes. After about 2 or 3 weeks this cough also disappears, and the child is completely convalescent. The whole illness has, therefore, an average duration of from 8 to 10 weeks; and the popular belief that whooping cough is not recovered from in less than 18 weeks is quite erroneous. I

speak, however, only of the average duration; for every practitioner will have known of cases which lasted 3—4 months. But the disease is only rarely continuous in these cases; for in the middle of the third stage it suddenly takes a fresh start, and then of course lasts much longer. In many cases even after the whooping cough is completely gone, a chronic catarrh of the larger bronchi persists; and whenever this is aggravated by a chance cold or by some other cause (*e.g.* measles), the fits of coughing also reappear, and their character still reminds one of whooping cough. Like Rilliet and Barthez I have seen attacks of this kind suddenly recur six months or even a year after the commencement of the disease. In one child the whooping cough lasted from July, 1881, to January, 1882, and then a free interval of 3 weeks took place. Then the cough began afresh, and in February it had become so severe that during the night-attacks a teaspoonful of blood was occasionally brought up. A fresh infection in such cases certainly cannot be assumed. We may much more readily imagine a reproduction of the infective material, which has not yet been completely destroyed or eliminated. I have never myself as yet met with a case of a patient taking ill from an undoubted second infection of whooping cough, which weighty authorities (Roger, West, Trousseau) say they have observed; and I regard with doubt all cases which the relatives have described to me as of this nature. For many cases of simple but chronic tracheal and bronchial catarrh are regarded as whooping cough by the parents, especially if the cough has a rough and slightly whistling character—which is a peculiarity of many children.

In many cases, however, there are considerable variations from the normal course of whooping cough, as I have hitherto described it—variations not only in regard to the paroxysm, but also as to the interval; so that the disease, which is not in itself a dangerous one, may become serious and threaten the patient's life.

Let us first consider the varieties of the paroxysms. I would first draw your attention to the dangerous character of those in which there is a prolonged period of apnœa, during which the child simply keeps coughing and inspires extremely little or almost not at all; and therefore, of course, no crowing sound is heard. You will observe these cases especially in little children in

the first year of life, who are by no means exempt from whooping cough; for I have repeatedly seen the disease in infants, only a few weeks or months old, who had been infected by older brothers or sisters. The cyanosis rapidly reaches an extreme degree, suffocation threatens, and may actually cause death—especially if the disease is complicated by diffuse catarrh or by that and broncho-pneumonia also. Under these circumstances we sometimes have either during the attack, or immediately after it, localised spasmodic contractions (squinting of the eyes, contractures of the fingers, toes, arms, &c.), or even general and fatal convulsions occuring either as the result of the continuous venous engorgement in the brain or of the accumulation of carbonic acid in the blood, which must follow want of sufficient inspiration. At the same time we must not omit to mention that the crowing noise during the attack may also be absent in older children without its justifying a bad prognosis, provided only that the paroxysms are short and the cyanosis and suffocative symptoms do not exceed the ordinary degree, or even fall short of it. Such cases are not very uncommon, and may even cause the physician to doubt whether the disease is really whooping cough. Some characteristic feature of the paroxysm may either be wanting or only slightly indicated, while all its other characters are there, and at the same time other members of the family are suffering from a similar complaint ("coqueluchette" of the French).

Brain symptoms may also be caused by the frequent repetition of the severe attacks above mentioned, which so much interfere with the excretion of carbonic acid, and they may persist in the intervals and cause death with symptoms resembling those of meningitis.

Wilhelm H., 1 year old, admitted on 14th February, 1873, with whooping cough. Very severe attacks with prolonged apœna and epileptiform convulsions, which at first came on only during the paroxysm, but after the 23rd occurred also during the intervals. On 3rd March convergent strabismus of both eyes was noticed for the first time and a staring look; on the 7th repeated chewing movements. After the 18th drowsiness, rigid retraction of the head from contracture of the neck-muscles, from the 19th also contracture of both arms at the elbow-joint, and of the flexors of the leg; steadily increasing coma which lasted till death on the 23rd. After 9th March there was remittent fever

(m. 101·1°—101·8°; ev. 102·6°—103·5° F.), which was found to arise from a double broncho-pneumonia of the lower lobes. On the right side the physical signs entirely disappeared. When the coma commenced the paroxysms of whooping cough became weaker but not less frequent, while the cyanosis considerably increased. The resp. did not fall below 50—60, but became weak and irregular. The temp. of the extremities fell, and bed-sores developed on the occiput and sacrum.

Thus we had here strabismus, staring look, chewing movements, contractures and coma—a group of symptoms which, lasting 3 weeks, decided me to give a diagnosis of tubercular meningitis. And yet at the post-mortem we only found marked hyperæmia of the brain substance, and in some places œdema of the latter. In the left lower lobe there was broncho-pneumonia, and in the right lung only diffuse catarrh. All the other organs appeared quite healthy. We see therefore, here, a state of hyperæmia from engorgement in the brain and pia mater, but particularly the carbonic-acid poisoning resulting from the very severe fits of coughing and from the broncho-pneumonia, all bringing about a misleading appearance of basilar meningitis. The persistently rapid respiration (50—60), and the steadily increasing cyanosis are in favour of this view.

Still greater danger, however, than those of the attack itself may lurk in the intervals. Among all the complications of whooping cough the commonest is diffuse bronchial catarrh, and the broncho-pneumonia which results from it (p. 390). If a child with whooping cough does not seem perfectly well in the intervals between the attacks, but breathes hurriedly and superficially, has a noisy expiration and is feverish—you may at once suspect this complication, and your suspicion will be confirmed on examination of the thorax. Although broncho-pneumonia carries off a large number of the children suffering from whooping cough, we must never give up hope. I have seen very young children who were very ill with extensive consolidation on both sides, completely recover even after variations in their condition lasting for weeks, and after they had been repeatedly given up. Even the occurrence of measles as a complication under these circumstances is not inevitably fatal, although it makes the case much more grave. Much less frequently I have observed croupous pneumonia and pleurisy; and we may almost always find emphysema of the apices and margins of the lung when there is extensive broncho-pneumonic consolidation. I have never myself seen the rupture of distended alveoli which has

been occasionally described, and which is followed by inter-lobular emphysema which may spread over the root of the lung into the neck and over a great part of the trunk. Nor yet have I seen pneumothorax.[1] I have, however, in a child suffering from phthisis, seen a tubercular ulcer in the right main bronchus give way during a paroxysm of whooping cough, and this was followed at once by extreme emphysema of the subcutaneous tissue of the neck and chest.

Broncho-pneumonia when it complicates whooping cough has—as I have already mentioned (p. 391)—a tendency to pass into a chronic condition and to last for months; and then the fits of coughing may persist with undiminished severity. In these very cases I have frequently found after death dilatation and partial fatty degeneration of the right side of the heart—changes which may be explained by the persistent venous engorgement, and by the resistance within the lung-tissue which the heart has to overcome. Under these circumstance I have repeatedly met with œdema on the back of the hands and feet, and with cases of sudden unexpected death from collapse and syncope. The cardiac debility also probably explains the extreme rapidity of the pulse, which has struck me as a peculiar feature in many cases of broncho-pneumonia complicating whooping cough where the temperature was comparatively low. This must not, indeed, be at once set down as a fatal symptom—as the following case teaches—but occurring under these circumstances it always shows that sudden exhaustion of the heart's action may easily come on.

Margarethe H., 1½ years old, admitted on 13th July, 1876, with rickets and whooping cough which had lasted about 4 weeks. Bronchitic symptoms during the last 5 days and severe dyspnœa. At both bases behind, slight impairment with indeterminate breathing and fine crepitations. Fits of coughing only seldom, even during the night-time. After the 18th, the respiration became quieter, the impairment disappeared, and nothing could be heard behind but sonorous and sibilant rhonchi, while the paroxysms of whooping cough (accompanied by vomiting) became worse and worse. After the 21st steady recovery, appetite, no dyspnœa. On 24th discharged. Whooping cough still persisting. During the course of this case we observed the following relations between the respiration, pulse and temperature:—

[1] Roger (*Recherches cliniques sur les maladies de l'enfance*, ii.: Paris, 1883, p. 554), obtained recovery in a case of this kind by puncturing the thorax.

