TEXT-BOOK
OF
PEDIATRICS

EDITED BY
PROFESSOR E. FEER
DIRECTOR OF THE UNIVERSITY CHILDREN'S CLINIC, ZURICH

TRANSLATED AND EDITED BY
JULIUS PARKER SEDGWICK, B.S., M.D.
PROFESSOR OF PEDIATRICS, UNIVERSITY OF MINNESOTA, MEDICAL SCHOOL

AND
CARL AHRENDT SCHERER, M.D., F.A.C.P.
DULUTH, MINNESOTA

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PREFACE

THE introduction of this work to the physician and student is made easy by the widespread and favorable acquaintance it has achieved among those who have studied on the Continent or have read it in the original. The preface to the first edition sets forth the value of a text produced by the collaboration of a number of authors, each a master of his branch of the specialty. The plan of collaboration has been carried on in the translation. Up to the present no similar one volume work has appeared upon the market.

A further distinct advantage is the concise treatment of the subject matter. The arrangement is such that no time is lost in referring to any one descriptive passage. The etiology, pathology, symptomatology and treatment are all complete, but as short as compatible with their purpose. The book covers the entire field of pediatrics as completely as more bulky volumes. The discussions of the individual disease conditions are absolutely dependable and the therapeutic measures advised are in line with the most recent accepted usage.

Due to the interest of the American collaborators much has been added to the original subject matter and, through the kindness of numerous friends, who know the book in the original, a large number of illustrations have also been added. The editors take this opportunity to thank their associates, by whose help we were enabled to bring this work to completion, for their keen interest and their painstaking study of the sections revised by them. We also wish to thank Dr. R. O. Beard, Secretary of the Medical School of the University of Minnesota, for his careful revision of the English. To J. B. Lippincott Company we feel especially indebted for their patience and their appreciation of the problems of the translation.

May the volume find as great a sphere of usefulness in this translation as it has had in the original.

J. P. S.
C. A. S.

November 1st, 1922.

PREFACE TO FIRST GERMAN EDITION

A LARGE number of medical text-books, the products of the collaboration of a number of authors, have recently appeared. The great favor with which these works are received is the best indication of their value. When, therefore, I was approached in 1909 by Mr. Gustaf Fisher with the suggestion that I edit a work of this nature, as a companion piece to the text on *Internal Medicine* by Krehl-Mering, I had no hesitancy especially since pediatrics is no longer a subject in which one author can have complete knowledge of all branches. I was able to interest a number of the most prominent pediatrists who were competent to write on the special subjects assigned to them. I believe that the work of these associates and the new method of presenting the subject in this volume with its numerous apt illustrations justify its production in spite of the great number of text-books on pediatrics already on the market.

In order to give the student and general practitioner an introduction into the subject and an understanding of the child itself, it has been thought fit to make the general part very extensive. In the special part, those diseases not peculiar to childhood and fully described in text-books of general medicine were merely touched upon, in order to give space to the diseases that present definite peculiarities in childhood or occur only in children. A certain amount of duplication must of course occur, as for instance in those diseases classed as acute infections or "children's diseases." These are all considered in texts on general medicine, but belong specifically to this work. Special pains were taken to give full space to those diseases of childhood that are barely touched upon in general texts, but are of great importance to the pediatrist. Thus the subjects of varicella, pertussis and measles, given only one, two and one pages respectively in the Krehl-Mering text, have been accorded seven, ten and two and a half pages in this book.

More space than is customary is devoted to early infancy. The disturbances of nutrition are discussed by the most able authors in accordance with most recent research. Those physicians who are accustomed to the old classification of dyspepsia, catarrh and enteritis, may be somewhat confused at first. The discussion of the disturbance of nutrition is based upon the study of the nutritional processes in the light of functional tests. These have led to a new classification and a more rational treatment. The older classification, though simple, is actually of little value as far as treatment is concerned. Those, however, who have already acquired this more recent view as, no doubt, all undergraduates have, will gladly recognize its great advances and will take advantage, clinically, of the benefits afforded by it in the treatment of the disturbances of nutrition, a most difficult phase of the practice of pediatrics.

As far as the use of varied sizes of print is concerned, the small print is used for introductions and notes of a more or less general nature, not

having direct bearing upon the subject under discussion, but essential for its understanding and to be especially brought to the reader's notice.

The excellent ensemble of the work is due to the interest and painstaking care of the publisher who has spared no means to produce the best at a reasonable price. His efforts have further made it possible to obtain new illustrations that are most applicable to the text.

May this new text-book be a reliable guide and advisor to both the undergraduate and practicing physician.

E. Feer.

PREFACE TO SEVENTH GERMAN EDITION

The demand for a new edition, so soon after the appearance of the last, has necessitated but few changes. Nevertheless, all the authors have revised their sections and made additions and improvements. Special pains have been taken to enlarge upon the therapy. By more concise handling of several sections, it has been possible to reduce the size of the volume by twenty-four pages.

In place of our late colleague, Professor Tobler, Professor Noeggerath consented to take on a portion of the work and has completely revised the section on genito-urinary diseases, bringing out the advances made during the last few years in the study of the nephropathies.

Furthermore, we must again report the loss of a colleague, Professor Martin Thiemich, who died February 16, 1921, following a long illness, at the age of fifty-one. This was a great loss not only to our text-book but also to the science of pediatrics and the University of Leipzig. His last work was the correction and revision of his section of this text.

E. Feer.

TEXT-BOOK OF PEDIATRICS

EUROPEAN CONTRIBUTORS

Feer, E., Professor Dr., Zurich.
Finkelstein, H., Professor Dr., Berlin.
Ibrahim, J., Professor Dr., Jena.
Meyer, L. F., Priv.-Doz. Dr., Berlin.
Moro, E., Professor Dr., Heidelberg.
Noeggerath, C., Professor Dr., Freiburg i Br.
Thiemich, M., Professor Dr., Leipzig.
v. Pfaundler, M., Professor Dr., Munich.
v. Pirquet, Cl. Frh., Professor Dr., Vienna.

AMERICAN COLLABORATORS

Brennemann, Joseph, M.D., Chicago, Ill.
Attending Pediatrist, Children's Hospital, Chicago.

Byfield, Albert H., M.D., Iowa City, Ia.
Professor of Pediatrics, College of Medicine, State University of Iowa.

Calhoun, Henrietta M.A., M.D., Iowa City, Ia.
Assistant Professor of Pathology, College of Medicine, State University of Iowa.

De Buys, L. R., M.D., F.A.P.C., New Orleans, La.
Professor of Diseases of Children, School of Medicine, Tulane University of Louisiana; Chief of Pediatrics, Turo Infirmary.

Dietrich, Henry, M.D., Los Angeles, California.
Attending Pediatrist, Children's Hospital, Los Angeles.

Fleischner, E. C., M.D., San Francisco, Calif.
Clinical Professor of Pediatrics, University of California.

Hess, Julius H., M.D., Chicago, Ill.
Professor and Head of the Department of Pediatrics, University of Illinois, College of Medicine; Chief of Pediatric Staff, Cook County Hospital.

Hoffmann, Walter H. O., M.D., Chicago, Ill.
Associate Attending Pediatrist, Children's Memorial Hospital, Chicago.

Hoobler, B. Raymond, M.D., Detroit, Mich.
Professor of Pediatrics, Detroit College of Medicine and Surgery.

Irvine, Harry Garfield, M.D., Minneapolis, Minn.
Associate Professor of Dermatology and Syphilis, Medical School, University of Minnesota; Director of the Division of Venereal Diseases, Minnesota State Board of Health.

Jeans, Philip C., St. Louis, Mo.
Associate Professor of Pediatrics, Washington University School of Medicine.

Meyer, K. F., M.D., San Francisco, Calif.
Professor of Research Medicine, University of California.

Ott, M. D., M.D., Minneapolis, Minn.
Associate in Pediatrics, University of Minnesota, Medical School.

Pierce, Naboth Osborne, M.D., Minneapolis, Minn.
Assistant Professor of Pediatrics, University of Minnesota, Medical School.

Scammon, Richard Everingham, M.D., Minneapolis, Minn.
Professor of Anatomy, University of Minnesota, Medical School.

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PROFESSOR DR. MARTIN THEIMICH,

Director of the University Children's Clinic, Leipzig.

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PROFESSOR DR. MED. AND PHIL. HEINRICH FINKELSTEIN,

Director of the Kinderkrankenhaus, Berlin,

and

PRIVATDOZENT DR. LUDWIG MEYER,

Chief Pediatrist to the Orphanage and Children's Asylum of the City of Berlin.

Revised and edited by

NABOTH OSBORNE PIERCE, M. D., Minneapolis, Minn.,

Assistant Professor of Pediatrics, University of Minnesota, Medical School.

SECTION II. PATHOLOGICAL CHANGES OF THE BLOOD AND BLOOD-FORMING ORGANS

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by

Professor Dr. Meinhard von Pfaundler,
Director of the University Children's Clinic, Munich.

Revised and Edited by

M. D. Ott M. D., Minneapolis, Minn.,
Associate in Pediatrics, University of Minnesota, Medical School.

SECTION III. DISEASES OF THE DIGESTIVE SYSTEM

by

Professor Dr. Med. and Phil. Heinrich Finkelstein,
Director of the Kinderkrankenhaus, Berlin.
and
Privatdozent Dr. Ludwig Meyer,
Chief Pediatrist to the Orphanage and Children's Asylum of the City of Berlin.
Revised and Edited by
Joseph Brennemann, M. D., Chicago, Ill.,
Attending Pediatrist, Children's Memorial Hospital, Chicago.

SECTION IV. DISEASES OF THE RESPIRATORY ORGANS

by

Professor Dr. Clemens Freiherr von Pirquet,
Director of the University Children's Clinic, Vienna.
Revised and Edited by
Walter H. O. Hoffmann, M. D., Chicago, Ill.,
Associate Attending Pediatrist, Children's Memorial Hospital, Chicago.

SECTION V.—DISEASES OF THE HEART

by

Professor Dr. E. Feer,

Director of the University Children's Clinic, Zurich, Switzerland.

Revised and Edited by

Henrietta Calhoun, M. A., M. D., Iowa City, Iowa,

Assistant Professor of Pathology, College of Medicine, State University of Iowa.

SECTION VI.—DISEASES OF THE GENITO-URINARY TRACT

by

Professor Dr. C. Noeggerath,

Director of the University Children's Clinic, Freiburg in Breisgau.

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by

Professor Dr. Jussuf Ibrahim,

Director of the University Children's Clinic, Jena.

SECTION VIII. THE ACUTE INFECTIOUS DISEASES

by

Professor Dr. E. Feer,

Director of the University Children's Clinic, Zurich.

Revised and Edited by

E. C. Fleischner, M. D., San Francisco, Calif.,

Clinical Professor of Pediatrics, University of California,

and

K. F. Meyer, M. D., San Francisco, Calif.,

Professor of Research Medicine, University of California.

SECTION IX. TUBERCULOSIS

by

Professor Dr. Clemens Freiherr von Pirquet,

Director of the University Children's Clinic, Vienna.

Revised and Edited by

Henry Dietrich, M. D., Los Angeles, Calif.

Attending Pediatrician, Children's Hospital, Los Angeles.

SECTION X. SYPHILIS

by

Professor Dr. Ernst Moro,

Director of the University Children's Clinic, Heidelberg.

Revised and Edited by

Philip C. Jeans, M. D., St. Louis, Mo.,

Associate Professor of Pediatrics, Washington University School of Medicine.

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SECTION XI. DISEASES OF THE SKIN

by

PROFESSOR DR. ERNST MORO,
Director of the University Children's Clinic, Heidelberg.

Revised and Edited by

HARRY GARFIELD IRVINE, M. D., Minneapolis, Minn.

SUPPLEMENT

GENERAL CONSIDERATIONS

BY

MARTIN THIEMICH, LEIPZIG.

I. ANATOMIC AND PHYSIOLOGIC PECULIARITIES

REVISED BY

Dr. RICHARD EVERINGHAM SCAMMON,
Professor of Anatomy, University of Minnesota Medical School, Minneapolis.

THE new-born infant is, by no means, to be considered a miniature of the adult. While obvious differences are shown externally in the relatively large head and small face, the short extremities, the notable arching of the thoracic walls, the undeveloped genitalia, etc; numerous other differences in anatomic and histologic structure and in the physiologic functions of the various organs and systems of organs are discoverable by careful study. Certain of these, of especial interest to the physician, will be described. Those which are merely of anatomic interest, or useful in the study of cases which come to autopsy will be specifically noted in Chapter III.

The body of the child contains more water than that of the adult; the largest percentage is found in the fetus; the proportion decreases rapidly toward puberty. The body of the new-born contains about 25 per cent. of solids and that of the adult about 33 per cent. Under normal conditions, an increased water content of the infant body causes a correspondingly increased turgor or sense of resistance of the skin and subcutaneous tissues.

Among the differences described in the chemical composition of the entire organism, special attention may be called to one item. In the last months of fetal life, a considerable deposit of iron salts is gathered in the liver, enabling the infant to exist for a varying length of time upon a diet as poor in iron as mother's or cow's milk, without lack of this essential material for the purpose of blood metabolism.

The Anatomic and Hemodynamic Relations of the Heart.—These show great departures from adult life. The absolute weight of the heart in the new-born averages about 20-25 grams, about one-twelfth the adult weight of the organ. At birth the organ forms about 0.7 per cent. of the body-weight compared to about 0.4 to 0.6 per cent. of the body-weight in the nursling. The relative or percentage weight of the heart usually falls to about 0.5 per cent. during the first year. The weights of the musculature of the right and the left ventricle is usually about equal at birth, but the left ventricle is double the weight of the right by the close of the first six months. The wall of the left ventricle is only slightly thicker than that of the right; and the heart, with its large, wide ventricles, has a much lower resistance to work

against because of the relatively large lumen and the larger sectional area of the arteries. This is shown by the systolic pressure, which is 80-90 mm. of mercury in the infant and 110-120 mm. in the adult. The pulse is more frequent, ranging from 134 during the first year of life, and gradually decreasing in rate to about 90 during the eighth or ninth years. It is more elastic and compressible. The complete circuit of the blood is more rapid than in the adult. When one considers that neither the heart muscle nor the arterial walls have been injured by the insidious poisoning of tobacco and alcohol, or by chronic and recurring infections, and that arteriosclerosis is a condition almost unknown in childhood, it may be readily understood that the vascular system can withstand the severest demands upon it and can compensate serious obstructions to the circulation for a long time. However, for a time, during the development of puberty, the heart does not keep pace with the rapid growth of the body and changes in its anatomic relations may cause functional lesions or cardiac insufficiencies.

During the first few days of life, the period in which the physiologic loss of weight, due to the output of large quantities of fluid occurs, the blood has a relatively higher percentage of hæmoglobin, an increased number of cells and a higher specific gravity. This concentration disappears during the first month, and after that a very gradual decrease in the percentage of hæmoglobin, in the relative number of erythocytes and in the slight leucocytosis continues, until, by the end of the second year, the blood of the child is the same as that of the adult. It may be questioned whether the concentration of the blood through the loss of fluids from the body is the main cause of the natal leucocytosis since the number of cells drops rapidly in the second and third days after birth while the body is still losing in weight. A slight secondary rise in the white cell count is often noted in the second week coincident with the detachment of the umbilical cord. In general the total leucocyte count after this time is little higher in the infant than in the adult. The lymphocytes form from 30 to 40 per cent. of all leucocytes in the first year and the polymorphonuclear neutrophiles form 50 to 60 per cent. From this time on the neutrophiles increase and the lymphocytes decrease in relative numbers. Their percentages are about equal (at about 45 per cent.) at five to six years. The counts of eosinophilic and basophilic leucocytes and of transitional cells are about the same in infancy, childhood and maturity. The blood does not assume its normal adult picture until about the time of puberty. During the years of infancy, however, the leucocytes remain slightly increased and a peculiarity in the percentage of their various forms is maintained to the end of childhood; the lymphocytes representing 50 per cent., while, later, they decrease to about 25 per cent. During the first two weeks of life, the normal hæmoglobin content is about 36 per cent. greater than in the adult. From these high values, the percentage begins to diminish at once and, after two weeks, the fall is very rapid. By the fifth month the value reaches very nearly the minimum and is far below the value of adult life.

The respiratory apparatus and its mechanics present important differ-

ences. The thorax of the new-born, with its high arch and its lesser length, is always in the phase of almost extreme inspiration, the ribs forming nearly a right angle with the vertebral column. As a result, the breathing is almost entirely abdominal or diaphragmatic. To compensate for the shallowness of the respiration, its frequency is increased and every added demand upon the respiratory function of pathologic origin produces an increase in the number of respirations. As the child grows older and is not continuously recumbent, the upright position causes a gradual change in the form of the chest which makes thoracic breathing possible. The weight of the abdominal organs and of the thoracic walls in the vertical position tend to draw down the anterior wall of the thorax. The larger air passages, larynx, trachea and bronchi, are also affected by the downward stress. The ribs, which were initially transverse to the vertebral column, not only take an obliquely downward position but also change shape by the formation of a distinct angle, which increases the thoracic space and gives more room for the lungs, both anteroposteriorly and laterally. The ultimate results of these anatomic changes upon functional activity are seen in the gradual assumption of the mixed type of breathing (thoracic and diaphragmatic) in the increased volume of the respiration and in the reduced frequency of its rhythm.

These changes are completed at about the end of the first year, at which time the number of the respirations has been reduced from 40 or 45 per minute, at birth, to 25. The respiratory volume ranges from 27 to 42 c.c. during the first six months of life; while it increases to 78 c.c. during the second six months and to 135 c.c. by the end of the first year (Gregor).

Later, the number of respirations is reduced very gradually, so that the average, during rest and sleep, at two years of age, is about 24 per minute, at five years about 20 per minute, and at ten years about 18 per minute. The individual respirations grow deeper, the mechanism works more economically and is readily able to overcome temporary demands for increased activity by its greater elasticity, indicated both in frequency and volume.

Attention should also be called to the fact that the respiratory rhythm, during the first months of life, and, at times, even up to the third year, is not always regular. In the young infant, pauses of varying length may occur (Czerny). The differences in respiration, dependent upon sex, the thoracic type of the female and the abdominal type of the male, do not make their appearance until after the tenth year. More or less permanent malformations of the thoracic wall may occur in infants as a result of pathologic conditions, such as forced respiration and an abnormal softness of the ribs. These may be due in part to an incurvation of the thoracic wall at the attachment of the diaphragm.

THE PHYSIOLOGY OF NUTRITION

The knowledge of the physiology of nutrition is of great importance in the understanding of the pathology of childhood. Because milk is the chief article of diet, during the first year we incline to classify the descrip-

tion of the digestive processes according to the kinds of milk in common use. Practically, it is necessary to consider, in addition to the human milk, only that of the cow and goat. Asses' milk, with its very low fat content, is hard to get and not indispensable.

In the following table[1] the more important constituents and peculiarities of woman's, cow's and goat's milk are arranged for ready comparison and as a basis for discussion.

PERCENTAGE COMPOSITION AND PECULIARITIES OF MILK.

	Woman's	Cow's	Goat's
Water	87	88	87
Solids	13	12	13
Total nitrogen	0.15–0.30	0.55	0.56
Nitrogen in protein	0.12–0.17	0.5	0.43
Total protein	1.0 –1.5	3.0–4.0	3.5
Caseinogen	0.6 –1.0	3.0	3.8
Lactalbumin and globulin	0.5	0.3	1.2
Lactose	7.0	4.0–4.5	4.4
Fat	4.0 (1.3–9.0)	3.0–4.0	4.0
Total ash	0.14–0.28	0.7	0.7–1.0
Calcium oxide	0.03	0.2	0.2
Phosphorus pentoxide	0.05	0.24	0.28
Iron oxide	0.0005	0.001*	0.003
Chlorine	0.043	0.1	0.1
Heat value (calories per litre)	650–750	650–750	
Reaction to litmus	alkalin	amphoteric	amphoteric
Combining power (acidity), per litre with blue litmus in N/10 acid.	85	320–550	

* According to more recent estimations, only a part of this iron is actually a constituent of cow's milk. The greater part comes from the utensils of transportation, etc.