	P.	R.	T.
13th July	200	60	101·5
14th „	180	60	100·4— 99·5
15th „	164	50	101·3
16th „	168	64	98·6—100·4
17th „	144	56	99·5—100·8
18th „	136	52	100·4
19th „	112	40	99·0
20th „	116	40	98·6
21st „	120	44	99·1
22nd „	108	30	99·5

Chronic bronchial catarrh and pulmonary phthisis are not uncommonly found as the sequelæ of whooping cough, the latter developing from chronic broncho-pneumonia which has become caseous. As a result of the enlargement and caseation of the bronchial glands which in protracted cases of whooping cough is set up by the accompanying catarrh of the mucous membrane, acute miliary tuberculosis or tubercular meningitis sometimes developes even after a lapse of years, when the whooping cough itself has long been forgotten. I may finally mention that in several cases I have seen a deformity of the thorax occur as the result of whooping cough, similar to that in rickets, namely, a very marked "pigeon-breast"; and these children had previously been of quite normal conformation and in no way rickety. The occurrence of this deformity is explained, I think, by the excessive atmospheric pressure from without, along with the deficient inspiration and the consequent incomplete expansion of the lung, but, above all, when there is a complication with broncho-pneumonia which keeps up these unfavourable conditions for some length of time.

We know practically nothing of the etiological conditions of whooping cough. It is certain that the disease occurs even in earliest childhood. I have seen it, as already mentioned, in children of 3—6 weeks who had been infected by older brothers and sisters. It occurs most frequently between the 2nd and 6th year of life; still, older children are also often affected, but adults very rarely. In the spring of 1878 I saw a case of whooping cough in a young lad of 16 who had caught the infection at the confirmation-class, and later on he infected not only his two sisters of 12 and 14 years respectively, but also his mother who was 35 years of age. In her case, however, the

disease only took the form of a catarrhal cough occurring in paroxysms with slight cyanosis. In the younger patients, again, there was distinct crowing, and in some also pretty copious hæmoptysis and final vomiting. Cases often occur of the mother being infected by the children; still the disease has generally a very mild form in their case.

There can be no doubt that whooping cough is infectious, and is readily transmitted from one individual to another, so that generally several children in one family suffer from it at the same time. It is therefore all the more difficult to explain the fact that, in my ward where the patients with whooping cough are never isolated, I have only exceptionally observed cases of transmission—which fact is entirely at variance with Roger's experience. As to the period of incubation I possess no definite experience, but I have frequently observed the fact that if a child introduced the disease from school into a family, it took at least 10—12 days before a cough was heard among the other children. It is naturally assumed that the contagion reaches the respiratory mucous membrane along with the inspired air, and thence exerts its action, and so, as a matter of course, bacteria have been described as the cause of whooping cough.[1] However probable this may be, it cannot be said to have been demonstrated by the conditions described as having been found, as they are not abreast of the present state of bacteriology. When we regard whooping cough as an infectious disease, it naturally follows that we look for a feverish premonitory stage analogous to that found in the acute exanthemata. I cannot altogether deny the occurrence of this, but I would remind you that the first stage of this, as of every other catarrh, may come on with great severity, and in that case be accompanied by fever. Trousseau also[2] speaks of a very acute catarrhal stage, and I myself have frequently observed it.

The action on the respiratory mucous membrane of this still-unknown infectious material is not confined to the setting up of an ordinary catarrh of the trachea and of the bifurcation, as many have maintained. I certainly shall not dispute the fact that such a catarrh is present or may be present; and this condition

[1] Letzerich, *Jahrb. f. Kinderkrankh.*, 1870, iii., S. 534; 1873, S. 436.—Tschamer, ibid, 1876, x., S. 174.—Burger, *Berl. klin. Wochenschr.*, 1883, i.—Deichler, *Deutsche Medicinalz.*, 1886, No. 74.

[2] *Clinique* i., 497.

has in fact been proved, by laryngoscopic examination to occur, at least on the mucous membrane of the larynx and trachea.[1] Everyone, however, who has once heard a fit of whooping cough must admit that there is something more in it than the mere catarrh—namely, a nervous element. It is this that gives the peculiar character to the attacks and manifests itself on the one hand by the spasmodic violence of the expirations, and on the other hand by apnœa, and by the crowing sound of spasmus glottidis. I would further remind you of the symptoms described (p. 453) as constituting the aura of the attack, and also of the almost invariable vomiting. I grant that the retching and vomiting of mucus at the end of the violent paroxysms must be regarded simply as a mechanical act, resulting from the violent contraction of the abdominal muscles in coughing; for we frequently see the same thing result in children especially from other violent paroxysms of coughing having nothing to do with whooping cough, if the stomach is very full. We must remember, however, that many children vomit even when the whooping cough is very slight; and likewise that cases occur in which the vomiting forms the most prominent feature of the paroxysm, and may even excite serious anxiety by its persistence. I have known children who, after a short attack with no crowing whatever, at once brought up the whole contents of the stomach, while others even in the intervals of the paroxysm vomited all their food and gradually sank into a state of serious debility, although no cause for this could be found in the digestive organs themselves. Such vomiting cannot be looked upon as other than nervous. It is as yet an open question whether a reflex excitability of the medulla oblongata acting through the vagus is to be blamed here, and in what way exactly the specific contagion exerts such an influence on the central nervous system. It is at any rate certain that pathological anatomy gives us no explanation of it, and that other changes which are found post-mortem—especially the much-talked-of enlargement of the bronchial glands—are only to be regarded as sequelæ or complications of the disease.

Whooping cough often occurs in more or less extensive

[1] Rehn (*Wiener med. Wochenschr.*, 1866, 52 and 53), Meyer-Hüni (*Zeitschr. f. klin. Med.*, i., Heft 3), and Herff (*Deutsches Arch. f. klin. Med.*, Bd. xxxix., No. 3 und 4), describe this catarrh while Rossbach (*Berl. klin. Wochenschr.*, 18, 1880) was unable to satisfy himself of its presence.

epidemics, which in general are not confined to any particular season of the year. A certain relationship to measles, which West has drawn attention to, cannot be overlooked. We often observe not only the combination or succession of the two epidemics, but also it appears to me that individual patients who are suffering from one of these diseases seem to possess a peculiar predisposition to the other. The combination of these two diseases in one and the same individual is always a serious matter; for in these cases there almost always arises an extensive and particularly obstinate broncho-pneumonia tending to become chronic. It is worse still if a child who is already suffering from whooping cough and broncho-pneumonia takes measles as well. In such cases I have seen cyanosis appear even before the outbreak of the eruption, the measles-rash at once becoming bluish; and after a few days death ensued with symptoms of carbonic-acid poisoning. Nevertheless, as I have already mentioned, even this complication is not necessarily fatal. The combination of whooping cough with diphtheria, which I have not uncommonly seen in the hospital, I regard as even more serious; but even here we must not at once lose courage. In a girl of 11, in whom a complete loss of voice had already made extension of the disease to the larynx probable, perfect recovery took place notwithstanding. I may mention that in this case, instead of the crowing inspiration during the paroxysm of the cough, a quite harsh, almost croupy sound was heard, evidently caused by the swelling and roughness of the laryngeal mucous membrane. Should tracheotomy have to be performed, the retarding influence of the whooping cough paroxysms is to be feared.[1]

You will have seen from this description that while the prognosis in whooping cough is favourable so far as the disease is concerned, yet serious danger to life may arise on the one hand from the extreme youth at which it sometimes occurs, and on the other from certain of its complications (bronchitis, broncho-pneumonia, convulsions). Further, even after complete recovery caseous deposits may be left behind in the lungs or bronchial glands, and may later on form the starting-point of miliary tuberculosis.

[1] In one case the wound broke open again after 2 months (Roger, *loc. cit.*, p. 614).

In the treatment, unfortunately, you will not acquire much credit. The enormous number of remedies recommended from of old for this disease, is of itself sufficient to prove their inefficiency. We do not possess any remedy capable of cutting short the disease, especially when at its height; while in the last stage, when natural recovery sets in, apparently every remedy is helpful. A second fact worthy of notice is, that whooping cough, like every other infectious disease, may occur in a very much weakened, so to speak abortive, form, in which it runs its course in a much shorter time than usual, and is recovered from without any other treatment. Every physician, like myself, has met with such cases, I suppose (although I regard as somewhat doubtful one mentioned by Trousseau, in which the disease is said to have lasted only for 3 days); and therefore I think we cannot be too cautious in judging of the results of treatment in this disease. You will therefore excuse me if I do not go over the list of all the drugs which during a number of years I have tried, either on my own initiative or acting on the recommendation of other people, and found ineffective. I have now come to put trust only in one, namely morphia (Form. 10), which is far more efficacious than the much-used belladonna—at any rate in relieving the violent attacks, especially those occurring during the night, and in diminishing their frequency. It does not, of course, influence the general course of the disease. In prescribing this remedy, however, especially in practice among the poor, you must never omit to charge the mother to stop the medicine as soon as unusual sleepiness shows itself. Owing to this precaution it has only once happened in my practice that a child slept uninterruptedly for 18 hours without being disturbed by a single fit of coughing; the attacks at once set in again when the narcosis passed off. Further, I knew another case of a child (6 months old) being poisoned in some inexplicable way, and who showed symptoms of collapse, narrowing of the pupils, and coma; fortunately he recovered under the use of cold douches and restoratives. I have always been very cautious in administering the medicine, and I have never yet had any mishap occur, even when giving 1—2 teaspoonfuls daily for weeks. I therefore prefer this medicine very much to all other narcotics, and especially to a drug so dangerous as atropine. Still I would only recommend

the use of morphia in severe cases with at least 20 fits occurring within 24 hours.