The total nitrogen consists largely of the nitrogen of the caseinogen, lactalbumins and lactoglobulins. A small fraction is found in ammonia and in extractives, which are probably excretion products of the lacteal gland; some may also be found in the questionable lactomucins. The caseinogen, or more properly the caseinogens, because we have to deal with different substances in the various kinds of milk, are acid protein bodies containing phosphorus. They are insoluble in water, but dissolve in acids, bases and salt solutions; and are held in solution or in an ultramicroscopically fine colloidal suspension in the milk by alkalis or, more properly, by alkalin earths. The lactalbumins and lactoglobulins are usually called soluble proteins, in contradistinction to the caseinogen which is called an insoluble protein. Clinically, great stress has been laid for a long while upon this difference, because the less digestible cow's milk actually and relatively contains more caseinogen than human milk. At first, it appeared that the greater digestibility of human milk was due to the presence of larger amounts

[1] This table, with a few minor changes, is taken from the chapter on milk by Raudnitz, in Pfaundler and Schlossmann's *Treatise of Pediatrics*, 2nd Edition, 1910, Vol. 1., pp. 133.

of the soluble protein, as well as to the demonstrated differences in the two caseins. When coagulated by acid or by the action of ferments, to the operation of which the presence of calcium salts is necessary, the caseinogen of cow's milk forms a more solid and a coarser curd than that of human milk; and, upon digestion *in vitro* with pepsin and hydrochloric acid, leaves a residue of "pseudonuclein," sparingly soluble and digested only after a long time and with great difficulty. This is not found in human milk. The facts, later to be discussed more fully, that the direct absorption of lactalbumin and lactoglobulin, formerly accepted as a fact, has been proved an error, and that the appearance in the intestine of this undigested "pseudonuclein" has no pathogenic significance, have limited the value of these findings.

Milk-sugar, of which human milk contains a larger percentage than either cow's or goat's milk, is chemically the same substance in the three varieties.

This is not true of the milk fats which represent complicated mixtures of various glycerin esters and free fatty acids. They are inconstant in their composition and are dependent, to a certain extent, upon the fats of the food digested by the milk-producer. Cow's milk contains, in round numbers, four times as much volatile fatty acid as does human milk.

The fat content (see table) shows greater minimal and maximal variations than any other constituent. Apart from individual differences, found alike in human and in animal milk, we note that in both the first portion of the milk extracted from the organ contains a smaller percentage of fat than does the later output, and that the fat content increases proportionately and gradually as the gland is emptied; showing the most gradual increase in breasts which secrete large quantities. The average percentage of fat content is therefore smaller in milk obtained from a freely secreting organ than from one which secretes less.

The various mineral constituents shown in the ash receive much attention at present. In part, these constituents are found in the organic components of the milk and especially in the protein bodies, in the molecules of which they are incorporated with greater or less stability; and, in part, they are found as certain preformed salts in diffusible and more or less ionized state in solution in the whey.

The quantity of mineral constituents is much greater in animal milk than in human milk, corresponding to the greater demand which the more rapid growth of the young animal makes. This is especially true of calcium and phosphorus, the two important inorganic constituents of bone. It is interesting to note that both human and animal milk are comparatively poor in chlorine. The mineral content of the ash of human milk has not the same relation to the body-ash of the new-born, as von Bunge has found to be true of the milk and body-ash of several very rapidly growing animals. It is adequate, however, with probably the single exception of its iron content, to the normal nutritive demands of growth and repair and of functional development in all the infantile organs.

The reaction of fresh milk to litmus is amphoteric or alkalin. Upon

standing, the ensuing bacterial action ferments the milk-sugar and produces acid. This is of great importance in milk intended for infant feeding and will, therefore, be discussed further in the chapter upon that subject.

The tendency of cow's milk to sour is twice as great as that of human milk. This is of major importance in the process of gastric digestion, for with cow's milk a much greater proportion of the hydrochloric acid secreted by the stomach is changed into combined form and the appearance of free hydrochloric acid may be greatly delayed or may entirely fail. While acid cells are found in the gastric glands both of the fetus and the new-born it is approximately two years before they reach their full development. The gastric mucosa of the infant is relatively thick and the muscular coat relatively thin, although all the layers of the latter are present. The elastic tissue of the stomach is limited to the walls of the arteries of the organ for some time after birth.

To the constant constituents of milk belong, in varying quantity, various ferments and certain immune bodies. The former have long been considered important to the process of digestion in the gastro-intestinal tract, while the latter have been supposed to be important factors in the development of the high grades of immunity which appear with the use of certain kinds of food. This view, on first consideration a very essential one, and chiefly because the usual practice of boiling milk for a short time, kills these ferments and immune bodies, but without affecting the food-value of the milk, does not seem tenable today.

Neither human milk nor cow's milk has the same composition at the beginning of lactation as it has when the function is fully established, save for relatively minor changes, it then becomes constant and remains so throughout the period.

The colostrum is the initial secretion of the functionating mammary gland; it is, at first, small in quantity, but gradually increases. It is a yellow fluid, viscid because of its high protein and globulin content, and coagulates upon heating. It contains about 3 to 5 per cent. of milk sugar; its fat content varies within wide limits. The fat is not chemically identical with the fat of the later milk of the same animal. In the woman, the transition from colostrum to milk is normally complete by the end of the first week.

The best evidence of the colostral condition of the milk is the discovery of colostral corpuscles (see Fig. 1) that is, of leucocytes loaded with coarse and fine fat droplets, which are, at first, very numerous in each microscopic field, but later are few and require careful search. Czerny has shown that they are leucocytes and, according to more recent investigation upon the human subject, are lymphocytes which take care of the unchanged, non-absorbable fat, present in the temporary hypersecretion of the mammary gland, by emulsifying it and removing it through the lymph channels, They are found whenever there is congestion of the gland and to this fact attaches their clinical interest.

Since the milk of animals is not used directly from the udder, but is usually pasteurized or sterilized and is variably diluted for infant use, it is

necessary to consider, from the viewpoint of clinical interest, the physical and chemical changes which follow.

Dilution produces a slower coagulation and a finer curd. Heating, and the effect is the same whether milk be heated to 70-80° C. (150°-180° F.), for a long time, or brought to the boiling point for a short time—causes a partial precipitation of the phosphates or alkalin earths and the formation of insoluble tricalcium citrate, which delays clotting, a process dependent upon the presence of soluble calcium salts, and thus forms a finer curd. Lactalbumin is partially coagulated at 55° C. (140° F.) but is not completely precipitated by boiling, a part being held in solution by the caseinogen and alkalin salts. A portion of the caseinogen is dissociated into casein and its

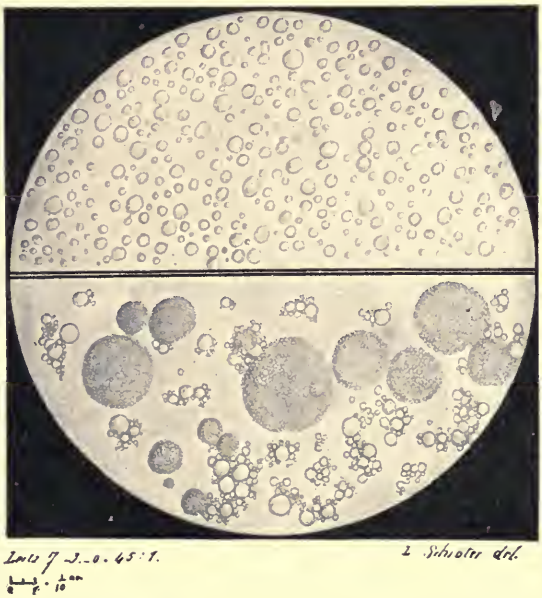

Fig. I.—Fat globules; above, in mother's milk; below, in colostrum.

base, which causes the formation of the skin or pellicle upon the surface of boiled milk. The milk-sugar is changed into caramel by continued boiling and the brown color of commercial preparations which have been excessively sterilized is due to a reaction between the milk-sugar and the caseinogen. Similarly, long continued heating causes a coalescence of the fat globules. The inorganic constituents are greatly changed upon boiling by the breaking up of their organic combinations, but nothing definite is known about the import of these changes in the physiology of nutrition. The ferments and the most of the immune bodies are destroyed at 60°-80° C. (140°-180° F.).

As the child develops, it is finally able to utilize the mixed diet of the adult, the discussion of which is not essential here.

During the first year of life, the child takes nourishment entirely by suckling and swallowing. It is only towards the end of the second year, when the premolars have developed, that the child learns to masticate his food.

In suckling from the mother's breast, even the new-born may develop considerable negative pressure. But this negative pressure of the oral cavity is by no means the only factor in the extraction of the milk. Besides this, the closure of the jaws and the pressure upon the musculature of the areola cause a reflex relaxation of the sphincter muscles. Psychic influence also plays some part in the relaxation of the breast. From the very complexity of these reflex reactions, we may anticipate that there are wide individual differences in the ease with which the breast may be emptied, even though the suckling powers of the children be equal. This proves especially true when the milk is expressed or pumped out.

The liquid nourishment passes the comparatively small mouth of the infant rapidly. According to Tobler's observations on a four-year-old boy, 3-5 c.c. of saliva are added to 100 c.c. of milk. In children during the first four months, in whom the mouth is comparatively dry and the secretion of saliva scant, there is probably even less admixture. By the fourth to the sixth month, the secretion is much more abundant and until the child learns to swallow the saliva it may run from the mouth.

Attention may be called to certain anatomical peculiarities of the infant's mouth which fit it for the mechanics of suckling. The inner margin of the lips is studded with numerous papillæ (the pars villosa) and the middle portion of the upper lip is prolonged in a median labial tubercle. These structures with a series of marked transverse ridges or *rugæ* on the hard palate aid in holding the nipple. The collapse of the lateral parts of the cheeks in the suckling act is prevented by the presence of the sucking pads, specialized masses of fat which lie below the superficial fascia and are pressed against the gums when a negative pressure is produced in the oral cavity. Hasse has pointed out that in sucking, the milk passes the mouth by way of two functional passages—the median salivary cavity, between the tongue and soft palate, and the lateral salivary cavity between the cheeks and gums. In either case the fluid is discharged into the pharynx through narrow posterior apertures lying between the soft palate and larynx medially and the posterior pillars of the fauces laterally. As the larynx lies at a relatively high level it is possible for fluids to pass through these openings into the lower part of the pharynx without entering the cavity of the larynx even when this structure is open.

Ptyalin is certainly found in the saliva of the new-born, although in small quantities, but there is nothing in the milk upon which it can act. When gruels or flour are added to the diet, its function is required.

The stomach of the young infant is but slightly developed as to the fundus; the lesser curvature, because of the fulness of the intestine, is more horizontal and its concavity is directed backward instead of to the right, as in the adult. This peculiarity changes as soon as the child begins to stand and to walk, when the vertical position of the organ develops. The capacity of the stomach is at first very small; varying in individuals and according to diet; it increases rapidly in size as the child grows older. Exact figures can hardly be given because the capacity and the distensibility are not identical under varying conditions of feeding. Such conclusions as may be

drawn from the quantities of food that the healthy child spontaneously takes are noted in the second chapter. It is an established fact, however, that the healthy breast-fed babe occasionally takes much greater quantities of food at one time than would seem possible, according to the capacity of the stomach, and it has been shown that part of the milk passes unchanged from the stomach into the intestine while the child is nursing.[2]

The histologic structure of the stomach wall evidently shows no great differences from that of the adult. The hydrochloric acid and all the digestive ferments are present in the stomach of the new-born.

The casein formation in the stomach occurs rapidly with cow's milk, after it has become acid in reaction. With human milk the process is slower. In the former larger flakes of curd are formed than in the latter. The whey, which is separated after coagulation and which contains the greater part of the salts, the milk-sugar, the so-called soluble milk proteins, and other constituents about which little is known, is soon acted upon sufficiently by the pepsin and hydrochloric acid to permit its passage, in fractional quantities, into the intestine for its further digestion by trypsin and erepsin. The casein in the stomach content, which gradually becomes more solid, is digested more slowly but in an analogous manner, the ferments attacking the outer surface and digesting it. If more cow's milk be put into the stomach before this dense mass of cheese is completely dissolved, the new milk spreads itself between the stomach wall and the outer surface of its older content and prevents its further digestion by taking up the hydrochloric acid and the ferments. It is possible that abnormal changes in the direction of decomposition may develop in this central mass.

The presence of free hydrochloric acid is dependent not only upon the total quantity secreted, but also and directly upon the power of the food to combine with it. For this reason it is present in the stomach of the breast-fed infant after one to one and a half hours, and in the child fed with cow's milk only after two and a half to three hours. This is important, because the free hydrochloric acid has an antiseptic action which the combined acid does not possess.

The total acidity, varying from 20-60 c.c. $\frac{N}{10}$ acidity per 100 c.c. of content is due to the organic acids and acid salts, and especially the acid phosphates present, rather than to the hydrochloric acid. The organic acids arise partly from the action of the ferment lipase upon the fats, which occurs in small measure in the stomach, and partly from bacterial action.

Flour or gruels, given with or without additions of milk, are not only

[2] The stomach of the new-born infant is usually almost vertical in position with the greater curvature to the left. The transverse type of stomach characteristic of infancy is established with the distention of the viscus at birth either with injected fluid or with air and mucus. Until this distention has taken place the anterior surface of the organ lies entirely under the cover of the liver and is usually covered in part by the gastric surface of the spleen. The average anatomic capacity of the stomach is about 1 ounce (33 c.c.) at birth. This is doubled in the first ten days, tripled in the first month and increased over 6-fold by the end of the first six months. After the first four or five days the size of the average feeding is about a fifth to a quarter more than the anatomic capacity of the stomach at the same age.

changed digestively by the long continued action of the ptyalin of the saliva, but are split further by bacteria mixed with them, inducing a fermentation, the products of which are usually, in part, low fatty acids.

The duration of gastric digestion in the healthy child depends upon the kind and quantity of the food. After an abundant feeding of breast-milk, the stomach is empty at the end of two hours, and after the same quantity of cow's milk it is emptied in three hours. Smaller feedings leave the stomach in a correspondingly shorter time. These digestive periods obtain only in healthy children; even the slightest disturbance may influence the motility of the stomach so greatly that the food may remain for an hour or so longer.

The rapidity with which the stomach empties is regulated by the closure of the pylorus which is stimulated reflexly by the content both of the stomach and of the duodenum. According to Tobler's investigations, food rich in fat delays its emptying.

Small quantities of salt and sugar solutions and of albumoses are absorbed by the gastric mucosa. The larger part of the food mass passes into the intestine.

The acidity of the gastric contents is reduced in the duodenum by the addition of sodium carbonate, of which the pancreatic juice contains a large percentage. This causes a withdrawal of alkali from the body.

The digestive changes in the intestinal tract apparently occur in the same manner as in the adult. All the ferments of the intestine and the contiguous glands, even including the prosecretin and the hormone secretin, which are found in the adult, are present in the new-born, and, for the most part, have been identified in the fetus.

A more detailed description of the digestive function is hardly necessary here; but a few special points may be emphasized. As we have said, the absorption of the so-called soluble milk proteins unchanged was formerly accepted as a fact. This, however, has been disproved. They, as well as the casein, are split, in part even in the stomach, to the finer divisions of the protein molecule (amino-acids, and peptids) and, if they are not oxidized, are used in the intermediate metabolism of the synthesized body protein. This is true even of the proteins of the human milk.

Milk-sugar (lactose) if not given in quantities beyond the limits of digestion and absorption, is split by the ferment lactase into dextrose and galactose. If this enzymic action is not complete and if the remaining portion is not fermented by bacteria in the intestine, the milk-sugar may be absorbed unchanged and is then excreted in the urine, as it is if parenterally formed. This incident plays a part in pathologic conditions.

For the clear understanding of many of the metabolic processes which are to be described later, attention must be called to the fact that not only are the products of the digested food absorbed during the whole course of gastro-intestinal digestion, but that there is, also, a secretion of a very considerable quantity of fluids containing proteins and salts into the tract. It should be noted, further, that the colon, in which no actual digestion occurs, is an organ of absorption, as well as of excretion, and especially for the

earthy and fixed alkalis and for iron and phosphorus. The time necessary for the passage of food through the intestine varies, normally, from twelve to thirty-six hours.

The feces, consisting of particles of undigested food and remnants of the secretions of the intestinal tract and of the glands accessory to it, together with a considerable number of bacteria, naturally vary in consistency, color, odor and mass with the kind and quantity of food ingested, with the intensity of the various secretory influences, and with the rapidity of the peristalsis.

The first evacuations of the new-born consist partly of epithelial debris and of the secretions of the fetal intestinal tract and its adjacent glands and, in part, of the constituents of the ingested amnionic fluid and of substances found in it, *e. g.*, lanugo, epidermal cells, etc. Its dark green color and viscid quality, giving to it the name of meconium, disappear so soon as the results of milk digestion appear in the stool. This usually occurs between the second and fifth day.

Under normal conditions the stool of the breast-fed infant has a salve-like consistency, is egg-yellow in color, and has an aromatic and acid odor. The bowel movements should occur once or twice in twenty-four hours. More frequent bowel movements, generally regarded as signs of chronic dyspepsia, occur with surprising readiness in children who are developing normally. They are thin or watery, non-homogeneous and lumpy, or contain shreds, and are full of minute particles of greenish mucoid material in which traces of fecal matter are imbedded. The odor alone is like that of the normal breast-milk stool; it may be of stronger quality, but it never has the offensive character of putrefactive feces. According to the researches of Gregor, we are justified in the belief that the appearance of such stools, which are usually poor in substance, is due to a relative reduction of the amount of fat in the breast-milk, although an increased irritability of the secretory and motor functions of the intestine must be presumed as a causative factor.

Attention must be called to the fact that stools which are yellow when passed may change to a green color when left exposed to the air for a time. While the conditions necessary for the oxidation of bilirubin to biliverdin, in the intestine or after evacuation, are not fully understood, it must be stated emphatically that the condition is not in itself pathologic.

In artificial feeding with dilutions of cow's milk, or with mixtures of cow's milk and gruels or flour, the stool is usually better formed, is lighter in color than the breast-milk stool, and has a slightly unpleasant or even a putrefactive odor. Its reaction to litmus is alkalin. Every deviation from type in the artificially-fed must be taken much more seriously than variations in the stools of the breast-fed infant and must be regarded as a possible symptom of disturbance of nutrition.

As the change to a mixed diet, with only moderate quantities of milk, is made, the normal bowel movements take on the characteristic consistency of the evacuations of the adult.

The entire digestive tract, from mouth to anus, offers a suitable soil for

the growth of numerous varieties and strains of bacterial flora. It may be readily understood that, even during the first few hours after birth, the gastro-intestinal tract becomes infected, both by way of the mouth and the anus, with numerous micro-organisms from its immediate surroundings. All do not enter in the same manner, nor do all find an equally fertile soil. It is manifestly true that the bacteria found in the stool of the breast-fed child are of other varieties and of lesser number than those seen in the stool of the artificially-fed infant.

In the bowel of the breast-fed infant, the number of the anaërobic bacillus, bifidus communis (Tissier) greatly exceeds that of the aërobic bacilli, coli communis and the bacillus lactis aërogenes (Escherich). Besides these, streptococci, the bacillus acidophilus, the bacillus butyricus immobilis (the bacillus perfringens of the French authors), the "Koepfchen bacteria," (Escherich) the anaërobic bacillus, butyricus mobilis, and the bacillus putrificans coli (Bienstock) which causes putrefaction, and several others, are commonly found. In the artificially-fed infant, the colon bacillus and the intestinal cocci are most common, but many of those named above are also present and usually in larger number than in the stool of the breast-fed child.

From the clinical point of view, and for several reasons, the intestinal bacteria are interesting. From experimental researches upon new-born animals, which die when a sterile gastro-intestinal tract is maintained,[3] it seems possible that an irreplaceable physiologic function is performed by them in the human infant. The fact that a continual germicidal activity is exhibited throughout the small intestine, especially in the intervals between the digestive acts, and that rapid bacterial growth occurs only in the colon, where the actual digestive process is completed, makes the problem a difficult one to solve. Nevertheless, the influence of the intestinal flora upon the reactions of the intestinal content has been established. These producers of fermentation and putrefaction exist in constant antagonism to each other and determine an acid or alkalin reaction of the feces, according to the predominance of each type. Of course, the kind of food, apart from the general condition of the organism, has an influence in determining this predominance.

Finally—another point not to be overlooked—is the possibility that under certain circumstances, with an increase of the usually harmless saprophitic bacteria, they may acquire a virulence which makes them pathogenic to the particular infant.

While the kidneys, relatively large in the new-born and during infancy, show normally some degree of fetal lobulation, they generally resemble those of the adult; so, also, does the urinary tract. The urine, during the first few days of life, is scanty and concentrated, tallying with the small quantity of fluid ingested and the large water output from the lungs. It contains a relatively large quantity of uric acid in an amorphous or crystalline form. The explanation of the cause of albuminuria in the new-born,

[3] Schottelius vs. Thierfelder, Nuttel, etc.

the subject of much discussion, an event common only during the first and second weeks of life and but rarely of longer duration, is no more definite than that of the occurrence of uric acid infarcts. Without going deeper into these questions, amply and critically discussed by Czerny and Keller, it may be asserted with confidence that this albuminuria and these infarcts, while probably non-physiologic, are, at most, conditions not particularly harmful to the individual.

The kidneys weigh about 25 grams in the new-born and increase in weight between 19- and 14-fold between birth and maturity. They double their weight in the first year and triple it by three years. They form about 0.6 per cent. of the body-weight in the new-born as compared with 0.2 to 0.3 per cent. in the adult. They occupy a relatively larger proportion of the posterior abdominal wall in infancy than in later life, their lower poles generally lying below the iliac crests and their upper poles extending to the eleventh or even the tenth ribs. Their adult skeletal relations are established as the child habitually assumes the erect posture and the lumbar region elongates.

The bladder is almost entirely an abdominal structure at birth and in early infancy. In the contracted state its apex lies about midway between the umbilicus and pubis on the anterior abdominal wall and its base lies behind the middle third of the symphysis pubis. When filled the fundus may lie above the level of the umbilicus

So soon as the course of nutrition is normally established a close relation between the quantity of water taken and the quantity of urine excreted becomes clear; from 60 to 70 per cent. of the ingested water reappearing in the urine. Of course this is true only under perfectly normal conditions. The urine is voided about three times as frequently as food is taken and, if water be given between meals, micturition may occur twenty or twenty-five times in the twenty-four hours. We are not justified in speaking of physiologic incontinence in the infant; for there is no continuous flow of urine.

The infantile penis varies greatly in size in different individuals. Normally there is more or less complete phimosis and usually but a pinhead opening, with complete adhesion between the glans and the inner surface of the prepuce. Neither this, nor the presence of epithelial concretions in the region of the coronary sulcus, should be, under any circumstances, an excuse for superfluous and disfiguring operations for phimosis. At the present time it is necessary to lay special stress upon this point.

The vulva of the female infant gapes because of the slight development of the labiæ. This physiologic prolapse is favorable to the entrance of fecal particles into the urethra and the resulting occurrence of cystitis. This etiology of cystitis is more fully discussed in the chapter on Genito-urinary Diseases.

The skin of the infant, because of the thinness of the epithelial layer and the greater vascularity of the papillary layer, is more tender, softer and more intensely colored than in later years. These differences are most marked in the new-born, whose skin is so vascular during the first few days that a physiologic erythema neonatorum is described. In all diseased con-

ditions and, especially in disturbances of nutrition, a distinct pallor rapidly takes the place of the blush of health, or, a more serious symptom, a slate-gray coloring may appear.

The subcutaneous fat is present and, in normal conditions, fairly well developed over the whole body and even over the extensor surfaces of the joints where it is absent later in life. When the watery and saline constituents of the organism are normal, the fat gives to the skin of the infant a tensity and elasticity—"turgor"—which, with its fresh color, is a distinct indication of health.

The anatomic development of the sweat glands is slight, while the opposite is true of the sebaceous glands which are well formed. The functional scantiness of the perspiration and the abundance of the secretion of sebum in the new-born tally with these facts.

Finally, the mammary glands, which are modified cutaneous organs, should be mentioned. In both sexes the body of the gland represents a flat disc, not more than one centimeter in thickness. It is usually surmounted by a pale, indistinct areola, in the centre of which is the mammilla about the size of a pinhead. An increase in the volume and an active functioning of the gland during the third or fourth day of life is quite a common rule. This is probably due to the action of the same hormone, circulating in the blood of the babe, which stimulates lactation in the mother. Since the secretion ("witch's milk") is not normally excreted and congestion results, the fluid has the consistency of colostrum. During the first few days of the first month, the secretion completely disappears. Histologic evidences of milk secretion, in the form of dilated ducts and alveoli containing remains of secretion, may be found six months or more after birth.

The body temperature[4] of the infant depends upon the temperature of its surroundings to a greater extent than that of the adult. This is especially noticeable in premature infants and in those who are congenitally weak, so that we may speak of a poikilothermia in these cases. This condition may also appear, however, in normally strong children. Its cause is in part found in the thinner epithelial layers and the greater vascularity of the skin. On the other hand, the low water output from the skin of the new-born and the relative excess of the body surface to its actual volume,[5] are material factors. Slight irregularities in heat distribution, which is largely a function of the body surface, may cause, therefore, slight variations of body temperature the more readily because the relatively small body mass is less able to equalize them by rapid changes in heat production.

It is not necessary to discuss, in this connection, the inadequacy of thermal regulation due to the non-development of thermotaxic and thermo-inhibitory centres, since analogies in other nerve functions, as in the motor field of the new-born, justify the assumption. However this may be, clinical

[4] See technic of examination.

[5] In the new-born we have, for each kilo of body-weight, approximately 810 square centimeters of surface; at six months, 620 square centimeters; at twelve months, 530 square centimeters; at four years, we still have 500 square centimeters; while the adult has only 300 square centimeters.

observation has proved that young infants may be cooled or overheated by external influences much more readily than older children. There is, at least, a reduced range of physiologic heat regulation. Within these limits, it is nevertheless very exact. The healthy infant, surrounded by non-conducting substances, maintains a temperature between 36.8° and 37.2° C. (98.2°–99° F.) almost continuously without the aid of external heat. These figures mark the slight morning and evening range of temperature by rectum. This practical monothermia, according to the careful studies of Jundell and Gofferjé, who took temperatures every two to four hours by rectum, is found only during the first few weeks of life. By the second month, the temperatures similarly taken, revealed variations up to 1° C. (1.8° F.) a range in which, under regular conditions of sleep and growth, may be recognized a nightly fall and a daily plateau. Irregular variations and increases over 37.5° C. (100° F.) in children who have not been artificially warmed by hot-water bottles or similar agencies, must be considered pathologic.

THE PHYSIOLOGY OF METABOLISM

Attention has already been called to the great importance of the rôle which the nutrition and allied phenomena assume in the life of the infant—an importance which will be emphasized in other portions of the work. It seems necessary, therefore, to devote a special chapter, in addition to the subsequent detail of the process of digestion, to the physiology of metabolism.

a. Nitrogen

Nitrogen is present in milk almost entirely in the form of protein. The researches of Bahrdt and Langstein upon new-born animals have demonstrated that even the protein materials of the milk of the mother animal undergo very complete metabolism into amino acid and amino acid complexes (peptids). There is no reason why the results of these researches may not be transferred to the human new-born. If this were done, the hypotheses based upon the differing effects of feeding homologous and heterologous proteins, as represented in natural and artificial feeding would lose support, unless we also assume that their comparatively simple cleavage products (amino-acids, etc.), retain the identical characteristics of the protein from which they are derived. In support of this latter position, however, no definite proof has as yet been advanced. Recently various researches tend to show that foreign protein (egg) may be absorbed unchanged in certain nutritional disturbances. These proteins may be found in the blood and urine; the quantities depend upon the severity of the disturbance.

The nitrogen requirement of the infant is evidently small, as indicated by the low nitrogen content of human milk. This should be remembered in studying the requirements of the nutrition and the period of the greatest relative growth, for it shows how little an excessive protein diet can be justified by the indications of the demands of natural growth. Certain

authors claim that the protein need of the growing infant is fully supplied when 7 per cent. of its caloric need is supplied in protein. The first few days of life only, the period of the so-called physiologic loss of weight, present an exception to this, for if the child be nourished during this period with milk of an established lactation there is a distinct loss of body nitrogen, which is avoided if colostrum, which contains much more nitrogen than does the full milk (Birk) be fed.

In the healthy child the absorption and retention of nitrogen is very complete. The dried feces contain only 4–4½ per cent. of nitrogen, and even this small amount consists only in part of the nitrogen of the food which has escaped absorption. At least an equal share comes from the nitrogen containing secretions of the intestine and its auxiliary glands and from the bacteria present. The retention is represented by the difference between the intake and the excretion in the feces and urine, but it may be said that a positive N. balance is not necessarily indicative of growth or, rather, of tissue building. Nitrogen retention may occur temporarily during periods of weight loss.

The end products of nitrogen metabolism in the urine are the same as in the adult, excepting that the amount of ammonia is normally somewhat greater. This will be more fully discussed in the chapter on Disturbances of Nutrition.

As in the adult, the addition of carbohydrate to the protein food leads to an increased N. retention, in spite of a poorer N. resorption, while fat reduces the N. absorption as well as the N. retention very slightly. This, however, is of no practical importance under normal conditions, since the nitrogen intake always exceeds the minimal requirements. It may become important in pathologic conditions.

b. Fat

The fat taken in milk feeding consists of neutral fats and contains only small amounts of free fatty acids.

As already stated, a slight fat-splitting by lipase occurs in the stomach of the infant. It is probably much less than takes place in the stomach of the adult, where the strong lipolytic secretions of the small intestine normally flow back into the stomach and initiate a more complete breaking up of the fats. The purpose of this is probably to facilitate the emulsification of the coarser fat droplets by the alkalin carbonate of the pancreatic juice, which is dependent upon the presence of free fatty acids. In the infant whose fatty food is taken in the form of a fine emulsion this is unnecessary.

The amount of fat taken with the food varies greatly in the breast-fed child, not only from day to day, but also in the several feedings; doubling in quantity, in some instances, with the increase of the volume and the fat content of the different meals. This is readily understood when we recall the variations of the fat content of the human milk stated above. In the artificially-fed child these variations do not usually occur, since in children fed with milk mixtures, of closely similar quality at each feeding, the total

amount of fat ingested, even when cream is added, is far less than the breast-fed babe receives.

More than nine-tenths of the fat ingested is absorbed from the intestine of the healthy infant. Part of the fat recovered from the feces probably comes from the intestinal secretions and a small portion of the volatile fatty acids may originate from the breaking down of the carbohydrates. The fat of the feces consists of neutral fats, free fatty acids, and the earthy alkalin and fixed alkalin salts of the fatty acids (soaps). It is found in varying quantity. This is shown, without further examination, by the consistency, the reaction and the odor of the evacuations, in so far as the fatty acids are in excess in the acid stools; while in the dry alkalin feces the earthy alkalin soaps are in excess, only a minor part (about 10 per cent.) consisting of neutral fat which shows very slight variation.

The rôle of soap formation in the metabolism of the fixed and alkalin earths is evidently a very complicated one and, up to the present time, is not fully understood. As these are of special importance in pathologic conditions we dispense with their full discussion here.

The absorbed fat is required by the body partly for combustion and in part as a reserve of storage food.

c. Carbohydrate

Only one carbohydrate, milk-sugar (lactose) is contained in human milk and in the milk of animals. It is a disaccharide and splits into one molecule of glucose (dextrose, grape sugar) and one molecule of galactose. These two monosaccharides are combined with a loss of one molecule of water.

Other disaccharides used in the artificial feeding of healthy infants are cane-sugar (saccharose = dextrose + levulose or fructose); and maltose (=dextrose+dextrose) the latter being either an important constituent of the commercial malt extracts, or a product of enzymic action upon starch.

Preformed monosaccharides are not contained in the nutriment of the infant. Only when the child is able to take honey and fruit does it receive levulose and glucose, as such.

Among the polysaccharides, starch and cellulose must be considered. The former is a constituent of the pure flours, partially dextrinized in toast (Zwieback) and found in several of the so-called infant foods; and the latter is a constituent of vegetables and fruits.

It is generally understood that only the monosaccharides are directly absorbed; other varieties of sugar being absorbed only after they have been split by the action of enzymes and, probably, by bacterial action as well. Only if greater quantities are ingested than can be broken up by the enzymes or fermented by bacteria, does the direct absorption of disaccharides occur; and then they are excreted unchanged in the urine, even as they are after parenteral absorption. It may be said, in this event, that the limit of assimilation has been exceeded. The polysaccharides undergo a complicated splitting before absorption.

It has been stated already that the various ferments necessary to carbohydrate digestion are found in the new-born, even though in very small

quantities. This is true both of the diastase of the saliva and the amylolytic secretion of the pancreas. In view of their scanty presence, it is probably true that a relatively large part of the carbohydrate, the exact quantity not being determined, is split by bacterial action (fermentation) with the formation of the acid products of this decomposition.

The limit of assimilation for milk-sugar and maltose is higher in the infant than in the adult.

The carbohydrates of the food serve in the infant, even as in the adult, not only as material for combustion and as a source of energy, but they facilitate or even actualize the normal potential combustion of the fats. If they are absent, a disturbance of fat metabolism results, recognized by the appearance of acetone bodies in the urine. This is more readily developed in the infant than in the adult. It has been determined that a moderate amount of lactose in the diet increases the nitrogen retention, but that large amounts cause a negative N. balance. Also the complete withdrawal of carbohydrates causes a negative N. balance.

The amounts of blood sugar, as recently determined by Goetzky using the method of Bang, are 0.085 per cent. average for infants of twelve days; 0.095 per cent. for one month; and 0.102 per cent. at one year. In older children other authors have obtained an average of 0.072 to 0.113 per cent. which are approximately the same as the variations in the adult.

d. Mineral Constituents

Human milk contains all the minerals necessary for the life and growth of the infant. In the milk of the domestic animals they are present in much larger quantity, corresponding to the more rapid growth of their young. So plentiful are they, in fact, that in the feeding of the healthy infant with the usual milk dilutions their quantity is ample, with the probable exception of iron which is very scanty in both human and animal milk.

The close relationship of inorganic to organic metabolism, and the relative independence of individual kations and anions in their migration through the organism, make it plain that one mineral substance can act vicariously for another in only limited measure. In fact, the continued absence of even one certain ion is incompatible with growth and life. The danger of its lack may be overcome for a time by the ability of the tissues when subjected to "salt hunger" to retain their mineral substances with great tenacity; but this temporary protection is broken down after awhile. These inorganic ingredients have a great influence, also, upon the water content of the organism.

e. Water

Water plays an important part in the life of the infant because the child takes more than double the quantity of water with his food per kilo of body-weight, than does the adult. His body, in fact, contains relatively more fluid. As with other tissue components, the organism regulates this water content, not according to the quantity obtainable, but according to its necessities. By the figures of Camerer it is shown that in the healthy

breast-fed child about two-thirds of the water ingested is excreted in the urine, and only 1 to 2 per cent. remains in the body; the remainder leaving the body by way of the lungs, the skin and the intestines.

The ingestion of larger quantities of water does not increase the storage but the excretion, especially by the kidneys; and since always it takes with it soluble substances (urea, salts, etc.) this probably leads to a partial leaching of the tissues. In order to increase the water content of the body, we must give salts or other substances, *e. g.*, carbohydrates, favorable to water retention, in addition to sufficiently large quantities of water. On the contrary, a reduction of the water retention can be accomplished only by restricting the intake of salts or carbohydrates, or by a pathologic increase of the secretion of water and salts. The simple reduction of the water intake has the ordinary result of reducing its excretion. These conditions probably play some part in the etiology and pathogenesis of the various disturbances of nutrition. It seems, at times, that the possibility of regulating the water balance in the infant is less developed than in later life. Perhaps on the other hand the amount of water excreted from the body by the way of the intestine and the power of water retention play a most important rôle.

f. Accessory Food Substances

It has recently been recognized by experiments on animals and by observation on the human that there must be other food elements which by their presence in the diet promote normal growth in the young and prevent so-called "deficiency diseases" in the adult. To these Funk has applied the name of "vitamins," but Hofmeister's term of "accessory food substances" seems preferable because it is more noncommittal. Their chemical and biological study still offers a large field for research, but it is generally recognized that they occur in small quantities in certain food, and that they are not of animal origin, but are transmitted through milk and meat from vegetable food. Their importance as an etiologic factor of beriberi and the analogous polyneuritis of chickens, of scurvy, and of keratomalacia has been proved. In rickets, however, it is apparently only a factor. The antineuritic principle is contained in most vegetable foods and the form of polyneuritis known as beriberi is produced only by a continued one-sided diet with polished rice. The antiscorbutic principle is found in fresh green vegetables and certain fruits (lemons, oranges, raspberries and tomatoes), and in small amounts in milk and meat. It is quite resistant to heat, drying and preserving. The antirickitic element is found in green leaf vegetables and in animal oils (butter, cod-liver oil, yolk of egg). Friese was able to cure the keratitis produced by the specific Hopkins diet by adding a small amount of fresh milk. The solution of the innumerable problems arising from the study of the accessory food substances is of great importance to the pediatrist.

g. The Total Metabolism

The total metabolism of the child demands a greater intake of food than in the adult because of the added requirements of normal growth. If the

larger output, through the several channels does not counteract this intake, the margin of increase will be very slight, since even in the period of most rapid growth the greater part of the gain in weight consists of water.

Thus Camerer estimates that in a child ten weeks old, weighing five kilos, taking 800 gms. of breast-milk per day, and showing an average daily gain of 25 gms., the increase consists of 18 gms. of water, 0.7 gms. mineral substance, 3.0 gms. of protein and 3.3 gms. of fat.

Actually, a greater intake is counterbalanced by a notably increased physiologic output. In the resting infant, this output is the larger on account of its relatively greater heat radiation. Attention has been called to the fact that the body surface of the infant, as compared to his weight or mass, is two or three times greater than that of the adult. And since the heat radiation runs parallel, in definite degree, to the surface area, the output of the metabolism of the infant would be two to three times as great as in the adult and the intake, in order to maintain a positive balance, would have to be that much greater. As an actual fact Rubner has determined experimentally, in his researches upon adult dogs of various sizes, that their carbon dioxide excretion is proportionate to their body surface, other things being equal; and that it does not rise or fall in proportion to their body-weight. Since the carbon dioxide excretion may serve as a measure of the combustion of organic substances, in accordance with demonstrated physiologic principles, a close relationship is thus established between surface area and food requirement. This food requirement may now be measured by its caloric value, and the experimentally established values of the different food substances may be rated as follows: for 1 gm. of protein 4.1 calories; [6] for 1 gm. of fat 9.3 calories; and for one gm. of sugar 4.1 calories. The salts do not present a calculable caloric value.

Putting the clinical conception of food requirement aside, in favor of the energy requirement or rather the caloric index, which lends itself readily to physical and chemical investigation, the latter has gained recognition in scientific pediatrics by the epoch-making work and studies of the elder Camerer, of Rubner and Heubner; and is both lauded as an important advance and condemned as unscientific and contrary to clinical experience. It may be acknowledged at once, that in considering food as an entity, basing its value upon its heat-producing power, we must accept as a premise that its component substances are capable of far-reaching physiologic interchange. This is based upon the idea of isodynamia, that is, that a calorie of one food component can be freely substituted for a calorie of another food component in the metabolism. At the same time, we must acknowledge that this premise is true only to a limited degree in the healthy infant, and is entirely untrue when applied to the child with disturbances of nutrition. Heubner, to whom the study of the problem of energy requirement in infant feeding owes its greatest advance, has replied to objections raised by Czerny and Keller, when they feared

[6] Protein gives 5.6 calories in the calorimeter, but of this only 4.1 calories are physiologically available as useful calories in the organism, while 1.5 calories are lost in nitrogenous excretions (urea, etc.).

that the physical viewpoint in the teaching of infant feeding would wholly supersede the physiologic-chemical view, by emphasizing that this is not the purpose of the proponents of the method, but that the aim is to give a better quantitative standard of the food requirement of the infant than was given by any former methods in determining the volume or weight of the food. He further asserts that with this common standard it is possible to obtain the most favorable quantitative variations in which the different food substances may and must be put together. The energy quotient furnishes us with a unifying principle and, whatever the choice or qualitative composition of the infant's food, informs us of the essential quantities, which, under all the variances in age and condition the infant demands.

Apart from the minor fact of the broad interchangeability of various food substances in infant feeding, the adoption by the clinic of the caloric method of treating the feeding problem meets with two other sources of error, which, while reflecting upon its absolute exactness, do not impair its approximate usefulness.

It should be noted, first, that a part of the raw calories taken in as food are lost to the metabolism, in that organic substances of definite caloric value are excreted unused in the urine and feces. The sum of the raw calories is, therefore, greater than that of the net calories which serve as the physiologic units of energy for growth and repair. A part of this error in reckoning the raw calories has been already noted (see footnote 6). The remainder because it is small and, in the healthy child at least, of slight variability is a negligible quantity.