The bacteriological explanation of the disease, although not yet proved, has at least had this effect, that attempts have been made in various ways to deal directly with the supposed germs of infection. Inhalations of carbolic acid vapour were first tried (Burchard, Thorner, and others). These were much praised and replaced the former plan of sending the patients to reside in gasworks, which I have always regarded as inadvisable, owing to the danger of catching cold.

My own experience as to this treatment does not allow of my giving a final opinion; because its results are sometimes strikingly favourable, sometimes doubtful, and sometimes there are none at all. I can say, at any rate, that I never knew of it doing any harm. We may either order a 1—3 per cent. solution of carbolic acid to be inhaled from a spray-producer several times a day, or if there is anything to prevent this we may charge the air of the nursery with the vapourised solution and hang over the head of the bed a sponge saturated with it. We may also order a sponge thus treated to be held before the child's nose several times a day, so that the vapour may be inhaled for several minutes. I have entirely given up other forms of inhalation—chloroform, benzoin, salicylate of soda, turpentine, tannin, quinine, &c. As to the painting of the pharynx and larynx with parasiticide (!) substances which has of late been much employed, we may object, to begin with, that we know as little concerning the position of the bacteria as we know about themselves, and we can therefore have no means of knowing whether we really reach them with the brush. The method, however, is certainly worth a further trial, as Moncorvo[1] says that he has seen good results from painting the entrance to the larynx with a 1—2 solution of resorcin. Also injections of salicylic acid (1:1000) or of corrosive sublimate (1:10,000) into the nose, as well as insufflations of quinine or benzoin into it have been recommended for the same purpose.[2] Finally, painting the pharynx and larynx with 5—15 per cent. solution of muriate

[1] "De la nature de la coqueluche et de son traitement par la résorcine". Rio de Janeiro and Paris, 1883 and 1885.

[2] Goldschmidt, *Deutsche med. Zeit.*, 1885, No. 61.—Michael, *Deutsche med Wochenschr.*, No. 5, 1886.

of cocaine is the most recent form of local treatment.[1] This deadens the sensibility of the parts, and is said to have frequently brought about a rapid diminution in the frequency and severity of the attacks. Moncorvo[2] recommends that the two methods should be combined (the treatment with resorcine to follow the painting with cocaine).

My own experience with cocaine has not been satisfactory. Several cases (treated in the ward) which were painted thrice daily were improved for a time, but not permanently. Others treated in the polyclinic (with only one painting daily) were even less successful. I do not think that this tedious and often difficult proceeding deserves the praises which many have bestowed upon it.

At any rate you must, I think, relinquish any idea of cutting short the whooping cough attack, and let the parents know from the first that nothing can be looked for beyond mere alleviation of the paroxysm. When the weather is fine, as much of the fresh air as possible should be allowed; on the other hand when it is windy and inclement—and also when the patient has bronchial catarrh—it is to be strictly forbidden. Very often, indeed, the neglect of this precaution avenges itself by an attack of broncho-pneumonia. When whooping cough occurs during the summer, you will often be asked whether a change of air might not do the child good. Although a number of physicians consider this beneficial and even recommend certain definite localities—*e.g.* residence on the coast of the North Sea—as especially favourable, my own experience does not permit me to agree with this view. I have often sent children who had whooping cough to watering-places with their parents, either on the sea-coast or among the mountains; but I have scarcely ever seen any good result from so doing. The patients go on coughing as before, and the only result in such cases is one not to be desired—namely, the infection of healthy children who came in contact with the patients at such places. Only in exceptional cases—as, for instance, in that of my own child—have I seen an attack of whooping cough which was in process of development, and had already the characteristic paroxysms, entirely disappear

[1] Barbillion, *Revue mens.*, Août, 1885.—Prior, *Berl. klin. Wochenschr.*, 1885, No. 45, 46.

[2] "De l'emploi du Chlorhydrate de Cocaïne dans le traitement de la coqueluche:" Rio, 1885.

in a fortnight spent at Reichenhall. Such isolated cases, however, seem to me (bearing in mind the occurrence of "abortive" whooping cough already mentioned) quite insufficient to prove the favourable influence of change of air or the merits of any particular locality. As to the treatment of the complications (eclampsia, broncho-pneumonia) you may consult the prescriptions already given for these diseases. Protection from whooping cough could only be guaranteed by the complete isolation of the children; and this can hardly be carried out in practice, especially since (according to Roger) the isolation must last from 2 to 3 months.

SECTION V.

DISEASES OF THE CIRCULATORY ORGANS.

Pathological changes in the heart are not much rarer in children than in adults. The age causes neither anatomical nor clinical differences of any essential importance, and I may therefore confine myself to a comparatively short description of these diseases.

I. *Affections of the Large Blood-vessels.*

There is very little to say about the affections of the large blood-vessels in children, for these are extremely rare. Although Hodgson has observed ossification of the temporal artery in a child of 15 months, and Andral calcareous plates in the aorta in a girl of 5 years—still, these are exceptional occurrences, and I have never had an opportunity of observing them; nor have I seen an example of aneurism of the aorta in childhood.[1] Also the congenital stenosis of the aorta, which is generally situated in the region of the ductus arteriosus or at the commencement of the descending aorta, are much more frequently diagnosed in youth, or even later, than in childhood; although some of them seem to have some connection with the involution of the ductus arteriosus which spreads to the aorta. I may take this opportunity of mentioning that the closure of this duct (which in new-born children is about the thickness of a branch of the pulmonary artery) is brought about by an endarteritis obliterans with the formation of new fibrous tissue, thickening of the walls, and narrowing of the lumen. The process is noticeable on the 9th day after birth, it has usually gone on to the formation of a stricture in the middle of the duct by the 14th day; it then proceeds further in both directions, and is generally completed by the end of the third week. The obliteration of the foramen ovale is completed, in 88 per cent. of

[1] Out of 98 cases of aneurism of the thoracic aorta, there was only one under 20 years; and among 59 cases of aneurism of the abdominal aorta, there was not even one under that age.

the cases, by the third month after birth.[1] Anything which causes a deficient filling of the left ventricle during the first period of life—such as extensive atelectasis of the lung-tissue, fœtal pneumonia, or stenosis of the pulmonary artery—must delay the process of closure of the ductus arteriosus. For under these circumstances the blood is continually flowing from the pulmonary artery through the duct into the insufficiently filled aorta. The delayed obliteration of the duct may therefore in such cases ward off during months the evil effects of the engorgement in the right side of the heart and the general venous system which would otherwise have taken place. The same may be said of the persistent patency of the foramen ovale, which, apart from the causes named, may be due to local abnormalities of the foramen or its valve.

II. *Congenital Cyanosis.*

The persistent patency of the fœtal channels—ductus arteriosus and foramen ovale—was formerly regarded as the principal cause of congenital cyanosis. As the cause of this was supposed to be the mixture of arterial with venous blood, it was thought that the abnormal colour was due either to the remaining open of these channels or to an abnormal communication between the two arteries or ventricles owing to an aperture in the septum between them. Now, however, we know that cyanosis also occurs when there is no mixture of the two kinds of blood, and that, on the other hand, such abnormal communications have been found in children—and even in adults—who during life presented no trace of cyanosis. Zeyetmayer's case is well known, in which the entire ventricular septum was absent, and still there was no cyanosis. Equally well known is that of Breschet, in which the left subclavian artery rose from the pulmonary artery; and yet the affected arm was normal in colour.

Let us consider cyanosis for a moment. From the time of birth, or at least very soon after it, there appears a bluish-violet tinge on the cheeks, point of the nose, hands and feet, especially on the nails and the visible mucous membranes (tongue, buccal mucous membrane, entrance to nostrils, palpebral conjunctiva).

[1] Thèremin, *Revue mens.*, Févr., 1878.