In the second place, a further error is incident to the fact that the caloric requirement is estimated by body-weight rather than as the theory strictly demands—by body surface.[7] This really important error is discounted in clinical practice by the fact that a gradual reduction of the caloric requirement is adopted corresponding to the decrease of surface as weight increases.

Attention is called to these limitations in order to protect the caloric method of determining food requirements, on the one hand, from the excessive enthusiasm of its advocates and from its indiscriminate application, and, on the other hand, from the unjust objections of its critics. Within these limitations, the method permits us to determine empirically a definite relation between body-weight and the total food requirement of the healthy child. Heubner has designated the number of calories which a normally growing child requires during the successive divisions of its first year of life, for every kilo of body-weight, as the energy quotient. The first figures given by Heubner were based upon a few observations only; but as experience has added to the sum of knowledge these figures have been, again and again, corrected; and today we use, in round numbers, 100 calories, or a little more, in the first quarter year of life; 90 calories in the second; 80 in

[7] The measurement of the body surface is an extremely difficult task; it takes a long time and is not applicable even in hospitals, to say nothing of private practice. The calculation of the surface area from body-weight, according to the formula of Meeh, demands a mathematical facility which is hardly to be expected of the physician and, therefore, it has not come into common use.

the third and 70 in the fourth, per kilogram of body-weight. For human milk, a caloric value of 700 (650–750) calories per litre has been adopted. The value for undiluted cow's milk is as great. The caloric value of various other milk mixtures will be given later in the chapter on Artificial Feeding.

The caloric requirement of the healthy, artificially-fed infant does not differ greatly from that of the breast-fed child. From various studies it appears that its demand is somewhat larger and these findings have been explained by the fact that the digestive labor is greater upon artificial food and that this necessitates an increased supply of food. Results recently reported by Engel and Samelson do not agree with this conclusion and it is a question whether other factors did not enter into the earlier observations; as, for instance, the greater loss of useful, but unused calories in the feces and urine; or, still more probably the less favorable nutritive balance induced by the greater restlessness of the artificially-fed child.

While the fact that the total metabolism of the child is more rapid than that of the adult has been incontrovertibly established, the rule of Rubner that metabolism and food requirement are, other things being equal, proportionate to surface area has not gone uncontradicted.

Several physiologists, Magnus-Levy, Sonden and Tigerstedt, and A. Loewy, have drawn the conclusion from their researches that a special increased energy is present in the infant because of his youth, which causes a peculiar excitability of the heat-producing agencies and, therefore, an excessive metabolism. Schlossmann and Murschhauser have gone over these results in very complete metabolic experiments in the calorimeter, but have found that they could not confirm them with the material used. They maintain that deviations from the rule laid down by Rubner are due to still another item, that of less economical muscular activity in the infant.

In the study of heat production in approximately normal infants from 19 days to 18 months of age, Benedict and Talbot found an average resting heat production of 65 calories per kilogram of body-weight. They state that "aside from a slight tendency for the total metabolism to be larger with increasing weight, no regular relationship exists with infants between the total heat production and the body weight, regardless of whether the body-weight was actually found, computed from statistics of average values for normal infants, or was the expected body-weight based on the birth weight." They conclude that the extent of metabolism is determined neither by weight nor by surface area, but by the mass of active protoplasmic tissue. The varying amount of fat, comparatively inactive tissue, may influence the variations of caloric requirement.

The requirements of the child for specific materials are similar to those of the adult. Water, salts and protein serve for purposes of growth and repair; fats and carbohydrates are used for fat deposits and, especially, for combustion. It is not immaterial to the organism whether this necessary energy is supplied exclusively or predominantly by fat or by carbohydrate. In the total absence of carbohydrates, as already shown, disturbances of the internal metabolism arise, because an interaction between the digestive products of the carbohydrates and of the fats is necessary to complete the

combustion of the latter. If these products of carbohydrate digestion are wanting, the acetone bodies remain as incombustible end-products. If, however, fat is absent, the integrity of the chemical composition of the body is endangered, as is shown in the retention of abnormally large quantities of water in the tissues. The cause of this water-retention is not known. The fact that the glycogen deposited in the body holds two to three times its weight of water is not a sufficient explanation, since the amount of glycogen is so small. The harmful influence of a disproportionately fatty or carbohydrate diet is important clinically only when it has continued unchangingly for some time.

Corresponding to its more active metabolism, the infant uses more oxygen and excretes more carbon dioxide, although the difference is very small when the exchange is calculated not in ratio to body-weight, but to body surface. The figures, obtained by Schlossmann and his assistants, of 12.85 gms. of oxygen used and 15.75 gms. of carbon dioxide excreted, per hour, per square meter of surface area in the resting infant, agree with the average quantities determined by Rubner in the resting adult.

A very considerable part of the gaseous metabolism is represented in excretion through the lungs with the aqueous vapor. The so-called insensible perspiration, according to a table by Camerer, Jr., is about 1.3 to 1.7 grams per kilo per hour during the first half-year; about twice that in the adult. These averages, however, have been obtained from greatly varying individual determinations. Thus, for instance, an infant when at rest may lose only 2 to 3 grams per kilo per hour, but when extremely restless it may lose from 10 to 15 grams per hour.

GROWTH

In the preceding pages so much stress has been laid upon growth as a visible phenomenon in the life of the infant organism that it seems necessary to treat the principle of growth more fully. Aside from minor indices of growth, of interest only from a clinical standpoint, as, for instance, the increase of the circumference of the head and chest, to be discussed in a later chapter, growth may be determined by measurement in two ways: first, as weight and, second, as total body length.

The figures cited at the close of the preceding section concerning the volume of the insensible perspiration, as well as those which bear upon the capacity of the stomach, suggest that to be of value for comparison the child's weight must be taken always at the same hour. This is also true for the measurements of length, since it is well known that a slight decrease (1-3 cm.) occurs after the body has been in an upright position during the day. The disturbing factor of variance in the amount of the stomach-content is best avoided by weighing the child immediately before the first or second feeding of the day.

The average birth weight of healthy children is 3400 gms. (7½ pounds) for boys, and 3200 gms. (7⅕ pounds) for girls. Great variations from these averages are possible under entirely physiologic conditions. Usually the

first-born children are smaller than those of later birth.[8] The size and weight of the mother do not always determine the size of the new-born child at full term and great variations, in both directions, often occur. This is also true in the degree of development of the subcutaneous fat.

The average weight of the American new-born (white) is about 3.45 kilos for boys and about 3.35 kilos for girls, being slightly above the European average usually quoted. Among the factors affecting the weight of the new-born are sex, activity of the mother in the last weeks of pregnancy, age of the mother, parity and race. Most of these factors apparently influence the new-born weight through their effect on the duration of pregnancy rather than through any direct influence on the rate of growth. There is no scientific evidence to show that changes in the nutrition of

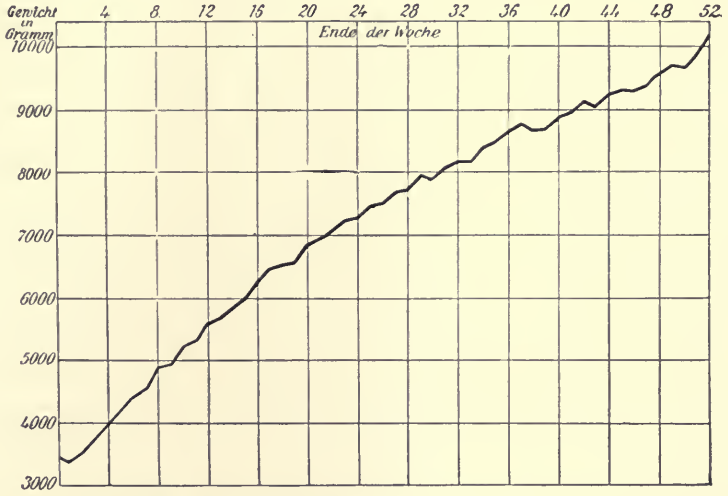

FIG. 2.—Average weight of breast-infants weighing more than 2750 grams (6½ pounds) at birth. (After Camerer, Sr.)

the human mother, within ordinary limits, has any constant effect on the weight of the offspring.

During the first two, three, or more days, all new-born infants show a physiologic loss of weight in varying amount,[9] which is equalized again, in breast-fed infants, by the eighth to the tenth day. Then follows, under normal conditions, a continuous gain, which may be interrupted, as shown in the daily weighings, by pauses or even by slight losses, but which, comparing one week with another, indicate an almost regular rise.

This regularity is, of course, definite only in individuals of undisturbed development. As soon as an average of any large number of children, who are not always weighed at the same interval, is taken, it is lost. The fol-

[8] To this is supposedly due, in part, the fact that the material of lying-in hospitals which consists largely of first-born children, shows a small average of weight.

[9] This physiologic loss amounts to 200 gms. (7 ounces) or more, especially in children of heavy weight at birth.

lowing tables were obtained by the elder Camerer from the study of 119 breast-fed and 84 artificially-fed infants, of more than 2750 gms. (6 pounds) birth weight, without consideration of sex. The table for breast-fed infants is presented in the form of a curve, so that it may be the more readily studied.

TABLE I.

Average weight during the first year of children weighing more than 2780 gms. (6.12 pounds) at birth.

Abbreviated from the table of Camerer, Sr. in the Jahrbuch für Kinderheilkunde, Vol. LIII, pp. 409.

End of Week	Breast-fed Infants		Artificially-fed Infants	
	Grams	Pounds	Grams	Pounds
Birth	3433	7.55	3467	7.64
1st	3408	7.49	3314	7.30
2nd	3567	7.85	3384	7.45
3rd	3781	8.33	3557	7.83
4th	4008	8.83	3683	8.15
8th	4907	10.80	4303	9.48
12th	5600	12.33	4911	10.82
13th	5693	12.54	5093	11.22
16th	6294	13.63	5532	12.18
20th	6824	15.00	6181	13.61
24th	7289	16.05	6836	15.05
26th	7505	16.53	7278	16.03
28th	7774	17.12	7207	15.87
32nd	8175	18.00	7783	17.14
36th	8655	19.06	8161	17.97
39th	8674	19.10	8470	18.65
40th	8855	19.50	8306	18.29
44th	9232	20.33	8782	19.34
48th	9589	21.12	9192	20.25
52nd	10141	22.33	9624	21.19

These figures of Camerer's may be considered rather high in their absolute values and indicative of a stronger tendency to growth than the average in these children. Every physician who has the opportunity to observe different types of children frequently meets with infants who, in spite of continuously undisturbed health and regular development, will not come up to these figures. Nor is the curve the same in all cases. In some, the greater increase occurs at the beginning, while a gradual flattening of the curve is evidenced by the sixth or seventh month; in others, an almost regular advance occurs during the entire first year. Between these extremes all possible transitional forms may exist. Further, attention must be called to cases in whom no actual increase occurs, during the first weeks, because of a slow increase in the quantities of breast-milk, but in whom the delay is fully equalized by the more rapid growth of following months. This is the best of evidence that a long continuance of even scant feeding at the breast has caused no lasting injury to the child.

From these figures it may be seen that the birth weight is about doubled by the beginning of the fifth month and is trebled by the close of

the first year. The differences in weight between boys and girls, which are not shown in the table, gradually become greater, being from 200 grams (7 ounces) to 500 grams (18 ounces) in favor of the boys by the end of the twelfth month.

After the second year, the weight increase is markedly slower. The following table gives in round numbers the yearly averages and also the sex differences of weight.

A number of formulæ have been developed for expressing the growth in height and weight during childhood, but most of these are too complex for immediate practical application. The following simple rules give

TABLE II.
Increase in Weight.

End of the Year	Boys				Girls			
	Body-Weight		Annual Increase		Body-Weight		Annual Increase	
	Kilos	Pounds	Kilos	Pounds	Kilos	Pounds	Kilos	Pounds
Birth.....	3.4	7.48			3.2	7.04		
1st.....	10.2	22.44	6.8	14.96	9.7	21.34	6.5	14.30
2nd.....	12.7	27.94	2.5	5.50	12.2	26.84	2.5	5.50
3rd.....	14.7	32.34	2.0	4.40	14.2	31.24	2.0	4.40
4th.....	16.5	36.30	1.8	3.96	15.7	34.54	1.5	3.30
5th.....	18.0	39.60	1.5	3.30	17.0	37.40	1.3	2.86
6th.....	20.5	45.10	2.5	5.50	19.0	41.80	2.0	4.40
7th.....	23.0	50.60	2.5	5.50	21.0	48.20	2.0	4.40
8th.....	25.0	55.00	2.0	4.40	23.0	50.60	2.0	4.40
9th.....	27.5	60.50	2.5	5.50	25.0	55.00	2.0	4.40
10th.....	30.0	66.00	2.5	5.50	27.0	59.40	2.0	4.40
11th.....	32.5	71.50	2.5	5.50	29.0	63.80	2.0	4.40
12th.....	35.0	77.00	2.5	5.50	32.0	70.40	3.0	6.60
13th.....	37.5	82.50	2.5	5.50	37.0	81.40	5.0	11.00
14th.....	41.0	90.20	3.5	7.70	43.0	94.60	6.0	13.20
15th.....	45.0	99.00	4.0	8.80	48.0	105.60	5.0	11.00
16th.....	50.0	110.00	6.0	13.20	52.0	114.40	4.0	8.80
17th.....	56.0	123.20	6.0	13.20				

weights and heights which fall within the range of normal variation from the average for American children.

Height (in inches) equals twice the age (in years) plus 32 inches (good from 3 to about 14 years).

Weight (in pounds) equals seven times age in years minus 2 pounds for each year under seven (good from 3 to 7 years).

Weight (in pounds) equals seven times age in years plus 4 pounds for each year over seven (good from 7 to 12 years).

A more rapid increase of weight during the years of adolescence is very plainly indicated in this table and a corresponding rise is shown in the table of body lengths. Since puberty occurs earlier in girls, their increases during the thirteenth to the fifteenth years exceed those of the boys, not only relatively but absolutely. During the succeeding years the increase is always less.

The growth in length corresponds to the weight increase, in so far as it is greatest during the first year of life and becomes more gradual with advancing age. The following table gives the yearly averages in round numbers:

TABLE III.

Growth in Height.

End of the Year	Boys				Girls			
	Height		Annual Increase		Height		Annual Increase	
	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches
Birth	50	20.0			49	19.6		
1st	75	30.0	25	10.0	74	29.6	25	10.0
2nd	85	34.0	10	4.0	84	33.6	10	4.0
3rd	93	37.2	8	3.2	92	36.8	8	3.2
4th	99	39.6	6	2.4	98	39.2	6	2.4
5th	104	41.6	5	2.0	103	41.2	5	2.0
6th	109	43.6	5	2.0	107	42.8	4	1.6
7th	115	46.0	6	2.4	113	45.2	6	2.4
8th	120	48.0	5	2.0	118	47.2	5	2.0
9th	125	50.0	5	2.0	123	49.2	5	2.0
10th	130	52.0	5	2.0	128	51.2	5	2.0
11th	135	54.0	5	2.0	133	53.2	5	2.0
12th	140	56.0	5	2.0	139	55.6	6	2.4
13th	145	58.0	5	2.0	146	58.4	7	2.8
14th	151	60.4	6	2.4	153	61.2	7	2.8
15th	157	62.8	6	2.8	158	63.2	5	2.0
16th	164	65.6	7	1.6	160	64.0	2	0.8
17th	168	67.2	4	0.8	161	64.4	1	0.4
18th	170	68.0	2					

Attention should be called to the fact that these measurements taken absolutely are, as already stated of the table of weights, rather high and that measurements which do not come up to them are still within the limits of normal individual variance.

A more rapid increase in girls, both relatively and absolutely, during the years immediately preceding early puberty is recognized. During the periods of sharply accelerated growth the child is commonly said to "shoot up." We distinguish such a period during the first year, which may be looked upon as a continuance of the rapid fetal growth; a second stage appears at about the same time in both sexes, in or near the seventh year; and a third acceleration, dependent upon the climax of puberty, sets in with boys from the fourteenth to the sixteenth year, and in girls from the twelfth to the fourteenth.

Besides these variations in the rate of increase, alike in weight and length, dependent upon the factor of age in children after infancy, Malling-Hanson first observed variations dependent upon seasonal change. These were confirmed later by the elder Camerer and by Schmid-Monnard. Three seasonal periods may be distinguished.

1. The period from the middle of August to the end of November or middle of December, the last third of the year, showing the greatest increase in weight and the least increase in length.

2. The period from November or December to the end of March, or the first part of April, the first third of the year, showing a moderate increase in weight and length.

3. The period from the end of March or the beginning of April to the middle of August, the second third of the year, exhibiting the greatest increase in length, with loss of weight.

It is probable that different modes of life and varying activities of children in the several seasons cause these altered relations in the factors of growth. Whether the sedentary habits and the long continued indoor life incident to attendance at school play an important part is not entirely clear.

The unfavorable influence of improper food and unhygienic surroundings, as indicated by the greater morbidity and the greater frequency of the severer forms of rickets among the poorer classes, is suggested also by the fact, established in many tables of statistics, that, as a rule, the children of the well-to-do exceed those of the poor both in weight and in height. This general observation does not, of course, exclude far-reaching individual differences.

The relations of age, weight and height which obtain in healthy children have been presented very clearly in von Pirquet in the form of the "measuring tape."

To these considerations of general growth should be added some emphasis upon the incidents of special growth.

The Brain.—The average weight of the brain at birth is 370 grams in male and 350 grams in female infants, while the adult brain weighs from 1260 to 1400 grams. One-third of this increase (300-350 grams) takes place in the first 9 or 10 months and the rest of the increase is attained by the middle of the third year. Roughly, the weight of the brain is doubled by the end of the first year and tripled at two and one-half years. The growth is completed by the 16th to 20th years. The sexual difference in weight of 10-15 grams at birth becomes greater, so that in adult life the brain of the male weighs 120 grams more than that of the female. Normal average weights for various ages are approximately as follows:

	Grams
New-born	370
At 2 mos.	460
At 4-6 mos.	600
At 11-12 mos.	850
At 2nd year	970
At 3rd year	1100
At 4th year	1190
At 5-8th year	1220
At 9-14th year	1300
At 15-20th year	1400

The variations in normal subjects are great and even during the first year may be 100-200 grams. There is no noticeable parallel between mental development and size of brain.

Skeletal Growth.—The centres of ossification may be studied in the living by the radiograph and are of clinical interest.

The ossification of the bones of the wrist is of particular interest as indicating the physiologic age of the child. All of the bones of the wrist are commonly cartilaginous at birth although one and sometimes two small ossification centres may be seen in particularly well-developed new-born children. Two centres are usually present at 1 year, 3 at 2 years, 4 at 3 years, 5 at 4 or 5 years, and 6 at 5 or 6 years. The ossification of the wrist proceeds more rapidly in girls than in boys, the former being a full year in advance of the latter at 6 years.

The centres appear normally in regular order, subject to little variation. The time of appearance is not so constant that the exact age of the child can be determined by their appearance. Any pronounced delay in the development of the ossification centres, however, must be considered pathologic and, under certain circumstances, as pathognomonic. It is not infrequently combined with a general delay of the growth in height.

Of the fontanelles, only the greater or anterior is normally open, that is closed only by a membrane, at birth. If the smaller or posterior fontanelle at the juncture of the sagittal with the lambdoid suture, or the parietal fontanelles between the temporal, parietal and occipital bones are still palpable as openings, these, as well as the open condition of the sutures, may be taken as evidence of retarded ossification. From birth to its complete closure, which occurs during the first half of the second year, the greater fontanelle decreases in size continuously. Any increase in area is to be considered pathologic and due either to rickets or to abnormal growth of the head. Complete bony occlusion before the end of the first year is found only in conditions where the growth of the entire head is abnormally retarded (microcephaly). There is great variation in the time of the complete closure of the anterior fontanelle, even in normal children. Collected statistics show that the structure undergoes but little change in size in the first 3 or 4 months after birth. It is closed in about 15 per cent. of all cases at one year and in about 50 per cent. at 15 months.

Dentition.—The physiology of dentition has always been of great interest to the physician. For hundreds of years,[10] this interest was inspired entirely by the fact that the causation of all imaginable diseases of the first years was ascribed to dentition. If this theory which, in its extreme conclusions has led to much useless interference and senseless prescribing and in its actual disregard of disease present has resulted in the death of uncounted numbers of children, could be completely erased from the minds of all physicians of today, it would be as unnecessary to discuss the course of dentition as it is to discuss growth changes in the bones or in other parts of the body. Since this is not yet true, the following statement may be presented.

The cutting of the teeth has been erroneously considered a mechanical process and this one-sided consideration of the growth of the teeth has led

[10] We must thank Ludwig Fleischmann, *Clinic of Pediatrics*, Vol. II, Vienna, 1877, and Kassowitz, *Diseases During the Age of Dentition*, Leipzig and Vienna, 1892, for interesting clinical and historical presentations of this subject.

to the complete oversight of the coincident growth of the jaw. Today, however, we know that most marked and rapid changes take place shortly after birth and that, running parallel with the development of the dental germ, there is an enlargement of the jaw and a gradual resorption of the tissues lying above and beside the growing tooth. This, curiously enough, is true not only during the later stages of development when the tooth has become hard, but, also, in the earlier phases when the tooth is but a soft sack which is capable of exerting only slight pressure.

The erupting tooth does not rupture the alveolus any more than the growing epiphysis ruptures the articular cartilage which covers it. It is rather a process of the spreading of the alveolus for the tooth, the opening out without force of a passage for it under the gum. The soft gum tissue then forms its only barrier, and this too is doubtless overcome, in an analogous manner, by the gradual resorption of the soft parts without noticeable pain and, at least in the nervously normal child, without any disturbance either of a local or a general nature.

This does away with any physiologic basis for the teaching of difficult dentition. In its place, we are gaining an increasing knowledge of the pathology of infancy and a clearer comprehension of the nature of all those disturbances and diseases which were formerly ascribed to it.

A knowledge of the normal course of dentition is of importance for another reason; that is, in regard to the time of the successive appearance of the several groups of teeth. First, between the sixth and the ninth months, the lower central incisors appear; a few weeks later, the upper middle incisors; and, in rapid succession, the upper lateral incisors. The lower lateral incisors appear somewhat later; so that by the end of the first year, at least, all of the eight incisors have erupted. Usually several weeks or several months after, the premolars appear, first above, then below, and but very rarely in the reverse order; and, by the end of the second year of life, the cuspids come through. In the third year of life, the first molars finally appear and with these twenty teeth the temporary dentition is complete. The following method may be used in writing the tooth formula of a child, the horizontal line representing the buccal opening and the vertical the median line.

$$\frac{c'\ c\ b\ a'\ a \mid a\ a'\ b\ c\ c'}{c'\ c\ b\ a'\ a \mid a\ a'\ b\ c\ c'}$$

Any notable extension of the period of dentition marked by longer pauses between the eruption of the several teeth, or any great variation from the natural order in which the teeth appear must be considered as an evidence of rickets.

The second dentition begins with the eruption, in both the upper and the lower jaw, of the third pair of molars (six-year molar); then the milk teeth gradually drop out, in about the order of their appearance, and are replaced by the teeth of the permanent set. Just before the beginning of puberty the fourth molars erupt and finally the fifth pair, called the wisdom teeth, because they usually appear sometime after puberty.

The Nervous System.—The central nervous system of the new-born and the young infant has practically the same form as that of the adult. When we consider that the brain of the new-born is remarkably heavy, even in comparison to the total body-weight (about 1:8 at birth; in the adult about 1:40), we may readily understand that man brings into the world with him a brain which is laid out in external outlines and form upon a remarkably large scale, but the interior may be compared to an unfinished house. The interior is not hollow or empty, nor are its ventricles larger than in later life; but the greater part of its mass consists of unfinished tissue which apparently serves only for scaffolding and framework and which, in the course of later development, is gradually replaced by specific nerve tissue—ganglion cells and nerve fibres. This development involves not only a quantitative increase but also a qualitative change from the simpler forms resembling embryonic types to the higher differentiations which mark the adult. The most noticeable difference in the macroscopic comparison of the infantile and the adult brain and spinal cord is found in the development of the myelin sheaths.

In the cord of the infant born at term this is complete, excepting for a small remnant. Only the directed and crossed pyramidal tracts are almost wholly unmedullated. The cauda equina, the medulla and the cerebellum contain numerous medullated tracts even at birth; while only a few fibres or bundles of fibres, in the cerebellum and proponderantly in the projection system, are medullated. Because of this, the entire white portion of the brain appears gray upon section, being only slightly differentiated from the gray of the cortex. Further development continues progressively, with slight variations of rapidity and order in different individuals, one bundle of fibres after the other becoming medullated.

At about nine months, most of the long association tracts, with the exception of the projection fibres, are medullated, while the shorter fibres, connecting closely neighboring regions, and the radiation fibres are much slower of medullation and are probably not completely covered and definitely developed by the end of childhood or the period of completed growth.

The brain has acquired nearly one-third of its adult weight at the time of birth and the spinal cord about one-seventh, whereas the body increases twenty-fold in weight between birth and maturity. Approximately two-thirds of the postnatal growth of the brain takes place in the first 18 months and over 90 per cent. is accomplished by 6 years. The different parts of the brain grow at somewhat different rates, the cerebellum and brain stem increasing more in postnatal life than the cerebrum. The primary and secondary fissures of the cerebrum are all present at the time of birth although some of the tertiary ones are formed during the first month after birth. It is probable that all of the nerve cells of the cerebrum and cerebellum are formed at birth.

The peripheral nerves of the new-born are very poor in covering and where neurilemma can be distinguished it is thin, unequally developed and frequently interrupted by non-medullated areas. The medullation proceeds

rapidly during the first few weeks; later more slowly, and is completed by the end of the first year. Other histological peculiarities of the peripheral nervous system of infants, which need not be specifically mentioned here, also disappear by the end of this period.

Of the cranial nerves, the optic nerve is only partly medullated at birth and in the region of the cribriform plate is entirely unmedullated. The medullation proceeds from the central to the peripheral end, that is in the reverse of the direction of the transmission of impulses through the fibres. The auditory nerve, on the contrary, is completely covered at birth.

The anatomical differences in the sense organs to which attention should be called, are first, the eye, which is hyperopic in the new-born, and second the middle ear, which is filled with mucous fluid, at least during the first few hours after birth and sometimes for a longer period. This probably comes from the amniotic fluid. The condition of the middle chamber might lead us to suspect a temporary deafness, especially when we consider, as already mentioned, that the auditory nerve is completely formed at birth.

To attempt a description of the psychic development of the child from birth to puberty, even though but the chief points were to be touched, would seem an impossible task. It is better that we refer to the works of Preyer, Compayré, Ament and others, where many references to the literature may be found. We can give only the following points:

At the age of three months, after the so-called stupid quarter of the year, the infant is so completely in control of all his senses that the movements of the eyes, incoördinate at first, have become completely balanced and objects which are not too small and lie in the line of direction of the vision will be fixed and followed. Frequently, images which the child has seen before, as for instance, the faces of his parents, are recognized and greeted with a smile. Similarly, there appears a tendency to turn toward the location of a noise, at first by turning the head and later by turning the eyes also.

Articles which the infant sees or which are laid in the palm of the hand are grasped. However, even up to the fourth month, the closing of the hand is accomplished by a palmar flexion of the hand as a whole. But shortly after, as evidence of purposeful central coördination, the closure of the hand occurs with a synchronous dorsal flexion. During the fourth or fifth month, active grasping motions are made.

The sense of smell, temperature and pain are soon much better developed than they are at birth.

Among coördinated muscular movements, the lifting of the head is the first to appear when the child is laid upon its abdomen; and this position may be maintained for several minutes. At a slightly later period, when the child sits with some support, the head is held erect and is freely turned from side to side; while unsupported, sitting is not usually possible before the sixth month. This is more constant in its date of development than the power of standing, which shows great individual differences. With support under the arms, strong children will stand for several minutes during the fourth or fifth month, and by the seventh or eighth month they will

stand if they can hold fast to something with the hands. However, they sometimes let go suddenly and fall down. Shortly after this children, with well-developed static function begin to take their first steps, while they hold by or lean against the furniture. Children remain at this stage for a longer or shorter time, according to their temperament, before they dare to walk without support. This is usually accomplished between the tenth and the fifteenth month.

Delays may be caused by disease, especially by long-continued illnesses which disturb the entire development. More frequently, rickets may postpone the date of any of these periods of progressively acquired function. Even the simple muscular stretching and the power of standing upon the feet which normal children attempt very early may be absent until late in the second year. These children, recognized for other reasons as backward rickitics, draw the legs up to the abdomen when they are lifted by the arms. Standing and walking, with them, may be delayed until the third or fourth year.

Marked delay in the development of coördinated motor function, even in the matter of holding up the head, is under certain circumstances to be considered an early symptom of imbecility. It doubtless depends, in the first place, upon the lack of attention and interest in the surroundings and is, therefore, to be considered an intra-psychic rather than a psychomotor defect. This is further shown in other respects, as in the reduction of the pain and taste senses phenomena of absence which can be demonstrated at one and the same time and can be explained in no other way.

The first motions of the new-born are in part automatic and in part reflex. This is true, not only of the complicated coördinated motions, such as suckling, but also of the mimic motions of expression.

It is a well-known fact that the new-born infant reacts to stimulation of the taste organs, by placing upon the tongue sweet, sour, salty or acid substances, by corresponding facial expressions. That these responses are brought about by reflex (subcortical) action, without psychical correlation is shown by the fact that hemi- and anencephalic infants in whom the cerebrum, the entire organ for psychic function, is absent, show the same responsive power.

It is very interesting to note how the subcortical reflexes disappear in the course of the first year or are rather replaced by cortical action. This explains the fact that the facial mimicry is often absent in older idiots. Their subcortical reflex is lost, but the cortical action has not developed because of the central defect.

The same condition is seen in the sucking reflex. In the first weeks, it appears unconditionally every time the lips or the neighboring region receive a sufficient stimulus, but later it occurs only when the child is hungry or is waiting for a feeding or the like, and then as a conditional reflex.

The light reflex and the corneal reflex are completely developed in the new-born, while the reaction of the pupil for accommodation appears in the second month. The blinking reflex, excited by the rapid approach of an object to the eye, first appears in the second or third month.

It must be noted that Babinski's phenomenon (the dorsal flexion of the toes, and especially of the great toe, and the spreading of the toes when the sole of the foot is tickled) is physiologic even to the second year.

The skin reflexes, frequently absent in the new-born, are usually very active in older infants.

The lachrymal secretion is absent during the first few months. The tendon reflexes are active in the new-born, as well as in older infants, and are easily brought out when the limbs are relaxed as during the act of nursing. At other times they are masked more or less completely by the physiologic hypertonia of the muscles. It is not easy to explain the condition upon which this "hypertonia," a distinctly increased resistance to passive motion, depends. It is a readily recognized stiffness and awkwardness of all active movements in the extremities of the new-born which exceeds the hypertonia of later infancy. That the muscle of the new-born animal does not respond to a nervous stimulus with lightning-like rapidity, but reacts with a more gradual and more or less tonic contraction—a fact discovered by Soltmann, has probably something to do with it, but the mechanism of the fact is far from clear.

Physiologic spasmophilia, in the sense of an increased excitability of the reflexes (Soltmann) exists neither in the new-born nor in older infants. Nor is the remarkably frequent occurrence of clonic and tonic convulsions at a definite period of infancy, chiefly during the second and third semesters of life, dependent so much upon any physiologic peculiarities of the infantile nervous system as upon special disturbances of metabolism incident to that age. (See chapter on Spasmophilia.)

The acquirement of speech, to which a certain degree of intellectual development is necessary, is timed rather closely. The child shows an understanding of words and simple sentences at about one year of age and soon after, say at about one year and three months, begins to speak. Even earlier, usually between the sixth and eighth months, the child exercises the mechanism of articulation with easy syllables, preparing for the function of speech. Such periods, however, are subject to great variations, in part due to the inherent conditions of the child itself and in part to its environment. They may be markedly delayed without the presence of any mental defect. Such defect should be suspected only when the attainment of speech is delayed until the third or fourth year.

The further development of speech and the exercise of the mental faculties varies so greatly, even in children of similar or nearly similar intrinsic quality, under differing environment, that it is not as yet possible to establish any definite criterion which would be useful in judging the milder grades of mental deficiency. Long continued disease, conditions of exhaustion, and especially defect of sense organs, may delay the acquirement of speech and the exercise of mentality, but a definite prognosis of future development is entirely impossible. In the healthy child differences in temperament are often noticeable at an early period, even in the first or second year, but in these, even, the influence of intentional and unconscious training is very great.

The sleep of the infant is normally sound and long continued. Healthy infants, during the first few months, sleep nearly all day, excepting when being fed or bathed or changed, and when asleep assume the position shown in Fig. 3, which is evidently a continuation of intra-uterine posture. It is usually an indication that the child is ill when the arms sink to the sides. The length of time spent in sleep is gradually decreased, from about twenty hours, by occasional periods of wakefulness, but even during the third to the sixth year the child still sleeps twelve to fifteen hours and at school age from nine to eleven hours.

Fig. 3.—Position of healthy infant during sleep.

Puberty, the period of the development of the genital functions and of the secondary sexual characteristics, gradually leads up to maturity and does not actually belong to childhood.

II. CARE AND FEEDING OF THE NORMAL INFANT

REVISED BY

JULIUS HESS, M.D.,

Professor and Head of the Department of Pediatrics, University of Illinois, College of Medicine, Chicago.

The science of the feeding of children and especially of infants, the differentiation of normal from pathological conditions, and a strict discrimination of what should be considered normal is more important in this than in any other division of pediatrics. The indefinite understanding of normality, the recognition or non-recognition, for instance, of constitutional anomalies even in the infant, the designation of nutritive results as satisfactory when we have only succeeded in causing a great increase in weight and large deposits of fat, have proved to be sources of serious error prejudicial to the science of pediatrics. In their Manual, Czerny and Keller define a new-born child as healthy, "when it is born of healthy parents in the mid-productive period, when it is carried to full term, is free from essential malformities, and is able, with the protection of non-conducting clothing, to maintain a normal body temperature." They further designate that method of feeding as suitable for a healthy child "by which the child develops normally in body and, so far as this depends upon food, psychically, and remains free from disturbances of metabolism, as well as of those diseases the occurrence of which is influenced by disorders of nutrition." Only a strict adherence to these definitions has put an end to the former chaos in the teaching of infant-feeding.

In the future, whoever tries a favorite infant-food upon a number of children, without considering whether, in the strict sense, they are well or ill; and who attempts to draw conclusions of the values or non-value of a feeding method by such means, shows that the entire progress of modern pediatrics has passed him by without leaving any impression.

a. Natural Feeding

The only natural food for the infant, during the first half year, at least, is the mother's milk. To what extent this may be replaced by the milk of other women will be stated later. It is sufficient here to call attention to the fact that so-called artificial feeding should never be considered natural feeding for children of this age.

OBSTACLES TO NURSING

Before we describe breast feeding, the following question must be settled. Can all new-born children be nursed by the mother? This must be answered in the negative. There are doubtless obstacles to nursing upon the part of the mother and upon the part of the child, but they are much more rare than is generally believed, even by physicians.

All diseases of the mother, whether they are connected with the process of giving birth to the child, or whether they be of an infectious or constitutional nature, are only conditional obstacles. Since the feeding of the child, at least during the first period of life, requires only small quantities of mother's milk, the supply makes no special demand upon the physical strength of the mother. Further, lactation is in many respects necessary to her own health. The physician should not always look upon severe acute diseases, as eclampsia and nephritis or pneumonia, as reason to discontinue nursing. Especially is nursing not contraindicated under conditions, involving even large loss of blood in parturition, which give promise of the comparatively rapid recovery of the mother. On the other hand, marked puerperal sepsis, typhoid fever, severe erysipelas, and the like, make nursing impossible because they mean danger to the child. Similarly, malignant diabetes and epilepsy, with numerous crises, are contraindications.

For want of experience unimpeachably beyond the contradiction of critics, views are divided as to the propriety of a tuberculous mother nursing her child. While formerly nursing was absolutely forbidden in every definite case of tuberculosis, a view Czerny and Keller take, this is contradicted by Schlossmann, who bases his argument upon the favorable influence of lactation upon the health and weight increase of the mother, as well as upon the greater resistance of the breast-fed child to tuberculous infection. This claim seems to be supported by several clinical observations. Still later (Deutsch, *Tuberculosis and Nursing*, Münchner, *Medizinische Wochenschrift* 1910, page 1335) facts have been reported on the other side, under rather meagre observations, which indicate that the mother with distinct tuberculous pulmonary disease should be absolutely prohibited from nursing, for her own benefit as well as for that of the child; while in mothers with suspicious changes it may be permitted only as an experiment under the observation of a physician. A positive von Pirquet reaction without physical lung findings, has never seemed to us sufficient reason to discontinue nursing or to interrupt it without mature consideration.

General weakness, anemia and emaciation, extreme youth, and in most cases, even a neuropathic constitution, are not adequate causes for the initial prohibition of nursing.

The majority of such women not only bear the added strain of nursing, but receive undeniable benefit to their own health which is not confined to the better and more complete involution of the puerperal organs—a well-known result, but manifests itself in the rarity with which carcinoma of the breast occurs in women who have nursed. No prediction can be made in the individual case as to whether the woman will lose or gain in weight while nursing. More usually the mother gains in weight, and hand in hand with this gain comes more blooming health, an increase of strength and an improvement in her general well-being. Loss of weight is not in itself a cause for anxiety, for the mother may be placed under treatment and the child may be weaned at any time.

Bearing-down pains in the breast or back, sometimes present at the beginning of lactation, or appearing after getting up are usually dissipated

by suggestive treatment, as, is my experience, is the rare tenderness of the nipples in neuropathic women.

More serious difficulties are presented by fissures in the region of the nipples because of the severe pain which some women suffer and because of the danger of mastitis. Their appearance is not always preventable, even by the massage of the nipples with spiritous solutions which is frequently recommended during the last months of pregnancy.

Skill in placing the child to the breast, so that it sucks from the entire areola and not from the nipple alone and the avoidance of too long periods of feeding are probably the most successful preventatives.

The milder cases of fissured nipple are relieved by various methods of treatment. Glycerin, or glycero-tannin (5-10 per cent.); or the so-called "black salve" (silver nitrate 0.1, Balsam Peru 1.0, petrolatum 10.0), or napthalin ointment; or an antiseptic drying powder (bismuth subgallate, or the like), applied between feedings and removed before putting the child to the breast, give opportunity for the reformation of epithelium. Anesthesin ointment (5 per cent.), may be used to allay pain or, better still, a solution of silver nitrate (3-5 per cent.), may be used. The latter causes an anesthesia of long duration after the short initial pain and repair occurs rapidly under the crust. In addition to these methods, it will be found necessary to bind up the breasts and to empty them frequently by nursing or expression. This may be done either completely, three or four times daily or by removing small quantities frequently. A nipple-shield of glass with rubber nipple may be used in exceptional cases, but the abuse of this device will be treated later. Only rarely do these methods fail to give relief, so that in few cases, and especially in hypersensitive women, nursing has to be discontinued.

Cases of mastitis should be treated surgically with ice-bags, or with warm moist applications, or by carefully applied hyperemia. Later, radial incisions which do not enter the mammilla, may be made as soon as the pus is localized, followed by expression from the incisions.

With this treatment we must provide for the same satisfactory emptying of the breast as in fissures, both for its beneficial effect by reduction of pressure upon the circulation and to prevent the arrest of the secretion. This emptying may be accomplished by putting the child to the breast without hesitation, since, supported by numerous experiences, the admixture of even large amounts of infected pus with human milk is not dangerous to the healthy infant.

By this means it is possible, in most instances, if the inflammation is not of a phlegmonous type and inclusive of the entire breast, to combine the recovery from the inflammatory process with the preservation of function. The secretion of the unaffected breast does not suffer and in case of necessity may be increased to such a degree that its output will be sufficient for many months of lactation.

The form of the breast and nipple makes the first attempt at nursing difficult or easy. Distinctly retracted nipples, rather rare, but which may occasionally occur on both sides, may be an absolute obstacle to

nursing.[1] Very flat, short nipples increase the difficulty of nursing, but do not make it impossible.

The only absolute and continuing obstacle to nursing upon the part of the child is a cleft palate. In children who are born weak or who have suffered considerably in the process of birth and have passed the first few days in a sort of comatose condition, nursing may be very difficult and, in certain cases and for the first few days, at least, may be impossible. In such cases, where suckling is temporarily precluded, the breast must be emptied artificially and normal nursing delayed. The expressed or pumped mother's milk may be given the child by teaspoon or by means of a pipette through the mouth or nose.

In severe coryza, with marked swelling of the mucous membrane, the

Fig. 4.—First position.

Fig. 5.—Second position.

Direct expression of milk.

application of epinephrin solution (1:3000) may relieve the difficulty. Congenital syphilis, frequently the cause of such snuffles, is never a reason for prohibiting the nursing of the child by its own mother since infection from infant to mother is impossible.