This is considerably heightened by screaming, crying, sucking, any energetic movement, or on being exposed to cold air. But during the intervals the cyanosis may be so slight as scarcely to be noticed by a non-medical eye. After it has lasted some time —but occasionally even in the first months of life—there is developed a club-shaped (or drumstick-like) enlargement of the terminal phalanges of the fingers and toes, and often a claw-like condition of the nails. Two or three times I have noticed also that the dark-violet gums presented a spongy character like that seen in scurvy. They bled readily either spontaneously or on being touched, and were separated from the teeth at their margins. In one girl of $1\frac{1}{2}$ years this appearance was so marked that her mother brought her to the hospital on account of it, although she had entirely overlooked the cyanosis. The temperature of the extremities is very low (sometimes as low as 89·6° or 82·4° F.), while that of the body as estimated in the rectum is found to be normal. Added to this we often have a condition of general debility, languid movements, sleepiness, backward growth and intelligence, and, finally, the whole series of well-known symptoms which are characteristic of the various kinds of heart disease—œdema of the hands and feet, epistaxis, dyspnœic attacks (especially after violent movement), fainting-fits, enlargement of the liver and spleen, &c. On physical examination we often observe a very distinct increase in the size of the heart, especially of its right side, systolic or diastolic murmurs, and perhaps a pulsatile thrill; but in many cases, also, no abnormality at all. Other malformations may be present at the same time, among which I may mention as rare conditions which I have myself observed, obliteration of the auditory meatus, malformation of the external ear, and eccentric position of the two pupils.

From these symptoms we may, it is true, diagnose with certainty the presence of a congenital malformation of the heart, but in most cases it remains an impossibility to discover the exact nature of the malformation. As I cannot here discuss the foreign treatises on this subject—which indeed are generally only compilations and criticisms—I would refer those who are interested in this matter to the excellent work of Rauchfuss,[1] who has had at command an unusually large

[1] Gerhardt., *Handb. f. Kinderkrankh.* iv., 1878.

amount of material of his own and has also brought together almost everything that is known on this subject. You must not, however, expect any great practical use from it. The author himself is obliged repeatedly to acknowledge that all endeavours to find definite diagnostic criteria for the different malformations, can only afford at most a more or less probable diagnosis. These malformations consist either in apertures by which the two auricles or ventricles communicate with one another, or in larger defects—which in their most extreme developments take the form of complete absence of the septum—or in stenosis and atresia of the conus of the pulmonary artery, of that vessel itself, of the aorta or of the auriculo-ventricular opening; finally, in transpositions of the large blood-vessels, the pulmonary artery arising from the left, the aorta from the right ventricle. The insuperable difficulties in the way of diagnosing these abnormalities are, moreover, increased by the fact that in the majority of cases there is a combination of two or more of them; and also that the symptom to which the physician's attention is principally directed —namely, the congenital cyanosis—may be completely absent. This visible symptom does not accompany every malformation of the heart. I have often met with such children in the first months of life or at least in the first year, who either suffered only from attacks of dyspnœa or else presented no cardiac symptoms of any kind, and were brought for treatment only on account of an affection of the lung or bowel. Of this, allow me to give one example:—

> Child of 30 days admitted with congenital syphilis. From 19th to 21st March, 1873, a febrile pneumonia of the right upper lobe (T. 97°—99° F.; R. 56—70). No cyanosis; no abnormality of the heart audible. On post-mortem we found (besides the pneumonia, syphilitic affection of the bones and interstitial hepatitis) considerable malformation of the heart. The ventricles communicated with one another by a large aperture, the septum being almost entirely wanting; and that between the auricles was very thin. The tricuspid valve was wanting and the mitral valve was inserted at one extremity into the right side of the heart. The arteries normal.[1]

If the children live for some years, there generally, of course,

[1] Very rare indeed are cases such as that observed by Barth (*France méd.*, Juni, 1880), in which congenital endocarditis was discovered even before birth by auscultation of the fœtus (loud blowing murmur replacing the first sound).

occur more or less marked symptoms, usually with cyanosis. They arise either under the influence of chance respiratory affections, or from endocarditis which developes in connection with the abnormal apertures or congenitally-affected valves and openings—just as in adults it arises in the neighbourhood of old valvular disease (endocarditis recurrens). Under these circumstances the hitherto latent malformations become manifest and we now recognise on examination (which in many cases, is now made for the first time) that there must have existed an abnormality of long standing. The cases of stenosis and atresia of the pulmonary artery or its conus usually produce the most marked symptoms, and they also form the commonest cause of congenital cyanosis. In many cases it is impossible to determine whether the stenosis and partial atresia of this artery are due to fœtal endo- and myocarditis or to a primary arrest of development to which an inflammatory process has subsequently been added. This stenosis must always give rise to dilatation of the right side of the heart and considerable engorgement in the entire venous circulation (of which, of course, the cyanosis is an expression). The cardiac dulness then extends beyond the right border of the sternum, the heart's impulse is visible and palpable over a larger area than usual, and a thrill can often be felt along with it. A secondary systolic murmur is also audible over the heart, being loudest over the orifice of the pulmonary artery and between that and the clavicle ; occasionally also over the whole thorax and back. Variations, however, in the symptoms may be caused by the presence of other malformations of the heart at the same time, which renders the diagnosis more difficult. Nor are examples wanting in which the heart sounds are quite pure, without a murmur of any kind. The diagnosis of malformations of other parts of the heart is still more difficult; and you will excuse me, if I do not enter further into particulars regarding it, as in practice the cases for which these hold good are of exceptional occurrence.

As to the course of cases of congenital malformation of the heart we can never predict anything with much certainty. The greater the obstructions to the venous circulation and the less they are counter-balanced by other compensating malformations (apertures in the septum, persistent patency of the ductus arteriosus)—the shorter will the child's life be. Children with

very marked stenosis of the pulmonary artery die early, even although the foramen ovale be still open and there is no cyanosis; while children with less marked stenosis may grow up to youth or even live beyond that, especially if the fœtal channels are not closed or if there are apertures in the septum. The same may be said of cases of stenosis of the aorta, which are almost all observed (and partially diagnosed) for the first time only at a late period of life. I have frequently seen febrile diseases (*e.g.*, the acute exanthemata) run their course in such children without doing any harm. The fatal issue takes place at last, in these as in all other diseases of the heart, either suddenly by syncope or from the result of some disease of the respiratory organs which would not of itself have been dangerous to life (diffuse catarrh or pneumonia), less commonly with symptoms of gradually increasing venous engorgement and dropsy. Caseous pneumonia also, which may be associated with similar processes in other organs and with miliary tuberculosis, is sometimes the cause of death, and the immunity of cyanotic patients from tuberculosis of the lungs, alleged by Rokitansky, is certainly not borne out by the actual facts.[1]

As has been already mentioned, it often occurs that on examining children who have been brought to us on account of some entirely different ailment, we find by chance valvular diseases and their results, which are causing either no subjective symptoms at all or at most a scarcely noticed palpitation or shortness of breath when the patient runs or ascends stairs.[2] Even the most careful history may fail to throw light on the origin of this affection, and we may be told that the children have always been healthy and have never suffered from rheumatism, scarlet fever or any inflammatory chest affection. We are therefore obliged in such cases, in spite of the absence of cyanosis, to assume that the disease has been congenital. I shall take this opportunity of reminding you that in very young, even in new-born children, small spherical projecting blood-extravasations occur on the cardiac valve, especially on the free border of the mitral, as

[1] Rauchfuss, *loc. cit.*, S. 92.

[2] Similarly, a boy of 8 years, on being examined during a slight attack of articular rheumatism, was found to have his heart on the right side. The cardiac dulness and impulse were only to be found on the right side of the sternum, the right nipple rose with the systole and the first sound was accompanied by a blowing murmur. The abdominal viscera were, however, in their normal position.

described by Luschka long ago.[1] More recently those valve-hæmatomata have been investigated by Parrot.[2] He has met with them often in new-born children at the venous orifices on both sides of the heart, in the form of very small projections (in some cases, however, even as large as a cherry-stone), black or violet in colour and of globular or conical form. These hæmatomata, which he attributed to a rupture of intra-valvular blood vessels, are situated under the most superficial layer of the endocardium. They seem to arise very soon after birth, perhaps even before it, and generally disappear within the first few months of life, their covering gradually shrinking, while at the same time there is a proliferation of the epithelium and connective tissue in the neighbourhood. It also appears that small hard nodules covered by epithelium and either with a broad base or pedunculated, which not uncommonly occur in the same situations and have already been mentioned by Cruveilhier[3] and others, may grow from the hæmatomata. It is, indeed, possible that owing to an abnormal process of resolution occurring in such hæmatomata, shrivelling of the borders of the valves and at the same time stenosis of the ostium, or incompetence of the valves may occur, but when these are found in older children, it is no longer possible to ascertain how they arose. The valvular disease would not in that case be really congenital, but would have arisen during the first few months of life.

The treatment of diseases of the heart which are either congenital or have arisen during the earliest period of childhood, must be limited to enjoining the quietest possible life; and the carrying out even of this prescription in older children—whom it becomes necessary to separate from their playmates—meets with great, even insuperable difficulties. In other respects also, the treatment is exactly the same as that of organic heart-disease.

III. *Inflammation of the Pericardium, Endocardium, and Myocardium.*

In many cases an attack of acute rheumatism can be

[1] *Virchow's Archiv*, xi., Heft 2.