The Ability of the Mother to Nurse

Up to this point, we have not raised the question whether there is always milk in the mother's breast. This question of the physical ability of the mother to nurse her infant is of general interest.

Comparing the reports of the large lying-in hospitals, according to which almost 100 per cent. of all women confined there are able to feed their children adequately, at least during the first nine to eleven days of the

[1] In this case the milk secretion is to be maintained by regular expression which can be accomplished throughout the period of lactation.

puerperium, with the experiences of private practice, in which a certain per cent. of all women either do not attempt to nurse or give up the attempt after a short time, because of the alleged lack of milk, we might arrive at the conclusion that the ability to nurse differs widely in the various social strata. Whether this is actually so or not cannot be determined, because the ability or the lack of ability to nurse is not a definite and unchanging fact, but rather a relative condition dependent upon many other factors besides the anatomic and physiologic structure of the gland. This has been often shown by the experience of many institutions when, as a result of a change in medical direction, the ability to nurse has enormously increased, a fact which teaches us to recognize the influence of their surroundings upon young mothers. Anxiety for the health of the mother and doubt of her ability to nurse may, from the first, weaken her desire to overcome the difficulties which present themselves. To strive by personal influence over the mother and, as a teacher of midwives and nurses, for the increase of nursing-power among all classes of the women offers a grateful task to every physician.

Cases in which the breast of the puerperal woman altogether fails of secretion are so extremely rare that they are of no practical importance. The real question to be met is whether we may expect an adequate secretion. Frequently, this can be determined neither before nor shortly after delivery, for the rapidity with which lactation commences differs widely in individuals. As a rule, it is slower in primiparæ than in women who have nursed before. Often it is impossible to express even a drop from the breast for the first two days and yet the milk secretion shortly becomes sufficient if the child is placed to the breast regularly and suckles strongly. We must admit that there are women whose breasts do not functionate normally and whose power of lactation remains inadequate, especially if the child does not nurse energetically and does not empty the breast completely.

The physiologic increase of lactation power, which may drag along for weeks in women whose breasts do not secrete freely, should be remembered in attempting to judge the value or the non-value of the various galactagogues. Up to the present time all the preparations which have been praised, with more or less clamorous advertising, as "milk producers" (somatose, sanatogen, malt-tropon, lactagol, etc.), are no more specific for the mammary gland than are excessive amounts of liquid (soups, milk, etc.), or solid foods. The inefficiency of such agents is shown in the fact that while in some cases no physiologic increase of the lactation power occurs or becomes adequate under forced feeding for a considerable period of time, yet in these very cases a continued and complete emptying of the breast gradually accomplishes its desired result. The recommendations given in good faith by physicians concerning the results obtained by the use of various galactagogues given them for trial, are not testimonials to the scientific knowledge of such men. Galactagogues should be used only for the psychic support they may add to other means advised by the physician.

The fact, which may now be considered firmly established, that the relation between the mammary gland and the reproductive organs, in-

cluding the placenta, is not a nervous one, but rather one of chemically active substances of the hormone type, present in the blood, suggests that the time is approaching when specific galactagogues will be isolated and adapted to therapeutic use. Very suggestive experiments in this direction have been reported by Basch.

Spontaneous failure of lactation is certainly extremely rare and probably always occurs in consequence of an incomplete emptying and an insufficient stimulation of the breast, as in cases where the child is weak and does not nurse properly. This is shown by the fact that this failure, frequently reported by the laity in private practice, is never, or hardly ever seen in institutions conducted by physicians. In spite of the reappearance of the menses, the normal duration of a well established lactation is almost unlimited and may, if a new conception does not occur, continue for several years in women of our race as well as in those of uncivilized peoples.

Eppstein's interesting case, in which a wet-nurse after a continued lactation of over a year, undertook the nursing of the next child of the same family without interruption and with the best results is by no means unique. It shows that the milk of so "old" a nurse may be used for a new-born babe. This fact of the theoretically unlimited duration of lactation is but rarely made use of in practice, because we have to recommend complete weaning by the ninth month or, at least, by the end of the first year, for reasons which will be discussed later.

THE HYGIENE OF THE NURSING MOTHER

The nursing woman should change her mode of living as little as possible, avoiding only harmful excesses both of work and idleness. Special attention should be directed to this with women of the well-to-do classes and with wet-nurses. So far as work is concerned, observations among the poor have shown that even a large amount of daily work in house or factory is borne without injury to the health, or to the secretion of milk of the constitutionally healthy nursing mother.

Psychic excitement, especially anger, pain, sorrow, etc., have no influence upon the qualitative or quantitative condition of woman's milk. The sudden stopping of the flow of milk, supposedly suffered under such conditions in especially sensitive women, is a psychic reflex and probably depends mainly upon the closure of the sphincter of the mammilla, which temporarily prevents the flow or makes the emptying of the breast more difficult. It may be gradually overcome, in every instance, by putting the hungry child to the breast at regular intervals. The idea of the so-called toxic effect of milk supposed to be sensitized by such circumstances should be relegated to the realm of the fable.

The nursing woman should take a sufficient amount of suitable nourishment, but should not limit herself to any particular diet. Nothing should be prohibited that agrees with her. She may eat, without fear for the consistency of her milk, not only spices and sour foods, but also lettuce, raw fruits, etc., with freedom, because in these foods the elements essential to the physiologic growth of the child are contained in larger quantities than

in many others. In women with small appetites as variable a diet as possible is to be recommended, while for those having a tendency to constipation it is well to give, instead of a largely milk and soup diet, foods yielding a large bulk of debris.

The quality of the milk, especially as to its fat content, varies in only slight and practically unimportant degree in any individual and cannot be influenced by the diet of the mother. Particular exceptions (Moll) are not proof against the argument for this view, when we consider that the child itself can regulate the quantity of food which it gets from the breast (Gregor).

Excessive eating and drinking, especially of such foods as milk and rich soups, do not lead to an increase in milk production, but merely cause the nursing woman to put on useless fat. Such excesses are not only useless but should be especially avoided in women who tend to corpulency and to insufficient physical exercise.

After lactation has been fully established, hunger is similarly without immediate effect upon the quality and quantity of the milk. Only after a long sustained and severe degree of starvation, when the bodily strength itself wanes and emaciation ensues, is a decrease in the quantity and probably, also, unfavorable changes in the quality of the secretion to be noted.

The fluid requirement of the mother is naturally increased during nursing. This may be met by drinking large quantities of water, if the food contains sufficient nutriment. The use of large quantities of rich soup has no more effect upon the volume of the secretion than has alcohol in the form of beer or wine. Nothing can be said against the use of beer or wine in temperate quantity. Traces of alcohol are to be found in the milk only when it is taken in very large amount.

The general hygiene of the nursing woman should be the best that her environment will permit. The drawing pains in the back which so commonly occur when the child is put to the breast may be relieved in many cases by supporting a pendulous abdomen or by taking a comfortable position while nursing the infant.

The breast, and especially the nipples, should be kept clean by frequent washing, for esthetic if not for hygienic reasons, even though the rough surface of the mammilla cannot be completely disinfected by ordinary measures.

Of the medicinal agents which it may be necessary to give internally to the nursing mother, only iodine, bromine and salicylic acid are excreted in the milk and these in absolutely harmless quantities. In animals, opium, morphine and atropine also pass through. Mercury, in event that the mother is treated by inunction, is excreted in the milk, but in such minute quantities that it is impossible to expect therapeutic results from it in the child. It may also be said that chloroform anesthesia in the mother is entirely without importance to the nursing babe.

After the confinement the menses do not appear at all, or only once some five to six weeks after, and then remain absent for months or until the end of lactation. Occasionally, they appear regularly during the entire

period of nursing. The occurrence of the menses has in itself no influence upon the quantity or quality of the milk, nor does it cause restlessness or digestive disturbances in the child. When the breast is functionating indifferently and the mother is much affected by the menstruation, an increased difficulty in emptying the breast may be experienced, together with an increased nervous irritability. This should never be considered cause for additional feeding.

To a certain extent pregnancy is more infrequent in nursing women than in others and doubtless the women who are amenorrhœic during the lactation period [2] do not conceive as readily as those who menstruate regularly. Neither the maintenance of lactation nor amenorrhœa give absolute assurance that conception may not occur during their course, but it is very probable that immunity from conception is present for several months of lactation and in nursing women a dangerously rapid sequence of conceptions is not seen as commonly as in those who do not nurse their children. The claim of some mothers that they have had to wean a nursing child because of a new pregnancy is often due to the error of regarding the absence of the menses as an indication of conception. Immediately upon weaning the infant, they conceive at the next ovulation. Other women, on the contrary, discover a pregnancy only after several months have passed, without either mother, nursing child or fetus suffering any harm from the continued lactation. This proves that pregnancy of several months' duration does not necessarily cause the secretion to dry up. The child, therefore, should never be weaned suddenly though pregnancy is suspected and even when it is definitely determined, the weaning should occur gradually.

All these facts tend to show that obstacles to nursing or legitimate causes for its interruption occur much less frequently than has been supposed both by the laity and the profession. The knowledge of this and the avoidance of those numerous rules and limitations with which the nursing mother has been unreasonably surrounded, have produced a gratifying increase in the number of mothers among the educated classes who are willing to nurse their children. It is knowledge, however, that must be brought home to all classes of people by the physician, and its spread will add greatly toward making mothers more ready and more able to nurse. Then, too, those social conditions which force the mother to seek employment will no longer necessarily rob the child of the food provided for it by nature. The value of this to the individual and to the nation is to be considered. (See Section IV.)

THE TECHNIC OF BREAST FEEDING.

The new-born should be put to the breast of the mother only after 24 hours. If the child seems hungry before this time has elapsed, it may be given, from a spoon, a little water sweetened with benzosulphinidum (saccharin). If the infant sleeps for a still longer time, the sleep should not

[2] This physiologic amenorrhœa depends upon a more complete puerperal involution of the reproductive organs, in consequence of which ovulation is arrested.

be interrupted. Quite a few children require no nourishment for 36 to 48 hours after birth.

Infants act differently in their first attempts at nursing. Some immediately suckle well and with much force; others will not take the nipple or let go after a few attempts. Nothing is to be gained by force. The child is to be put back into its crib and the attempt repeated after an interval. With patience and continued application to the breast every normal child will learn to nurse. If the breast is very tense and difficult to empty it may

Fig. 6.—Method of holding baby during nursing.

be made more responsive to the effort of the child by expressing or pumping off small quantities of milk.

Emphasis should be put upon the fact, from the very first, that the child must take not the nipple only, but also almost the entire areola into its mouth; for the greater the portion of the breast included, the greater is the area affected by the stimulus of suckling and the greater is the reflex response. This practice is also less liable to tear the nipple and to produce fissures.

The nipple shields or protectors, consisting of a glass to cover the mammilla and a rubber nipple for the child to suckle, which are in common

use and are intended to make the first application of the infant to the breast easier, or are used out of an excessive fear of mastitis, are dangerous. Their harmfulness lies in the fact that while they may make the work of suckling easier for the child, the unphysiologic stimulus makes the emptying of the breast much more difficult. The continued use of such an apparatus almost always leads to the drying up of the secretion and the under-nourishment of the child.

Conical breasts are more easily emptied by milking movements which make slight rhythmic pressure upon the areola; flat breasts are best emptied by pumping apparatus. Of pumping devices there are numerous types. The *teterelle biaspiratrice,* which was formerly in common use, consists of a glass bell placed over the nipple and supplied with two pieces of tubing, from the one of which the mother draws the air, while from the other the child takes the milk. In this form, the saliva of the mother easily flows into the apparatus, which does not seem to be very desirable. Of the various models in which a negative pressure is produced by a rubber bulb, the pattern designed by Ibrahim (Fig. 7) is easily handled and readily cleaned. With rhythmic compression and release of the heavy walled bulb, the action of the normal suckling apparatus may be imitated to some extent; but here also the rhythmic massage of the areola is lacking and the breast is never emptied as completely as by the suckling of a strong child.

Fig. 7.—Breast pump (Ibrahim)

It is not only necessary to empty the breast completely in order to obtain sufficient milk for the child, but also because this is the only method which stimulates and increases the initial secretion and prevents congestion. If the breast is not completely emptied, the irreparable result, an unavoidable one during the first few weeks and often within a few days, is the complete failure of the secretion.

The colostrum secreted during the first few days is very small in quantity and often measures only a few cubic centimeters. Usually, after the third or fourth day, but often not until the fifth or sixth, a rapid increase of the secretion ensues, which in many women is accompanied by the subjective feeling of the "shooting-in" of the milk. At the same time, the number of colostrum corpuscles rapidly sinks to a minimum.

The further increase of the quantity of the milk is dependent on the one hand, upon the amount of glandular tissue in the breast and, on the other hand, upon the demand made upon it. These two factors determine the total production of the secretion, as well as the time at which the function is fully established.

The question whether the child should be put to one or both breasts at each feeding can be answered only in a general way by saying that the

object of the nursing should be to empty the breasts as completely as possible and that congestion must be avoided. Other things being equal, this occurs to a greater extent in small breasts than in large ones. It is permissible at times, therefore, to apply the child to both breasts after an interval of at least three hours, taking care only that the child receives the second breast after it has emptied the first one sufficiently. By beginning alternately with the breast from which the child has nursed last, all these demands are fulfilled in the best manner. It is only with very well developed breasts that we may persist in giving only one breast from the beginning.

The ease with which the breast may be emptied, whether artificially or by the nursing infant, varies greatly in individuals in whom the quantity of secretion may be the same. It often happens that while the child is nursing from one breast, the milk drops or even spurts from the other.[3] The number of feedings which the healthy infant will take spontaneously from a freely secreting breast varies between five and six in 24 hours; but it may occasionally fall to four or be increased to seven. It is recommended and is generally accepted, to-day, that three-hour intervals should be allowed between the feedings during the day and that during the night two longer intervals are most satisfactory to the favorable development of the child.

A large experience with new-born and older infants has shown, and the method has been generally adopted by numerous pediatrists, that an interval of four hours is even more satisfactory. This is true both from the standpoint of the child and of the mother. The editors have used the four-hour feeding for a number of years. The schedule is placed in force 24 to 48 hours after birth. On the part of the child, less digestive disturbances are encountered and on the part of the mother, the great advantage lies in the fact that she is given more freedom for work or social duties. Even prematures are more successfully fed on this schedule. The hours for nursing most frequently recommended are 6 A. M.; 10 A. M.; 2 P. M.; 6 P. M.; 10 P. M. and one night feeding at 2 A. M., if necessary.

It is usually superfluous to prescribe the length of the individual period of nursing for the healthy infant, because when satisfied it stops suckling, often falling asleep at the breast. Some children suckle for a moment and then stop, and when the attempt is made to remove them from the breast grasp it greedily, to suckle again for a moment. In such children the time of nursing should be limited to fifteen or twenty minutes at the most.

It may be shown, by weighing the child at equal intervals of about five minutes, that the quantities taken during successive periods of nursing decrease very rapidly and that only a few grams are taken after the first

[3] After long intervals between nursings, and especially at night, is this apt to occur. In many women, some milk flows from the breast after long intervals in nursing and more especially after the night interval. This becomes annoying and disturbing only when it occurs immediately after putting the child to the breast and when because of an abnormally low tone of the sphincter mammillæ, a sort of incontinence or galactorrhea results. Obstinate eczema of the areola and of the skin over the breast may ensue. The treatment of this anomaly, which usually appears to be of neuropathic origin, is generally quite useless and must confine itself to the application of dressings to absorb the milk and to the treatment of the eczema with ichthyol or with silver nitrate solution (2-3 per cent).

twenty minutes. The long-drawn-out nursing period not only wastes the mother's time and strength but is actually dangerous because of the maceration of the nipple and the consequent liability to fissures.

The quantity of milk taken at each nursing, determined by weighing the child before and after putting it to the breast, varies within wide limits. Usually the secretion is most abundant at the first morning feeding and is often two to three times as great as that of the smallest output which is usually had late in the afternoon. This is most definitely observed after an interval of the whole night especially in breasts of large capacity.

The total quantity of the twenty-hour hours' secretion is of greater import to the physician. It will be seen from the above statement that this total can never be obtained by multiplying the amount secured at one nursing by the number of feedings, but must be determined by weighing the child before and after each nursing. The quantity of milk which the infant receives within the 24 hours varies also from day to day; the variance running at times from 200-300 c.c., so that the knowledge of the quantity of any one day's feeding permits only indefinite conclusions upon the total amounts regularly taken. On this account, Czerny and Keller have adopted as a standard the average of the total output of five days. Excepting from this estimate the first 8 or 10 days, when great irregularities occur, it has been determined that the amount of nourishment taken during the first few weeks equals about one-fifth of the body-weight. This quantity is gradually reduced to about one-sixth or one-seventh, between the first to the fourth month, and at the end of the first six months it equals about one-eighth of the body-weight. This gradual reduction of the relative quantities of food stands in close relation to the conditions described on page 22. When the quantities of food taken and the body-weight are represented by curves a marked divergence appears, in that the weight curve continually rises while the food curve, representing food quantities, becomes more and more flattened. It must be said, however, that cases have been observed in which the two curves ran parallel for months.

Under normal circumstances both breasts commonly secrete like quantities, but it is not exceptional to find women in whom one breast secretes more freely than the other throughout lactation. Similarly, women are quite often found in whom one breast has been depreciated by a former mastitis, but are able, for many months, to produce sufficient milk with the remaining gland which has become structurally and functionally hypertrophied. This is especially interesting when there are twins, in which case each child usually suckles one breast.

When the quantities of food fall markedly below the average given and still produce satisfactory increases in weight, we are justified in the supposition that this depends upon a higher fat-content of the particular secretion. This has been proved in a case reported by Heubner. The occurrence of such individual differences, uninfluenced by the mode of life of the nursing mother, has been established by careful observations made under due precautions. It would seem, generally speaking, that the fat-content of milk from breasts giving small total quantities is relatively much greater than in

breasts which secrete greatly in excess of the quantities required by any one child. Wet-nurses, for instance, are found in institutions, who give two to three litres per day of such milk. When several children are put to the same breast in succession, the first child to nurse receives milk containing the lowest percentage of fat.

The weight increase is looked upon as the chief index to the results of feeding. This view is probably justified by the large consideration to be given to the congenital tendency to growth and the many-sided import of

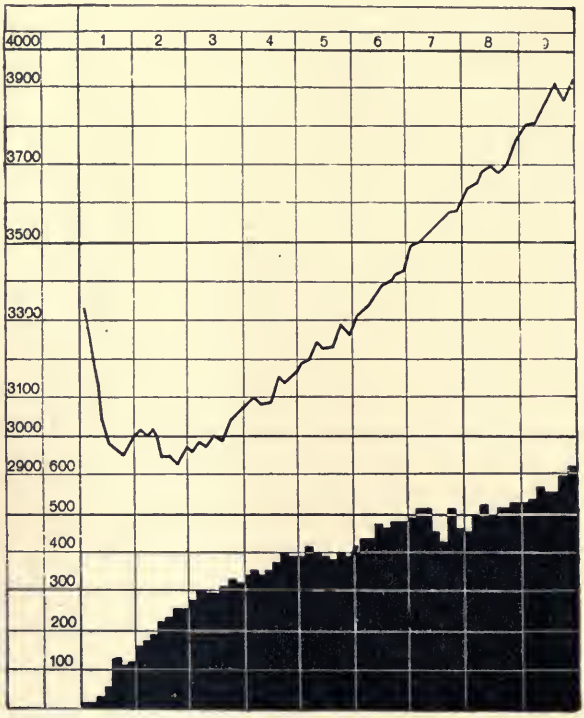

Fig. 8.

weight increase, but it should not be permitted to overshadow such other signs of general well-being in the child as its color, turgor, disposition and sleep, the development of its static functions and its reaction to infections. The normal condition of the child in all these respects is more important than the rapid increase of weight.

In healthy children who develop well, the weekly increase varies in the second and third quarters of the first year between 150 and 250 grams and occasionally even 300 grams; and in such a regulated manner that especially large increases of one week will be equalized by smaller increases in the next, and *vice versa*.

If the increase in weight remains for a considerable period below what would naturally be expected, the cause should not be laid unconditionally

to underfeeding. We should rather seek to determine, by the methods already cited, whether the quantity of food is actually too small and if so whether the fault lies in a positive lack of milk, in a weakness of suckling, or in anorexia in the child due, for instance, to parenteral infection. The last of these alternatives is much more common than is generally believed. To avoid serious errors, every other cause of disturbance upon the part of the child should be certainly excluded before means are taken to increase the food artificially.