[2] *Arch. de physiol.*, Nos. 4 and 5, 1874.

[3] For another explanation of these "nodules" based on development, see Pott, *Jahrb. f. Kinderheilk.*, 1878, xiii., S. 29.

assigned as the starting-point of organic disease of the heart. The time when this disease was regarded as of rare occurrence in childhood has long passed away. Since I shall have to return to this disease on a later occasion, I shall only remark here that although its occurrence in children is, as a rule, less common and less severe than in adults, the complication with endo- or even with pericarditis, is much commoner in them than in adults. Even in quite slight attacks of rheumatism (with but little rise of temperature) which occasionally appear as hyperæsthesia of the limbs or joints without the latter being swollen, you must never neglect to examine the heart. You will often be surprised to find peri- or endocarditic murmurs in these cases, although owing to the apparent slightness of the affection you were not prepared to find them. In cases of advanced valvular disease we very often find from the history that one or more attacks of acute rheumatism—especially in the joints—had occurred months or years before. As these diseases of the valves and their results so completely correspond to the same conditions in adults, it is unnecessary for me to consider their physical signs more fully. With regard to the subjective symptoms, I shall only mention the fact, that although in adults cases of prolonged compensation and consequent latency of the valvular disease are not rare, the same thing appears to me to be still commoner in children. No noticeable distress is caused by the violent movements in playing or running up stairs, and in many cases the disease is first discovered by the mother observing the violent motion of the heart when she strips the children to bath them. It is only when the compensation begins to be disturbed, that the cardiac symptoms, which you are well acquainted with, set in; and these sooner or later bring about the fatal issue. Even from an anatomical point of view the disease is just the same in children as in adults; in the one case as in the other we find dilatation and hypertrophy of the ventricles, the brownish-red induration of the lungs, the hæmorrhagic infarcts, the congested kidneys and liver, the enlargement and induration of the spleen, the œdema, and the dropsical effusions in the various cavities and in the alveoli of the lungs.

Although in many cases the valvular disease due to rheumatism first appears months or years afterwards, still, on the other hand, examples do occur of a much more acute course.

Anna M., 7 years old. Formerly always healthy. Acute articular rheumatism, especially in the lower limbs, in the middle of December, only lasting a few days. Between Christmas and New Year when she was feeling quite well again, she suddenly fell ill once more, with palpitation, diminished secretion of urine, coughing, and frequent pains in the region of the heart. Admitted into the ward 12th February, *i.e.*, about two months after the beginning of the illness. On examination we found general anæmia, catarrh in both lobes (especially in the left), cough, and dyspnœa. The cardiac dulness reached to the right border of the sternum, above to the third rib, on the left to the mammillary line. Heart's impulse heaving and diffuse. Indistinct apex-beat outside the mammillary line in the 5th intercostal space. The first sound of the heart obscured by a loud systolic murmur; both second sounds pure and unusually loud. Pulse small, 120—144; no fever; urine very scanty, marked albuminuria. On the 21st the temperature suddenly rose to 104° F., then fell again rapidly, and by the 24th had not risen again above 100° F. On the morning of the 22nd distinct pulsus bigeminus; pericardial friction at the left border of the sternum. Increasing collapse (T. 98·1° F.), slight cyanosis, extremely rapid breathing (84). Death in the night of the 24th.

P.-M.—Heart about thrice its normal size, both ventricles much dilated and hypertrophied. Aortic and mitral valves thickened along their free margins, somewhat retracted and covered with greyish-red warty growths. Recent partial adhesions of the two layers of the pericardium on the anterior surface of the septum ventriculorum. Diffuse bronchial catarrh; œdema and brownish-red induration of the lungs.

In a girl of 7 years, who in October, 1874, had had a slight attack of acute rheumatism combined with endocarditis, I found (March, 1875) not only the signs of mitral incompetence, but also even then very considerable hypertrophy and dilatation of both ventricles.

A boy of 7 (May, 1882) had, 12 weeks after the beginning of a rheumatic attack, shown signs of extreme eccentric hypertrophy with changes in the aortic and mitral valve, and a consequent well-marked bulging of the precordial region.

In a boy of 10, who took ill in May with an attack of acute rheumatism and peri-endocarditis, and since that time had had repeated relapses, we found (on the 19th December) cyanosis and all the symptoms of far-advanced heart disease. At the post-mortem we found incompetence of the mitral valve, hypertrophy of both ventricles, complete adhesion of the pericardium, brown induration of the lungs, &c.

A girl of 10 years took ill with acute rheumatism (with slight choreic symptoms) in September, 1886. By the middle of November she presented the symptoms of incompetence of the aortic valves and hypertrophy of the left ventricle.

You find, therefore, in these cases eccentric hypertrophy of one or both ventricles already developed as the result of valvular disease only a few months after the first onset of acute articular rheumatism. In the first case the course was so sudden and acute that compensation was altogether out of the question, and the end was further accelerated by complication with diffuse catarrh and by the recent peri- and endocarditis which were finally added to it. This "endocarditis recurrens" we have often found post-mortem in old cases of valvular disease, which was either congenital as in the case above or else acquired at a later period. Although this process is usually discovered first at the post-mortem, it can occasionally be recognised at the bed-side.

In September, 1872, I had under treatment for acute articular rheumatism with endocarditis a girl of 5 years who had previously been healthy. After her recovery the systolic murmur at the mitral valve persisted without disturbing the child's general health, and of this I was able to convince myself after a year's interval, in November, 1873. In January, 1875—*i.e.* about 3 years after the beginning of the illness—a fresh endocarditis developed in the already much dilated and hypertrophied heart, manifesting itself by fever, increased loudness of the murmur, and extreme dyspnœa, and ending fatally.

On the other hand experience teaches that children get over rheumatic endocarditis better than adults do, and are more likely to recover completely from its results. In the whole course of my practice I have only had one adult patient under treatment for rheumatic endocarditis of many months' duration in whom I have observed a musical murmur entirely disappear and complete recovery take place, which I know to have been permanent. In children recovery is more frequent, although even in them a permanent valvular lesion remains in the great majority of cases.

Clara F., 3 years old, took ill in October, 1871, with rheumatic pains and swelling of the joints of hands and fingers. There was high fever, rapid breathing, and at the end of the 1st week a loud systolic murmur at the apex, without any change in the percussion. Bronchial catarrh. After 14 days, all the symptoms had vanished except the murmur, which in spring, 1872, gradually began to grow fainter, and by November had entirely disappeared.

Paul H., 6 years old. In beginning of February, 1868, he complained of pains in the upper part of the abdomen (especially on

stooping), dyspepsia, and moderate fever. On the 16th a warm bath, in which the child took a severe chill. After 1½ days violent fever, pain and slight swelling of the joints of the right hand and foot; flexion of the right knee-joint and adduction of the thigh. Both of these could only be overcome with severe pain. During the next few days the joints of the hand recovered, but pains with difficulty of movement appeared in the left thigh. Fever moderately persistent, bronchial catarrh, heart unaffected. After a temporary improvement all the symptoms became worse again. On the 29th high fever, loud diastolic murmur over the heart, especially in the mammillary region, disappearing as one passed upwards. Vesicant, calomel with digitalis. General improvement. On 22nd March, normal in every respect with exception of the anæmia and the persistent diastolic murmur. In the spring of 1869 this also had entirely disappeared, and the boy remained healthy henceforward.

In the last case we see the endocarditis first appear with the exacerbation of the fever and other symptoms of rheumatism, on 29th February; while the first 9 days of the disease passed without any affection of the heart, and we were already expecting convalescence to begin. Such occurrences are by no means rare.

On 19th June, 1875, I was consulted about a boy of 5 years who had already been ill for a week, with acute articular rheumatism. In the middle of the second week the fever and the pains ceased for 3 days. Then, however, a fresh exacerbation suddenly took place, and with it an affection of the heart. Pains in the region of the heart and loud friction along the sternum, following both sounds of the heart, put pericarditis beyond a doubt. By local blood-letting, inunction with mercurial ointment, calomel and digitalis, considerable abatement of all the symptoms was brought about after 8 days; the fever was quite gone, the friction could no longer be heard, but in its place a loud systolic murmur was now audible. Some months afterwards, when I again examined the child, this murmur still existed.

In both of the last cases the heart-affection set in for the first time along with a fresh exacerbation of the rheumatism. But cases do sometimes occur in which endocarditis appears as the first sign of the rheumatism, and the joint affection is only found later on.