As we have already said, the time of the "coming in" of the milk and the rapidity of its increase in quantity—stimulated by the suckling of the child and by the measure of its food requirement—varies greatly in different individuals and is usually slower in primiparæ than after repeated lactations. This possibility and the axiomatic fact that even scant nourishment at the breast for a certain period does not injure the child, justifies the physician in keeping the patient under careful observation and waiting, as long as possible, for the full establishment of lactation in these cases of delayed beginning, or slowly increasing output. As a guide, it may be stated that after the physiologic loss of weight has occurred (see page 24), a lesser loss of 5 to 10 grams a day may occur and that such loss may be permitted to continue for one or two weeks, giving the child only a little water sweetened with benzosulphinidum (saccharin). If no increase in milk secretion or in the body-weight of the child occurs by this time, it becomes necessary to supplement the insufficient breast-feeding and usually with some artificial mixture.

It may be possible, in rare cases, to give the child who does not receive sufficient milk from its mother, several adequate feedings a day at the breast of a wet-nurse. It may be stated emphatically, that in case of necessity, the child may receive each feeding from a different wet-nurse, as has been done in certain institutions, and it may still develop exceptionally well.

The addition of artificial food to the breast feeding, a method to which the French term *allaitement mixte*, or mixed feeding, has been applied, serves much more favorably for the development of the child than artificial feeding alone, and it should be recommended more frequently by physicians than it is. Since the once deep-rooted prejudice against this mixed feeding, or the alternation of natural and artificial food, has proven entirely without foundation, the lack of sufficient secretion by the mother's breast should never be an occasion for weaning, but always an indication for the addition of other food. In such cases, it may be well to give the bottle once or twice daily, or later, at the most, three times during the 24 hours, in place of the breast; or instead of this to the scanty breast feeding, the artificial food may be immediately added once, or several times, a day in sufficient quantity to satisfy the infant.

Each of these two methods has been successfully used and has been recommended; either is justified in suitable cases. It is of prime importance, whichever method is followed, to prevent the child from being weaned. Further, preference may be lent to the easier way of giving the added nour-

ishment. The surest avoidance of weaning is found in the first form of mixed feeding, that is, by the alternate feeding at the breast and from the bottle. This type of mixed feeding is, too, the only possible one when the mother is hindered by outside demands from nursing the infant regularly. Either method, however, should be carried out under strict observation,

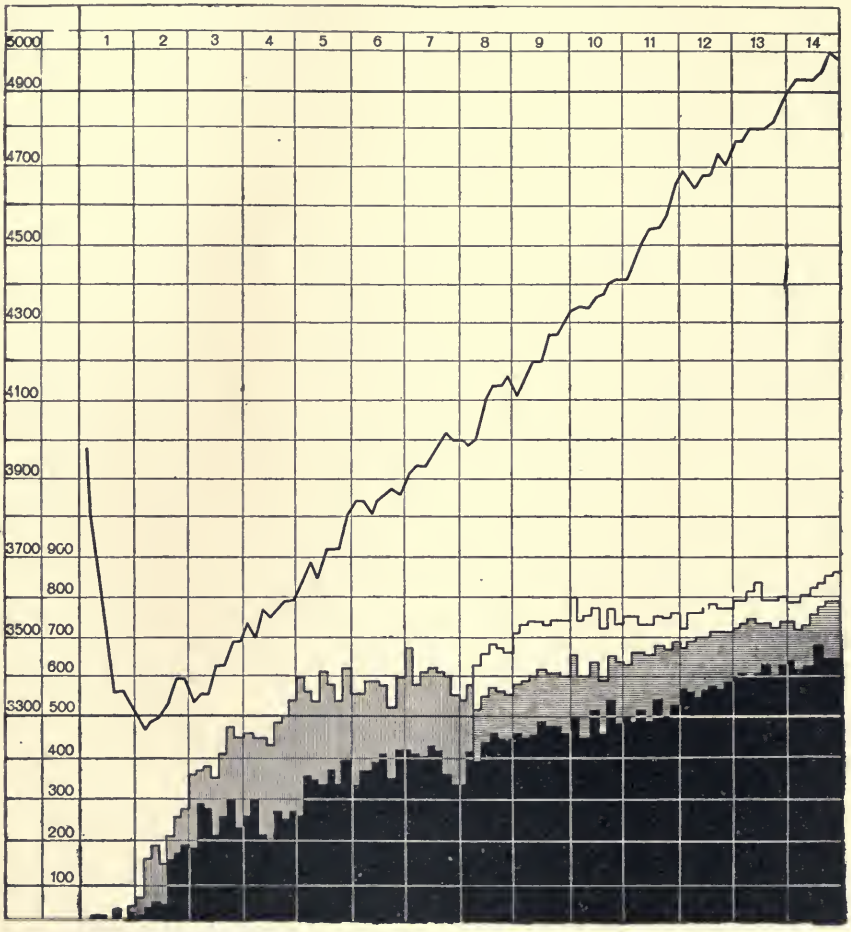

FIG. 9.

giving the child only such quantity of artificial food as is absolutely required, so that it will continue to empty the breast completely and so prevent congestion and the arrest of the secretion. Otherwise a result, frequently seen in practice, soon obtains in the infant's refusal to nurse the scantily secreting breast, thus "weaning itself," because "it does not care for the breast any more." The choice of the food to be used in mixed feeding depends upon the accepted standards of artificial supply.

If the occasion for mixed feeding disappears, it may be possible to dis-

continue it in favor of exclusive breast feeding. Thus, with an initially scanty secretion, the continued strong suckling of the infant may stimulate the breast to the point of sufficiency; or, in event of hitherto enforced absence, it may become no longer necessary for the mother to be away from the child for hours at a time. Since the secretory capacity of the mammary gland is dependent within wide limits upon the demands made upon it, the quantity of the secretion usually becomes sufficient in amount within a few days after the abandonment of mixed feeding. It is even possible to bring a breast which has almost entirely dried up back to full function again. This so-called re-lactation, however, occurs only when the colostrum formation has not advanced too far.

WEANING

In spite of the fact that the period of lactation itself, as already said, is almost unlimited and that the secretion itself may be adequate to produce satisfactory and normal gains in weight even beyond the first year, it is neither customary nor advisable to continue breast feeding exclusively for so long a time. In the first place, the normally developed breast-fed child will indicate its desire for other food much earlier; often, at about the sixth or eighth month taking bread and the like in its mouth and eating it. In the second place, the additional food given at this time acts in a clearly favorable manner, maintaining the natural fresh color and sound turgor, promoting the development of the bones and the exercise of static functions.

Whether this beneficial action of the additional food is due to the added carbohydrate supply or to the increase of inorganic salts, particularly calcium and iron, both of which are present in relatively small amounts in human milk, is beside the question. It is certain that the natural transition to the mixed dietary of adult life is satisfactorily made in this manner.

The time as well as the methods of the transition show great variation in accord with the customs of the country and it is not always made in a proper manner The error of too early feeding, so early indeed as the third or fourth month with large quantities of sweet or starchy foods and breads or with animal milk, is much more common than the error of too long exclusive feeding with breast-milk

We recommend the method of Czerny, now very generally accepted by pediatrists, of beginning at the sixth or seventh month to substitute for the noon-meal of breast-milk some 5 to 7 ounces of farinaceous soup prepared with meat broth. The soup-stock should be prepared from a quarter of a pound of meat and should contain the same condiments (salt and vegetables) as may be used for adults, but the fat should be completely skimmed off and it should not contain so large quantities of lime salts as may be found in broths made from calves' feet or young fowls, because these will produce diarrhœa in some children.

After several weeks, or even after several months, if perfectly safe milk cannot be obtained, as may be true during the summer months, a second breast feeding is replaced by an artificial feeding consisting of cow's milk and flour soup, or milk and toast. These feedings should be in accord in

quantity with those recommended for a child of given age and weight. Gradually, with intervals of at least several days, the remaining breast feedings are replaced in a similar manner. During the process, however, the breast and the artificial feedings should alternate, in order to prevent marked congestion of the milk in the breast. When the weaning is complete, the child's menu may be varied by the addition of any one of the large variety of soups (rice, sago, potato, legumens, etc.), or of one or two tablespoonfuls of mashed vegetables (carrots, spinach, cauliflower, etc.), or by replacing the milk and flour soup, or the milk and toast by a more nourishing milk pap, made with cereals, rice, etc.

Stewed or raw, scraped vegetables should, for pedagogic reasons, be given only after the child has acquired the taste for several of the mashed vegetables, otherwise it may be difficult to teach the child to eat the latter. Even with the greatest variety of food, care should be taken to prevent the child from being "spoiled" and capriciously selecting its own food. When vegetables are given, it is well to warn the mother to expect the appearance of undigested shreds in the stool, for if this is not done she may become frightened and discontinue their use in the belief that they are indigestible.

Even large and strong children should not be given more than one quart of milk daily at the end of the first year. The milk other than contained in gruels, etc., should not be fed from the bottle. It is better, even if somewhat more troublesome, to teach the child to take the food from a cup or mug after it has learned to take soup from a spoon, because this is the natural mode of drinking.

The unpleasant custom, a widespread one in some countries, of allowing children, who are running about or even of school age, to take their milk from a bottle, because they will drink more by this means, is not only irrational and unesthetic but is a practice harmful, as all long-continued sucking is, to the process of dentition.

WET-NURSING

The only perfect substitute [4] for mother's milk is the milk of a wet-nurse. The only prerequisites are that the wet-nurse be healthy and that she have sufficient milk. The qualitative differences that may obtain in the milk of different women play no part in the nourishment of a healthy infant. As an actual fact, everything that has been reported concerning the unfitness of the milk of certain mothers is due to the improper interpretation of superficial observations.

Even though difficulties in the establishment and maintenance of wet-nursing are very frequently encountered in private practice, these difficulties almost always result from avoidable errors due to a lack of

[4] This is not true in an ethical sense. The mother who does not nurse her child, but leaves its feeding and care to the wet-nurse, has later great difficulty in regaining the tender love of her babe which naturally turns from her to its nurse. She risks, further, the danger of having the child acquire the personal characteristics and habits of the nurse, which are often far from desirable. That individual peculiarities may be transferred with the milk is not true, although it was formerly believed. Their acquirement is entirely due to the unconscious mimicry of the child.

knowledge of the basic principles of normal lactation or of the physiology and pathology of the breast-fed infant. The conditions for wet-nursing are the same as obtain for the feeding of the child at the mother's breast. The frequent change of wet-nurses is, therefore, in most cases, a poor testimonial to the knowledge of the physician in charge.

A very common error is made in demanding for a weak infant who can take but small quantities of food, a wet-nurse who has a large supply of milk, a condition always distinctly specified alike by physicians and parents. The natural results, in congestion of the breast and in decrease and final disappearance of the secretion, may be avoided, often, only by pumping the excess of milk or by permitting the wet-nurse's own child,[5] which she also nurses, to empty the breast completely. It is best, however, when selecting a wet-nurse to see that there is a reasonable relation between the quantity of milk secreted and the food requirement of the child.

The provision of wet-nurses was, up to a few years ago, a very serious matter and is still difficult from the medical as well as the sociologic and ethical viewpoint.

Wet-nurses recommended and introduced by employment agencies, by which they are tempted with promises of well-paid positions, are usually in the stage of well-developed congestion of the breasts and are often in danger of losing their milk when they accept a position. It is not surprising, under this method of hiring a wet-nurse, that her family history and the detail of her past life, previous illnesses, etc., most certainly necessary to establish her fitness as a wet-nurse, are usually intentionally falsified; while the child which is shown to the physician is often not her own, a fact which develops most unexpectedly and unpleasantly later on.

Her physical examination should, of course, be very complete and should be directed especially to the discovery of tuberculosis, syphilis, gonorrhea, and parasitic and infectious skin diseases. In view of the frequency with which the von Pirquet reaction is positive, indicating latent tuberculous foci in the adult, it is not a sufficient reason for rejecting an otherwise satisfactory wet-nurse. The Wassermann reaction should be made as a matter of routine. The inspection of the wet-nurse's child, which is not always possible, is much less certain than the blood examination in the exclusion of syphilis. With practice, it is possible to determine approximately by palpation of the breast, which is not intentionally congested, whether there is a sufficient output. There should be sufficient tense glandular tissue, which is harder than the surrounding fat. The skin over the breast is warmer than over the sternum and has numerous large veins passing through it. Exact knowledge of the quantity of milk may be obtained only by weighing the child before and after feeding.

The numerous dangers which arise in hiring a wet-nurse from an employment agency, briefly indicated above, may be avoided if the wet-nurse is taken from an institution, an infant and lying-in hospital, etc.,

[5] This method, which is advisable because it is humane, is practicable in sufficiently well-to-do families and should be considered whenever possible.

in which she and her child have been observed [6] by unprejudiced attendants, for weeks or months, as to their health and general characteristics and the adequacy of the breast yield. They have usually learned, in such an institution, to handle and to properly care for an infant.

The old rule that the wet-nurse's infant should be about the same age as the child that she is to nurse is now believed to be without foundation. It is rather desirable for the reasons cited to select a nurse for even a new-born infant, whose lactation has been established for several months. If we can be certain that the lactation is well established, a mother who is nursing for the first time will prove entirely satisfactory.

Even though she takes care of the child and helps with the housework and is not fed with too great luxury, the wet-nurse tends to be a great expense because of her high wages and the necessity that a room be provided for her.

Mixed feeding (*allaitement mixte*) secured by the employment of a wet-nurse who comes to the home to nurse the infant once or twice a day, is much cheaper and is in most cases a satisfactory substitute for the exclusively used wet-nurse. Every healthy woman who has sufficient milk, if she wishes to nurse the child several times a day, may accomplish this either by going to the child or by having the child brought to her. If the child is given the breast three times a day, it will be necessary to give two artificial feedings in the intervals. When the nurse comes to the house for nursing periods only and keeps her own child, also partially fed at the breast, most of the unpleasant features of the situation arising when the wet-nurse must be taken into the house and treated as a companion disappear. The test weighings of the child, before and after nursing, to ascertain the quantity of milk obtained from the nurse, are to be recommended as a matter of reassurance to the parents and as a control measure when the child does not develop as well as might be expected. In this method of mixed milk feeding, a complete knowledge of the physiology and pathology of infancy is as necessary as it is in all other forms of feeding. It is especially important for the physician to meet the many superstitions of the mother, who will see a dietetic error of the nurse in every cry and in every irregularity of the stools of the infant.

b. Artificial Feeding

Artificial feeding, surrounded by a grim troop of dangers, stands in distinct contrast to the certainty with which normal growth and development occur at the mother's breast. This is proved most clearly by the high mortality rate of the artificially-fed. These dangers depend partly upon the decomposition of the component parts of the animal's milk used in making the artificial food, and partly upon chemical differences between human and animal milk which cannot be entirely met. These dangers are greater the earlier the artificial feeding is begun.

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[6] In such institutions the wet-nurse's child will find a home and be cared for, which, in part, reduces the expense. A law regulating wet-nurses, with special regard for the wet-nurse's child, has been rightfully demanded from various sources.

MILK FOR INFANTS

The first of these difficulties of artificial feeding may be measurably reduced if the most scrupulous care is exercised in the dairying and handling of the milk. Milk so secured and treated is usually marketed under some distinctive name as "milk for infants," certified, sanitary, or inspected milk. Under insufficient legal regulation, no name in itself offers any assurance of the purity of the milk, unless it stands for actual inspection and certification. On the other hand, milk not sold under label, is not necessarily contaminated and unfit for infant feeding.

Aside from intentional contamination, punishable by law, such as partial skimming or the addition of water or preservatives, to hide already advanced souring, and aside from the accidental admixture of pathogenic organisms, the chief source of the impurities of milk lies in the implantation of greater or less numbers of saprophytes which, with varying rapidity, induce its complete decomposition.

The larger number of these organisms come from the impurities which fall into the milk during the process of milking, and consist of hairs, epidermal scales, manure, particles of food and stable dirt. These impurities are carried into the milk by air currents, or from the hands of the milker, or from dirty vessels and implements, or by dirty water with which the latter are rinsed.

Therefore, the extreme cleanliness of the cow and especially of the cow's udder is important. Care should be taken not to stir up dust in the stable from bedding or foddering shortly before milking and the greatest possible cleanliness of the entire equipment, not only of the hands and clothes of the milkers but also of the vessels used in milking and in gathering and measuring the milk, of the straining cloths, etc., should be secured. In other words, asepsis, as complete as possible, should be practiced in obtaining the milk to be used for infant feeding. Further bacterial contamination occurring in the handling and transporting of milk, in pouring it into different containers and in measuring it for sale, is relatively small. The primary contamination which the milk receives in the dairy increases steadily and extensively, in proportion to the degree of its first infection, to the length of time during which it is kept at body temperature and to the period of time consumed in carrying it from the dairy to the consumer.

Thus it will be seen that the proper care of milk demands the greatest precautions in milking, in immediate chilling, and in rapid delivery to the consumer. After it has reached the home, the responsibility of its keeping rests with the housekeeper.

It is self-evident that the milk of diseased animals should not be used for infants. The question whether the milk of cattle which give a positive reaction to tuberculin, but present no clinical evidence of tuberculosis and, especially, no sign of tuberculosis of the udder, is unsafe and one most difficult to decide and is still under discussion. There is no doubt, however, that strict adherence to this rule will increase the price of milk very materially. In the better dairies, which furnish special milk for infants, it has been found sufficient to have the dry udder frequently and carefully examined, say at

least once a month by a veterinarian, thus insuring the timely discovery of tubercles, as well as of the streptococcus infection or mastitis.

Green fodder and silage have to a great extent replaced dry fodder in the diets of dairy cattle. Such feedings must be regulated so as not to cause diarrhœa which increases the difficulty of obtaining sanitary milk on account of the soiling of the udder and the probable introduction of enormous numbers of acid-forming bacteria into the milk.

Where there are local dairies or institutions marketing milk for infant use which are well equipped and carefully managed, the milk should, of course, be procured from them. But the necessity of transporting milk a long distance by rail or wagon in the summer, may make the advantages of sanitary milking and immediate cooling somewhat illusive; so that it is usually better to purchase milk from a near-by source, which may not have been obtained under as sanitary circumstances, or even to secure it fresh from the cow several times a day and to use it immediately.

Where neither the one nor the other method of obtaining pure cow's milk is practical, all the difficulties may be surmounted by keeping a goat. Goat's milk is as satisfactory as cow's milk for infant feeding. Besides, tuberculosis is very rare among goats; the animal is more easily kept clean and the milk is more readily obtained in a sanitary state.

A far-reaching control of milk is possible under a system of municipal inspection. Such control concerns itself not only with examination of the milk as to its dilution, skimming, etc., but it also determines the presence of dirt, the number of leucocytes (Trommsdorf test) and of micro-organisms, and the degree of acidity, according to the method of Soxhlet-Henkel.[7] Since the housekeeper has to depend upon the very indefinite tests of appearance, taste and smell, or finally, upon an experimental boiling, the knowledge of the source and freshness of milk is all the more important to her.

The Pasteurization of milk in bulk, which has been advised so frequently and is quite practical, cannot be recommended for milk intended for infant feeding. In the ordinary milk purchased in the open market, which is often consumed without being boiled, Pasteurization guarantees the destruction of pathogenic organisms; but in milk for infant use, which is always boiled before being used, it hides the important indications of age and insufficient cleanliness by destroying the relatively harmless acid-forming bacteria while the much more dangerous spore-forming peptonizing bacteria (Flügge) remain active. Moreover, as the possibility of subsequent contamination makes the reheating of the milk in the home necessary, it is certainly not a particularly advantageous thing.

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[7] One degree of acidity is the amount of acidity in 50 c.c. of milk which will be neutralized by 1 c.c. $\frac{N}{4}$ sodium hydroxide. Phenolphthalein is used as an indicator. Fresh milk has 2-4 degrees of acidity and remains at this stage (incubation) for from 3-8 hours at body temperature; or at 10° C. for 52-72 hours, depending upon the degree of cleanliness in milking. Milk curdles upon boiling when it has 5.5-6.5 degrees of acidity. Spontaneous curdling occurs in milk of 15-16 degrees of acidity. Thus we are able to determine age and composition approximately by titration. (Plaut; quoted from *Finkelstein's Text-book.*)

The question, actively discussed for years, whether the injuries connected with artificial feeding may not be more or less avoided by giving raw milk, may now be considered answered in so far that no recognizable advantage is seen in feeding the healthy infant raw milk rather than Pasteurized or sterilized milk. The theory that milk becomes unfit for infant feeding by brief boiling, seems most effectually contradicted by the observation that human milk, even after boiling ten minutes, is as effective in relieving seriously ill infants as is the mother's milk unboiled. On the other hand, the view is justified that boiling for a long time (½-1 hour) or very intense heating for even a short time, causes a definite denaturization. Clinical experience, at least, has shown that the excessively sterilized milk of commerce causes anemia and under certain circumstances and if used for a long time produces scurvy.