Paul F., 5 years old, had been out of sorts for about 12 days, with irregular fever, loss of appetite and unusually rapid breathing. It was only 5 days ago that the physician in charge had been able

to discover a systolic mitral murmur, and therefrom to diagnose endocarditis. When called in on 13th May, 1875, I was able to confirm this. The boy complained on this day for the first time of pains in the limbs, and in the evening an attack of multiple rheumatism suddenly came on in the joints of the feet, knees, and arms, with severe pain, stiffness, swelling and sleeplessness. T. 102·2°—104°. No change during the next few days, Digitalis tried and found useless. From 26—27th May the rheumatism spread to other joints. Extreme dyspnœa; sternum and neighbouring parts dull on percussion, heart-sounds and murmur weaker, so that a complication with pericardial effusion seemed probable. Death on 3rd June from rapid increase of this condition, the pulse becoming small, the skin cyanotic, and the area of dulness rapidly extending. Post-mortem refused.

Here, then, you see the endocarditis not following the onset of acute articular rheumatism, but preceding it by at least 5 days; for I am of opinion that the indefinite illness which the boy had suffered from for 12 days was due to the endocarditis, even although its presence could not be discovered on physical examination. So long as the endocarditis does not affect the valves or the openings, no abnormal murmurs may be present. Indeed it is proved by certain cases of endocarditis ulcerosa in adults—*e.g.* during the puerperium—that even ulcerative lesions of the valves may exist without being accompanied by adventitious sounds. I shall never forget the wife of a medical man, who for at least a fortnight presented no symptom beyond general malaise and remittent rise of temperature with a very quick pulse; no organic lesion could be made out anywhere, in spite of the most careful examination. It was only after a lapse of 14 days that I discovered a steadily increasing systolic murmur over the heart and diagnosed endocarditis, which the post-mortem examination confirmed. We find in children also cases of this kind, which for some time are not recognised, and may readily pass for typhoid. In a boy of 3, who had suffered some months before from a slight attack of rheumatism, I found endocarditis, the presence of which was only indicated by high fever (103·1°—104·9° F.) during 3—4 days. It was only after that period that endocardial murmurs were heard, and they were soon followed by friction. The case last given was quite similar to this one (Paul F., p. 480). The occurrence of rheumatic pericarditis, which appeared as a complication in both cases and which is by

no means rare, may render the diagnosis difficult owing to the addition of its auscultatory signs.

Emil P., 11 years old, about whom I was consulted on 19th December, 1877, had taken ill about a week before with a feverish sore throat. A few days afterwards painful swelling and immobility of both ankles and knee-joints, for which acid. salicyl. grs. ivss. every 3 hours, was given with good results. Since the 17th, sudden violent pains in the left side of the chest and increased fever. P. 132 regular. There was a loud systolic murmur at the apex which became less distinct above, and at the same time a friction-sound over the lower half of the sternum accompanying both sounds of the heart, and extending beyond the epigastrium, and as far as the mamma. Percussion unaltered. Blister between the nipple and sternum; digitalis. Eight days later the fever and pericardial friction had disappeared. The endocardial murmur, however, remained unchanged, and the boy still complained of sharp pains, and a feeling of oppression, and was often obliged to stop for breath in the midst of talking. Pot. iod. On 3rd January, 1878, the child was well, but for rheumatic pains in the left shoulder. The mitral murmur was still present for 2 years after, so that there must have been permanent valvular disease.

Carl S., 8 years old, took ill in the end of December with a slight attack of articular rheumatism. A few days after, endocarditis set in (high fever, quick breathing, pains in the left side of the chest, and loud blowing murmurs, accompanying both sounds of the heart). Ice-bag and digitalis. After some days no heart-sounds could any longer be heard, but only two murmurs. Blister. Two days after, the murmurs were less loud, and both sounds of the heart could again be made out; at the same time, however, there was pericardial friction at the middle of the sternum and at its right border. The cardiac dulness now gradually extended beyond the sternum, and on the 13th January reached to about $\frac{5}{8}$ inches beyond its right border, while the dyspnœa was considerably aggravated by the occurrence of pleuro-pneumonia of the left lower lobe. P. 150 pretty full; R. 50—60. Dry-cupping, digitalis, wet compresses, ice-bag over the heart when the pain was severe. Although the disease had taken the form of pneumonia migrans, and had affected the left upper lobe by the 17th, there nevertheless occurred, to our surprise, a gradual improvement of all the threatening symptoms. The pericardial friction had disappeared by the 15th. The enlarged cardiac dulness (pericardial effusion) receded within its normal limits, and by the 27th the child was able to leave his bed. The striking fact remained, however, that the apex beat could always be felt $\frac{3}{4}$—$1\frac{1}{2}$ inches outside the left nipple line, even when the child was lying on his right

side (adhesion). Several years after, I found on examination all the symptoms of incurable valve-disease.

In both cases, then, pericarditis was added after a few days to an already-existing rheumatic endocarditis. And the results of this disease, if we are to judge by the physical signs, may indeed appear to be recovered from more satisfactorily than those of endocarditis, but still adhesions of the two layers of the pericardium, or of the pericardium to the pleura, may be left behind. As a rule, when pericarditis sets in, we find the friction first over the base of the heart, while the systolic murmur is most generally found at the apex.

As to the relationship between chorea and rheumatic heart-affections, I have already given my opinion (p. 207), to the effect that both chorea and endocarditis rise from the same source—namely, from rheumatism—but that the former is not to be regarded as depending on the heart-disease alone. I would further call your attention to the fact that the fundamental rheumatic condition may be very trifling, and may even be quite overlooked, especially in children who are only suffering from vague muscular and articular pains. Likewise, that the secondary endocarditis and chorea may be the first conditions that come to the knowledge of the physician; and he is then inclined to attribute the neurosis to the former alone.

Scarlet fever may cause endocarditis, although much less frequently than rheumatism does, and may leave behind a permanent valvular lesion. Although we must not regard every passing systolic murmur that occurs during the course of scarlet fever as a sign of endocarditis,[1] still it cannot be doubted that it is such when the murmur continues for some time unchanged and is accompanied by a feverish condition. We observe this complication both during the fever itself and in the course of the subsequent nephritis.

Willy K., 5 years old, admitted on 1st February with scarlatina simplex. The fever, which persisted without ascertainable reason during the desquamation temp. (m. 101·3°; ev. 102·9° F.)

---

[1] Any high fever may, as is well known, make the first sound of the heart temporarily prolonged or even blowing. We must also guard against mistaking for a heart-murmur a harsh respiratory murmur which occurs in cases where the breathing is much accelerated.

fell in end of the second week to 101·3° F. in the evening, and the child felt quite well. On the 12th February, a short systolic murmur was heard over the heart for the first time. This became every day more distinct, and was especially loud in the region of the apex, and the pulmonary second sound was somewhat accentuated. Apex-beat and dulness normal. P. 136, somewhat irregular. During the next few days we heard, besides the systolic murmur, a short crackling sound to the left of the sternum, on the level of the third rib, during the height of inspiration; but it was often also synchronous with the systole. The origin of this sound was the less clear to me, because, during the next few days, it was sometimes audible, and sometimes had disappeared. As, however, the temp. again rose in the evening during this time to 103·6° F., I ordered 6 wet-cups to be applied to the præcordium, and gave calomel and digitalis. From the 17th only slight rise of temperature in the evening; pulse normal, the systolic murmur becoming weaker. After the child had gone through an attack of nephritis, with œdema and ascites, the murmur at the apex was still audible on 22nd April; on the 25th it had quite disappeared.

That this was really a case of scarlatinal endocarditis (and, indeed, of slight pericarditis also), is proved by the persistent fever, the rather quick irregular pulse and the systolic murmur, which took two months to disappear entirely. It is to this long duration and slow disappearance of the murmurs that I attach an especial significance, such as cannot be claimed for merely temporary murmurs. Thus, in the course of scarlatinal nephritis I have observed two cases in which there was a mitral systolic murmur, only audible for 24—36 hours. In one of the cases this was associated with irregularity of the pulse, and disappeared without leaving a trace. In other cases, again, there was a reduplication of the first sound or a "galloping rhythm" of the heart-sounds, which lasted some days or even weeks, and then disappeared without leaving a trace. Further, in a case of scarlatinal synovitis of the acromio-clavicular joints ending in suppuration—in which the diagnosis was confirmed post-mortem—there was a systolic murmur which was only heard during the highly febrile onset of the disease, but had ceased to be audible by the following day; and at the post-mortem the valvular apparatus appeared quite normal. On the other hand it cannot be denied that the scarlatinal joint-affection, like the rheumatic, is apt to be associated with inflammatory

processes in the endocardium, less commonly in the pericardium.

Richard Sch., 6 years old, admitted into the ward with scarlet fever on 14th February. Complication with slight bronchial catarrh; heart quite unaffected. On 19th, beginning of desquamation, fever still continuing (ev. 102·6° F.), owing to the presence of bilateral cervical adenitis and of right-sided otitis. On 22nd (T. m. 103·8°; P. 108) we heard over the heart a distinct systolic murmur, especially loud at the level of the 4th costal cartilage and the left border of the sternum; area of dulness normal, apex-beat in the 5th intercostal space abnormally distinct. On the following day, pain in the joints of hands and feet, but no swelling (T. ev. 104·4°; P. 100—104). During the next few days, pains also in the knees, hips, elbows, and shoulders. After 26th, abatement of all the symptoms and disappearance of the murmur. On 1st March, nothing to be made out beyond the ordinary so-called "galloping rhythm." From then till the 25th April (on which day the child was discharged) no abnormality was observed.

Similarly, in a child who was suffering from simple scarlatina, I observed a fresh exacerbation of the fever (to 102·6° F.), with the onset of synovitis in the joints of the hands, fingers, and feet, during the second week of the disease; and, 4 days after, a loud systolic murmur at the apex, which was still present when the child was discharged from the Charité.

That under these circumstances chorea may also set in, I have already mentioned (p. 210), and I have given one of my cases—which, however, does not prove that the latter depends upon the endocarditis. For chorea has also frequently been observed as a result of scarlet fever where there was no synovitis and no heart-disease. In considering this fever I shall again have to speak of scarlatinal endocarditis.

Pericarditis (which on the whole is commoner in children than in adults) may arise from morbid conditions of neighbouring parts by the extension of the inflammatory process from these to the pericardium—especially from left pleurisy, less commonly from right pleurisy, pneumonia and caries of the ribs[1]—as well as from the causes already mentioned (rheumatism, scarlet fever). At the same time there occasionally occurs sero-fibrinous or purulent effusion in the pericardium; but, when the disease is chronic, extensive adhesion of the heart

[1] *Cf.* the cases given on pp. 424 and 426.

to the pericardium is commoner. And this condition is not unfrequently left behind after absorption of the fluid effusion. I have observed purulent pericarditis along with purulent pleurisy, especially in very young children; and in these cases the diagnosis was rendered very difficult, on the one hand by the small amount of pus in the pericardium, and on the other by the extensive dulness caused by pleuritic effusion (p. 424).

> Richard L., 8 months old, admitted into the ward 10th March. Rickets, very rapid, noisy breathing; face distorted with pain on coughing. Over the left side of the chest absolute dulness and bronchial breathing. No displacement of the heart to be found; heart-sounds pure. T. 99·7° F.; P. 140; R. 60. During the next few days the bronchial breathing in front disappeared; the breathing was now no longer audible, and the dulness extended about ⅔ inches beyond the left border of the sternum, although I was unable to make out any distinct displacement of the heart to the right. Exploration by means of a hypodermic syringe on two occasions yielded no result. The temperature almost always remained subnormal (96·8°—99·0° F.); R. 54—60; P. varying much (108—156) extremely small. The increasing collapse prevented any operative procedure. Death on 21st. At the post-mortem we found the whole left pleural cavity filled with purulent effusion. Compression of left lung; fibrino-purulent pericarditis (pericardium not much distended, containing two or three table-spoonfuls of pure pus; both surfaces covered with recent fibrinous lymph).

Endocarditis also may develope under these circumstances. In a girl of 3 to whom I have already alluded (p. 424) I found, besides an old encapsuled pleuritic effusion of the right side, considerable adhesion of the layers of the pericardium, and very marked thickening and incompetence of the mitral valve, with stenosis of the ostium venosum which had even been diagnosed during life. In two other children of 2 and 4 years suffering from extensive broncho-pneumonia of the left lung, there occurred an endocardial systolic murmur which lasted in one case till death, and in the other till after recovery from the lung-affection at least.

Tuberculosis is to be regarded as a frequent cause of pericarditis in childhood. The occurrence of miliary or sub-miliary nodules in the pericardium, especially in its visceral layer, is, according to my experience, certainly not very common in general tuberculosis; but pericarditis with sero-fibrinous or

blood-stained effusion occasionally occurs without these local formations.

Helene W., 21 months old, admitted on 26th May, 1883; anæmic, badly-nourished. At the lower part of the left border of the sternum, a doughy, œdematous swelling, with dilated veins. R. rapid, superficial; much coughing. Numerous râles in both lungs. Heart apparently normal, abdomen distended. T. 103·5° F. Wet compress to the thorax. On 28th a red, fluctuating swelling appeared to the left of the ensiform process, which was opened on 29th, and half-a-pint of thin pus was let out. Drainage and corrosive sublimate dressing. On the 30th, death in a state of collapse.

P.-M.—Close under the ensiform process there was a sinus, which admitted the finger. It had led to great undermining of the abdominal muscles, and extended downwards between the rectus and the obliquus externus abdominis to beneath the umbilicus, and upwards as far as the left costal margin. It here ended in a fistula, which penetrated the diaphragm in the neighbourhood of the ensiform process immediately below the costal margin, and led into a cavity of the size of a hen's egg in the anterior mediastinum. There was another passage leading also into the mediastinum over the costal margin between the 5th and 6th ribs, to the left of the sternum. In the mediastinum there was a completely encapsuled empty abscess-cavity, which communicated above with numerous blind sinuses, and in its thick walls there were numerous tubercles. Ribs and sternum normal. Much sero-fibrinous exudation in the pericardium (cor villosum), occasional tubercles in the serous membrane covering the heart; valves normal. Bronchial glands caseous, in the lower lobe of the left lung a caseous mass the size of a walnut, with numerous miliary tubercles in its neighbourhood.

This case seems to have begun with purulent, tubercular mediastinitis, and this apparently caused on the one hand burrowing of pus between the abdominal muscles, and on the other acute tubercular peritonitis. In the following case we find the mediastinum and pericardium free from tubercle, although acute pericarditis had arisen by extension of inflammation from the left pleura, which was highly tubercular, and had in the end caused extensive adhesion of the pericardium. Such adhesions sometimes contain firm fibrinous matters, which are partly caseous and partly studded with tubercle.

Paul M., 8 years old, admitted into the hospital on 20th May, 1878. Formerly healthy. Said to have been feverish and out of

sorts for the last 8 days. Very pale. R. 36; T. 101·5°; P. 136. In the region of the heart and for ⅘ inches beyond the right border of the sternum, loud friction accompanying both sounds was to be heard. Percussion normal; apex-beat not distinctly felt. 8 dry-cups, ice-bag, digitalis. During the next few days the patient complained much of sharp pains in the region of the heart. R. rising to 60; T. to 103·1° F. By 24th, the friction had disappeared, and the cardiac dulness now reached upwards as far as the 3rd rib, and ⅘ inches to the right of the sternal margin. Pulse very small. A blister to the region of the heart; calomel gr. ½ every 2 hours; after the 28th pot. iod. grs. ii. The temperature now gradually sank, only temporarily rising again to 102·7° in the first days of June, when an attack of catarrh raised the respirations again to 60. The pulse, however, gained in strength, and, although there was no change in the percussion, we again felt a weak diffuse apex-beat on 6th June, and also heard both the heart-sounds quite pure, although weak. On 13th June we could again hear distinct friction accompanying both sounds (R. 50—60; P. 132—156), and the dulness no longer reached the right border of the sternum, while on the left side it did not extend to the mammillary line. On 29th, the friction was still audible over the upper part of the sternum, while the sounds seemed pure lower down. T. in the morning normal, in the evening still 100·9° F.; R. 28—32. On 6th July nothing was left but very faint friction over the sternum, everything else normal, and so the boy was discharged as cured on 7th August. In October he was again brought to the clinique on account of considerable ascites. The description of this phase of the case will be given under Chronic Tubercular Peritonitis—for that was what was the matter. I may only mention here that during the whole period of his residence in hospital, up to 5th May, 1879, not the slightest abnormality could be discovered in the heart, in spite of frequently repeated examination. Of the conditions found at the post-mortem, I shall only mention those which are interesting in this connection.

The whole left pleura costalis thickly-studded with tubercles, the pleura pulmonalis less affected. The pleural cavities empty. The cavity of the pericardium entirely obliterated, by the complete adhesion of its two layers to one another, and the heart covered all over by thick fibrous tissue. On careful examination we found the muscular substance at different parts of the anterior wall of the right ventricle almost entirely converted into fibrous tissue. Pericardium and heart quite free from tubercle. Valvular apparatus perfectly unaffected. The anterior mediastinum very œdematous and thickened. Also tubercular peritonitis and meningitis.

In this case we find, as we often do, adhesion of the entire

pericardium, producing no symptoms whatever; in particular no systolic in-drawing of the chest wall was observed in any situation. The implication of the myocardium, at least that of the right ventricle, took the form in this case not only of peripheral fatty degeneration—which is common in pericarditis—but of interstitial myocarditis with formation of fibrous tissue; and this is but rarely observed in children, and could no more have been discovered clinically than could the adhesion of the pericardium. Quite similar to this was the case of a boy of 6, who died after measles with symptoms of chronic tubercular peritonitis, and at whose post-mortem we found, besides this, tuberculosis of the pleura, lungs and liver, and complete adhesion of the pericardium. This formed two fibrous layers studded with tubercles, between which there were some partially-softened caseous nodules. Here also nothing abnormal was discovered in the heart during life; nor yet in the following case—which, however, had nothing to do with tuberculosis.

Richard L., 5 years old, admitted on 4th February. Scarlet fever 2 years ago; said to have been only 14 days ill (?). Much coughing and dyspnœa. Pallor and emaciation, well marked œdema of the face and lower extremities (circumference of abdomen 28 inches). Liver-margin hard, extending 3 finger-breadths below the costal margin. The intercostal spaces on the right side of the thorax were expanded, bulging out somewhat; circumference 12¼ inches, that of the left only 9½. Percussion dull all over the right side; bronchial breathing and bronchophony, here and there rather sharp râles; catarrh on the left side above. Size of the heart normal, sounds pure but weak; no indrawing with the systole. Instead of the apex-beat, a more diffuse impulse. Urine scanty, 10½ oz. daily, normal. Digestion good, no fever. P. 120, regular. On the 7th, after exploratory puncture, half-a-pint of clear serum removed from the 5th right intercostal space by means of Dieulafoy's aspirator. This serum contained extremely little albumen. After the 15th, fever (101·5° F.), restlessness, great dyspnœa. Death under chloroform before the second puncture.

P.-M.—In the abdomen half-a-pint of serum; the right pleural cavity likewise quite full. The right lung the size of a man's fist, solid. Also in the left side of the chest about half-a-pint of serum. Pericardium adherent all over; in the fibrous adhesions extensive disseminated dry yellow masses. Right ventricle small, with very thin walls, which are fibrous in many places. Fibrous thickening of the pleura all over. Spleen very large. Liver enlarged, its surface uneven, the capsule slightly thickened, with

many fibrous bands passing through it; anæmic. In the jejunum 2 little ulcers about ½ inch in diameter. Kidneys indurated, large, smooth.

In this case we found nothing of the nature of tubercle in any organ, for the yellow nodules (fatty and amorphous débris) in the fibrous tissue of the adherent pericardium could not be set down as tubercular without further proof. Although there was nothing in the history which could be held to support the assumption of a syphilitic origin (suggested by the fibrous degeneration of many organs and especially of the liver), still, the whole anatomy of the case is such that it may almost with certainty be regarded as one of syphilitic pericarditis and myocarditis with the formation of gummata in the fibrous tissue of the pericardium—examples of which are very rare in childhood.[1]

Apart from these cases I have only very rarely seen myocarditic processes in children:—*e.g.* in a boy of 10 years.

Pericardial cavity very large, containing half-a-tablespoonful of blood-stained fluid. Heart very large. Walls of both ventricles dense; slight fibrous thickening of the epicardium all over, and in some places papillated. At the apex of the left ventricle there was, besides very extensive atrophy, an aneurismal dilatation the size of a hazel-nut, at which point the wall of the heart was scarcely ⅛ inch thick. Endocardium of the left auricle much thickened. The mitral and aortic valves shrivelled and nodular.

During life, in this case, we could only make out the symptoms of valvular disease and hypertrophy of the heart. From an anatomical point of view, however, the case is so far interesting, that it adds one more to the small number of cases of aneurism of the heart which have hitherto been observed in children. Owing to a localised chronic myocarditis, which had developed along with endocarditis and inflammation of the epicardium, the affected portion of the muscular tissue had gradually degenerated into a thin layer of fibrous tissue, which was rendered thinner and thinner by the blood-pressure. Extensive chronic fatty degeneration of the heart muscle, such as so often occurs in adults, either with or without degeneration of the coronary arteries, I have never as yet seen even once in

[1] *Cf.* Von Dusch in *Gerhardt's Handbuch der Kinderkrankh.*, iv., S. 298.

childhood. I have, however, on several occasions observed a localised fatty degeneration—especially in the right ventricle—in children with prolonged whooping-cough and chronic pneumonia (p. 461). In such cases it arises as the result of the resistance which the heart has to overcome in the pulmonary circulation; and it may occasion passive dilatation of the cavities, and death by syncope. To the same class also belongs the localised fatty degeneration, which takes place towards the end in hypertrophied hearts in cases of valvular disease. When I come to consider the infectious diseases, I shall return to the fatty albuminous degeneration of the heart-muscle which occurs pretty often after acute infectious disease—especially scarlet fever, diphtheria, and typhoid—and clinically gives rise to no symptoms except perhaps those of cardiac debility.

I have also but rarely met with simple hypertrophy and dilatation of the heart in children—twice following chronic nephritis, and twice in very young children whose hearts had probably been too large from birth. In other cases some affection of the valvular apparatus was generally to be found as the cause of the hypertrophy and dilatation. In a few cases also a recent attack of nephritis (very specially of scarlatinal nephritis) was the cause; but of this I shall speak again under scarlet fever. Of the acute form of dilatation of the heart described by Steffen and others, the diagnosis of which rests only on percussion, and which is said to come on in endocarditis (as the result of engorgement and overwork of the heart), as well as in infectious diseases—I think I have myself observed a few cases. The most distinct of these was one which I shall give by-and-by under scarlet fever. I must, however, admit that in regard to the majority of these cases I feel a little doubtful, owing to the rapid recovery from the dilatation within a few days. We must always bear in mind how many causes of error in the percussion of the præcordium may arise from the varying degree of expansion of the left lung, and from the restlessness and screaming of little children. The mere theorist may imagine that in determining the limits of the heart in childhood, he can arrive at results with mathematical exactness; but whoever has any considerable experience can only smile at such an idea. Further, we have the fact that even in the dead body the different degrees of contraction and fulness of the heart, as well

as the difference of age, may leave it doubtful—in the less well-marked cases—whether the case is one of hypertrophy or of dilatation. The old comparison with the fist of the individual—although it is generally sufficient for practice—is yet by no means satisfactory for scientific purposes.[1]

In addition to this consideration we must not omit to mention that many children (and, according to my experience, more boys than girls), from the age of 10 up to puberty, complain of palpitation of the heart, and of shooting pains in the cardiac region; and they also occasionally complain of shortness of breath, and especially of headache. I have only been able to discover anæmia in some of the cases, and on examination I have almost always found nothing but a diffuse heaving cardiac impulse without any change in the normal signs of percussion and auscultation. As a matter of fact I have never seen anything bad happen in such cases, as far as I was able to follow them. Indeed, the heart-symptoms gradually got less and disappeared, and one might have accepted the view—which used often to be expressed—that there was taking place a gradual adjustment of the relations which had existed physiologically during childhood between the heart and the body-weight—if it were not that according to Beneke's researches the heart is relatively smallest in the years just before puberty, and then rapidly increases while puberty is in process of development.

The treatment of heart-disease is the same in children as in adults. Although the chronic forms (valvular disease followed by hypertrophy) are generally better borne by children owing to the rarity in them of myocarditic processes and of disease of the vessels, and the patients live to puberty, or even longer, before any serious disturbance of compensation sets in; still, cases do occur in which active medical treatment becomes necessary on account of distressing symptoms. I have been struck by the commonness of anæmia in these children, and by treating this symptom suitably with preparations of iron (Form. 12) I have often succeeded in relieving at least some of the symptoms (palpitation, debility), and considerably improving the general condition, although the heart-disease itself remained unaffected thereby. The iron never had any bad effects in these cases.

[1] *Cf.* Beneke, *Die anatom. Grundlagen der Constitutionsanomalien.*: Marburg, 1878.—Von Dusch, *Gerhardt's Handb. d. Kinderkrankh.*, iv., S. 267.

The treatment of acute (inflammatory) heart-affections must, of course, be antiphlogistic. We require local blood-letting, ice-bags, calomel, digitalis, and blisters. The cases which I have given will best illustrate to you the action of these remedies. In pericarditis, with an extremely large amount of effusion, the imminent danger may be averted by puncture and aspiration of the pericardium, or else by incision. Examples of this have been published,[1] but I myself have no personal experience of the matter.

[1] Cadet reports 9 cases with 5 recoveries; Rosenstein (*Berl. klin. Wochenschr.*, 1881, No. 5), after puncturing unsuccessfully, incised the pericardium, inserted a drain and procured recovery in 10 days; Gussenbauer also (*Revue mens.*, Janv., 1885, p. 37), West (*Jahrb. f. Kinderheilk.*, xx., 462), and others have reported cases successfully treated by incision and drainage.

END OF VOL. I.

LONDON:
Printed by Jas. Truscott & Son,
Suffolk Lane, E.C.

Date Due

Demco 293-5			

RJ
88
1