In the home, only Pasteurization, that is heating to 60° or 65° C, (140 °-160° F.) for 30 minutes or boiling for a shorter time, by the method to be discribed are to be considered. Pasteurization offers no advantage over the boiling of milk for infant feeding and is so awkward in process and so uncertain of results, even with the best apparatus, that it has never become popular and cannot be recommended. The method of choice, therefore, is boiling. It is of no importance whether the whole quantity intended for the 24 hours' feeding is boiled at once, in a glazed or enamel vessel, or whether the separate feedings are boiled in the bottles. The latter method is most readily accomplished by means of the well-known apparatus of Soxhlet. Instead of the patent rubber cover, which is drawn in by the negative pressure as the bottle cools and which seals the bottle hermetically, a metal or glass cap may be placed over the mouth. The time for boiling may be gauged by allowing ten minutes from the first appearance of steam. By sterilizing the separate feedings, every possible contamination by later handling is most certainly avoided; but the likelihood of this is sufficiently reduced by boiling the entire mixture in a kettle, if everything else is cleanly. In this method of minute boiling the milk may be kept from running over by using a double boiler or one of the various milk cookers with perforated cover. In every case, where this method of sterilization, without immediate bottling, is employed, it is essential to cool the food as rapidly as possible by placing the container in running water and keeping it iced. In order to accomplish this, with the use of small quantities of cold water or ice, Flügge has recommended a cooling-box constructed upon the lines of the fireless cooker.

It is self-evident that the physician must frequently prescribe, and often in detail, the methods of maintaining absolute cleanliness of all the utensils which come in contact with the infant's food. Special points should be emphasized. All portions of the food which are left in the bottle, after the child has finished nursing, must be thrown away and the bottle must be washed immediately to prevent the drying of particles which would then be difficult of removal. The nursing-bottle and nipple with rubber fittings and glass tubing should be absolutely prohibited because it cannot be kept clean. The simple rubber cap nipple is best cleaned by washing it

in running water after each use and boiling it once a day. In the intervals the nipple should be kept dry in a clean covered glass or cup, rather than in an antiseptic solution.

THE TECHNIC OF ARTIFICIAL FEEDING

In the practice of artificial feeding from birth, no food is given during the first day of life as with the breast-fed infant. In further imitation of the natural conditions, only three or four feedings are given during the second, third and fourth days, and five feedings only after the fourth or fifth days. Nor should this number of feedings be exceeded later on, the longer time required for the digestion of cow's milk in the stomach making the greater intervals (preferably 4 hours) necessary. Artificial food, given in excess of the requirements of the new-born, is much more dangerous than is human milk.

Since the caloric values of cow's milk and of human milk are approximately equal, it might seem rational to give the infant such quantities of undiluted cow's milk as are taken by the child on breast feeding and this is, indeed, still recommended by certain authors. The preponderant experience of almost all physicians has shown, however that good results may be more certainly obtained by the customary use of milk dilutions.

The researches of the past few years have taught that the advantage of such dilution cannot be laid to the indigestibility of the casein of cow's milk, for this has not been proved. On the contrary, it is equally questionable whether the avoidance of overfeeding with cow's milk of so reduced concentration is the only active factor of benefit. Probably other causes, which cannot be considered here, play an important rôle.

The reduction of the food value which results from dilution may not be equalized by an unlimited increase of quantity, without causing injury through the excess of fluid. It is customary, therefore, to select food substances which may be added to the diluent to equalize or diminish this deficit. Theoretically and empirically, sugar of milk seems to be the most acceptable item for this purpose. Cow's milk, diluted with two parts of water, to which has been added one level teaspoonful (3-4 grams) of sugar of milk for every 100 c.c., (3½ ounces) of fluid, is to be recommended for a two-day-old infant. Even if, at the end of the first week, the infant is taking five feedings of 100 c.c. (3½ ounces), each, of such a preparation, its actual food value is so low that the danger of overfeeding is most certainly avoided. Indications for more or less rapid increase of the food, quantitatively or qualitatively, are gained by observation of the infant in regard to weight, stools and other clinical conditions.

In increasing the volume of the feeding,[8] to meet the indications of need, we have a relatively definite standard in the volume of the daily food taken by the healthy breast-fed infant of normal weight and development.

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[8] If the amount of each feeding is to be measured by means of marks on the nursing-bottle, it is well to be informed as to the accuracy of the markings represented by each line. Even with bottles of the same model it is impossible to depend upon their accuracy.

When we remember that the daily quantities of food taken represent one-fifth of the body-weight during the first week and that they should gradually sink to one-eighth of the body-weight by the end of the first half-year and when we see to it that this physiologic volume of the liquid food is not exceeded to any appreciable extent in artificial feeding, we soon find it necessary to increase the concentration, that is, the food value of the dilution, if the requirement of the child is to be met.

The concentration may be increased either in definite steps by passing from the proportion of one-third to one-half and later to two-thirds of milk, or it may be gradually secured by adding a few spoonfuls of milk without changing the amount of water. The latter method seems to resemble more closely the gradually increasing volume of food which the breast-fed child receives.

Since we do not wish to give undiluted cow's milk during the first month of life, and it has proved empirically undesirable, it will be necessary to increase the food value of the mixture by suitable additions and this is done by adding fats or carbohydrates. The latter need is met, in part, by the addition of sugar of milk as already suggested. The fat content may be increased most readily by the addition of fresh cream, as Biedert first proposed.

Because ordinary centrifuged cream, intended for the use of adults, does not fulfill the requirements of milk for infant feeding in the matter of freshness, low bacterial count and special selection of the cattle from which it is taken, it is better to prepare the cream from well-chosen milk at home. For this purpose the milk should be placed on ice in shallow vessels and be allowed to stand until the cream has separated. After the milk has stood for one or two hours an approximately 10 per cent. cream, or about 100 cc. per litre of milk, may be obtained by careful skimming.

This cream forms the basis for Biedert's "natural cream mixture." For its graduation to the age of the child, he has evolved a scheme which need not be given here because it is not commonly used for feeding healthy infants. It seems more practical, without reference to any particular schedule, to add to the diluted milk and milk-sugar mixture, such quantities of fresh cream as are necessary to increase its food value to the desired degree, as may be borne by the infant without gastro-intestinal disturbance. Additions of cream exceeding a total 3 per cent. of fat in the whole mixture are hardly ever beneficial to the infant.

The ordinary proprietary preparations present absolutely no advantage, either theoretically or in their practical results in the feeding of the healthy infant, over the milk and milk-sugar dilution mixed with fresh cream. That they receive high recommendations and are frequently prescribed by midwives and physicians is due to the facts that they are sold with directions on the container and that no special knowledge of pediatrics is necessary in dispensing them. The thoughtful and conscientious physician robs himself in their use of the opportunity of changing the value of the food by varying its individual components. And this is a necessary opportunity, because the mere fact that the coarse chemical composition of such manufactured

food is roughly similar to human milk does not make it at all the equal of breast-milk in value. It is useful only with those children who would do equally well upon a simple milk dilution sweetened with sugar. In many other children, the high fat and sugar content, which is borne without the least difficulty in the form of human milk, causes, with these preparations, diarrhœa and vomiting and permits only slight increases in weight which are not improved by increasing the quantity of the food.

Recently Czerny and Kleinschmidt have put forth a very valuable method of utilizing butter fat in infant feeding by their method of preparing the "Butter Flour" mixture. By this method it is possible to feed large amounts of fat to very young and poorly nourished infants and those which are difficult to feed by other methods. It may be continued for long periods and the gain of weight and general development is comparable to that of the breast-fed infant.

The food consists of a basic diluent prepared as follows: Seven grams butter are melted over a slow fire allowing it to fry until all the volatile oils and free fatty acid is evaporated. Then seven grams wheat flour is stirred in, allowing the mixture to brown a little. To this mixture is added 100 c.cs. boiling water in which five grams granulated sugar has been dissolved. This is brought to a boil, strained to remove any lumps and cooled.

A. Graeme Mitchell recommends the following practical formula: Butter 2 level tablespoonfuls, flour 2½ level tablespoonfuls, sugar 1½ tablespoonfuls and water 10 ounces. The proportion of this mixture is constant and equals 26.6 calories per ounce.

For children of less than 3000 grams body-weight, one-third milk is added and for larger infants ⅔ to ½ milk. The milk should be certified grade or Pasteurized. The butter is sterilized by the heating. The authors advised 200 c.c. per kilo body-weight per day.

Various sorts of sugar and flour in the form of gruel, may be used to increase the food value of diluted cow's milk and, in particular, to enlarge its carbohydrate content. Of the former, sugar of milk has been repeatedly mentioned. Soxhlet, Heubner and Hofman have recommended its use in such quantities that the food value of the milk, lost by dilution will be restored. To do this, it is found necessary to use concentrated solutions of sugar which decidedly exceed the physiologic sugar content of human milk (6-7 per cent.)[9] It is doubtless possible to feed many healthy infants successfully with mixtures containing so large percentages of sugar of milk, but it must be emphatically said that many infants will not tolerate it and that it will not produce satisfactory increase of weight. For this reason, it seems advisable to limit its addition so that the entire mixture shall not contain more than 6 per cent. inclusive of the 4 per cent. of milk-sugar contained in the cow's milk itself and to make up any deficit, if necessary, by concentrating the milk or by adding flour.

Cane-sugar, because of its sweetening power, has played an important rôle in the artificial feeding of infants, even before the science of pediatrics

[9] Milk-sugar is the only sugar that may be used in such quantities without making the solution nauseatingly sweet.

was established upon a scientific basis. In young infants it is better to avoid it and to substitute milk-sugar because of the readiness with which cane-sugar ferments; but in older children, if it fall short of such concentration as to make the food sickeningly sweet, it is often well tolerated.

Malt sugar (maltose) is used in the form of the various malt extracts which also contain dextrin, nitrogenous constituents, water, etc., but it is not commonly given to the healthy child. In the feeding therapy of sick infants, it plays an important part.

The gruels, representing the insoluble carbohydrates (polysaccharides) may be used even for very young infants. These gruels are prepared by long continued boiling (½-1 hour) of either oatmeal, rolled oats, rice, cracked or crushed barley, etc. The quantity of each of these cereals required for preparing a suitable gruel cannot be definitely stated because of the variability of the manufactured products. A thin gruel should remain liquid when cooled, while a thick gruel should gelatinize. The various gruels do not in themselves present any essential differences in their use for infant feeding. They are distinguished from the flours in that the former do not consist so largely of starch, but contain, also, a variable but greater quantity of vegetable protein. The amount of solids contained is low, especially in thin gruels, and consequently their food value is small.

Of the flours, we must consider oatmeal, which is distinguished for its content of over 5 per cent. of fat, wheat flour and corn flour. Even though a large amount of undissolved residue always remains in the preparation of gruels, which should be separated by pouring off the supernatant fluid or by straining the gruel, the mixture after boiling for 10 to 20 minutes, contains exactly the same amount of food material in solution or rather in a colloidal state, as was added in the beginning. The food value of these gruels is therefore, high. Corn flour considerably exceeds the other cereals in its solubility, so that a 2 per cent. gruel of corn flour equals a 5 per cent. gruel of wheat or oat flour in its consistency.

According to the experimental researches of Klotz, the various flours show important differences in their intestinal digestion as well as in their intermediate metabolism. It would appear that these differences do not permit the several flours to be used interchangeably in artificial feeding; but as yet no definite clinical observations are at hand. Nevertheless, a change from wheat to oat flour, or *vice versa*, if the child is not doing well may be justified. All flours have an extremely low content of mineral matter, so that the addition of a small amount of table salt (.3-.5 per cent.) to all gruels is necessary.

In the practice of artificial feeding, it is well to avoid the addition of flour to the food of the new-born or of infants in the first two or three months. I feed cereal waters at the end of the first month and believe they are well digested and beneficial. The gruels are used in very thin form at first and are gradually thickened, and this only if sugar of milk cannot be used successfully which is often possible in healthy children up to the sixth or seventh month.

Certain limitations, which it is dangerous to exceed, are set to the addi-

tion of flour, even after the third or fourth month, because of the limited amylolytic function of the infantile digestive tract and the too great density of concentrated flour preparations. Further increase of the food value may be met by the use of sugar.

Toasted and baked flours in which the starch is partially dextrinized by the heat and is thus changed into a soluble form, stand in close relation to the simple flours. According to all observations recorded to the present time (Hedenius), they are not digested any better by young infants, but rather not so well, as are the pure flours. Some toasts, especially prepared for infant feeding, contain added salts, notably calcium phosphates, but no advantage attaches to this because they are given only at an age when the child receives the required mineral substances in soups, vegetables, or fruit, and in much more natural form and concentration. The numerous proprietary infant foods should be avoided in the feeding of healthy children, as a matter of principle, because, on the one hand, they serve no better than simple flour or sugar and because advertised as the best or the only substitute for mother's milk, they are responsible for many dangerous dietetic errors and for their invitation to the weaning of many children whose mothers are able to nurse them.

A peculiar position is occupied by condensed milk, which is still in very general use. It is whole milk which after the addition of large quantities of cane-sugar, is sterilized and condensed to a paste-like consistency. Certainly it is no better for feeding healthy infants than fresh sweetened milk dilutions. It should be given only temporarily when other milk cannot be obtained and even then it is a fair question whether the excessive amount of sugar present may not be harmful to the child.

The food requirement of artificially-fed children may be taken as approximately the same as that of breast-fed infants of the same age and weight. Even though the metabolism of the breast-fed infant is probably maintained more economically as we have shown in the first chapter, it must still be remembered that the breast-fed infant is able to stand a considerable amount of overfeeding which, in the artificially-fed would result in injury. We may, therefore, transfer the caloric requirements, cited for breast-fed infants on page 22, directly to the artificially-fed child and may safely go even below them.

The following caloric values of the foods, commonly used, may serve for comparison:

	Calories
Whole milk per litre	700
Cream 10 per cent. per litre	1300
Flour (100 gms.)	400
Sugar (100 gms.)	400

From these few equivalents, the caloric value of a food mixture may be approximately calculated and from these calculations we may draw conclusions as to whether the daily supply of food contains the required number of calories. By way of example: An eight weeks' old infant weighing 4500 gms., may receive five feedings of 280 c.c., or a daily total of 900 c.c., of a mixture consisting of one-half milk, one-half water and 20

grams of sugar of milk. The food value of this mixture is 315+80=395 calories. In this mixture, there would be 55 calories less than the required 450, a deficit which could be made up by six grams of fat contained in 60 cc. of 10 per cent. cream, or by 14 grams of sugar. Since the total sugar content would be 18 grams contained in the milk, +34 grams added, =52 grams or 5.8 per cent., which still lies within permissible limits, the latter would be the easiest way of adding the required calories.

While, on the one hand, this mathematical method aids in the avoidance of definite errors,[10] which are not uncommon in the choice and dosage of artificial food mixtures, yet, on the other hand it does not follow that food known to contain sufficient calories will necessarily produce the proper development of the child. The limitations of the usefulness of the caloric method of infant feeding have been already discussed in Section I.

It is impossible, therefore, to formulate a schematic table for the feeding of the healthy infant, much as this trouble and thought-saving device might serve for the busy physician. In the difficult field of artificial infant feeding, which requires both a knowledge of theory and the practice of clinical experience, no goal can be successfully reached without scientific observation and careful consideration of each individual case.

The French obstetrician, Budin, has achieved great renown by his researches in the physiology of infant feeding. He has instituted the so-called "Budin's factor," which gives a reasonably safe standard for the quantity of milk to be allowed. He proposes that the artificially-fed infant receives about 10 per cent. of his body-weight in cow's milk daily. To this quantity of milk, which does not fully meet the caloric need, may be added cream or sugar, or sugar and flour to balance the requirement.

Pfaundler has devised a similar schematic formula; as follows: "Take the tenth part of the infant's body-weight in cow's milk and add to it one-hundredth part of its body-weight in carbohydrate, not exceeding, however, 50 grams a day; add water to make 1 litre. Divide into five feedings and let the child take as much at each feeding as it will. In young infants the carbohydrate may be given in the form of milk-sugar or dextri-maltose; in older children, in the form of a 2 to 3 per cent. oatmeal gruel or a 3 to 4 per cent. flour paste." This formula will not apply in all cases. The caloric requirement of a child fed with food prepared according to this prescription is met only when the child takes relatively larger quantities than a breast-fed child of the same weight will do. This is usually true if the feeding intervals are not intentionally shortened.

In the application of the rules for the feeding of normal healthy infants it must be remembered that each infant must be fed to meet its individual requirements. If milk dilutions with the addition of carbohydrates are used, the simplest and most natural standard would be one that would tell us how much milk and carbohydrates per pound or per kilogram body-weight the baby should get. To be exact we should express, or at least be aware of the number of grams of protein, fats, carbohydrates and salts that the infant is receiving for each pound of its body-weight. We believe that

[10] Especially the errors of underfeeding.

if statistics on infant feeding were collected on this basis rather than in percentages of the ingredients in the milk mixtures (the total mixture being of such variable quantity) the collected data would be far more valuable as a basis for future work in infant feeding.

The average infant fed on cow's milk will metabolize advantageously 1.5 grams of protein, 1.5 to 2.0 grams of fat, 4.0 to 6.0 grams of carbohydrates, including that contained in the milk and cereal for each pound of body-weight.

Feeding the following amounts of the milk and carbohydrates will approximate these requirements and furnish between 43 and 55 calories per pound body-weight:

To meet these protein and fat requirements, the average normal infant will require each day a minimum of 1½ ounces (45 milligrams) of cow's milk per pound of body-weight.

Infants under five months of age will frequently require amounts approximating 2 ounces (60 milligrams) of cow's milk per pound body-weight, except during the first few weeks of life when smaller quantities of whole or skim milk are indicated.

In beginning feeding with cow's milk, mixtures must always be started as weak formulæ, more often using only 1 ounce (30 milligrams) of cow's milk to a pound body-weight, gradually increasing the strength to meet the infant's needs.

Underweight infants should at first be fed according to their present weight, gradually increasing the strength of the mixture as rapidly as consistent with the baby's ability to handle the diet, and thus approximating the needs of a full weight baby of the same age. These babies will frequently take over 2 ounces (60 milligrams) of milk per pound body-weight.

With the institution of a mixed diet, the infant thrives with less milk per pound body-weight.

From birth to the fifth month the average healthy infant may be satisfied with an amount of food approximating 2 ounces more per feeding, than the infant is months old.

In our own experience we have found that a concentrated milk mixture does not disturb the infant's digestion when the milk is boiled or alkalinized.

The amount of water is calculated by multiplying the number of feedings by the amount of each feeding, and subtracting the milk to be given.

Having the necessary amount of milk and water, we ascertain the carbohydrate to be added.

Cane and milk-sugar are added in such quantities that the normal infant in its food mixture receives a total of from 4 to 6 grams of carbohydrates per pound of body-weight per day, including that contained in the milk in the mixture. As 1½ ounces of milk contains approximately 2 grams of lactose it will be necessary to add from 2 to 4 grams of carbohydrates for each pound of body-weight to the diet besides that contained in the milk. One-tenth of an ounce (3 grams) of cane or milk-sugar per pound body-weight answers the needs of the average normal infant in its first months.

Cereal water may be added to the diet after the infant is one month old. One-sixtieth to one-thirtieth of an ounce (0.5 to 1.0 gram) of starch for each pound of body-weight may be added to the mixture. This is best given in the form of cereal waters or well-cooked cereals.

We find it especially valuable in those cases in which we are feeding cane-sugar, and in which the infant takes a dislike to its food because of the intense sweetness of the mixture.

In underweight infants the amount of sugar and cereal to start with should be calculated on the basis of the present weight, approximating the quantity needed for a full weight infant as rapidly as the sugar tolerance permits.

When more than one quart of milk mixture is needed to properly nourish the infant, the age has been reached when a mixed diet should be instituted. No infant should be fed more than one quart of cow's milk in 24 hours.

It is to be remembered that the amounts of food recommended are relative and must be increased or decreased according to the infant's progress and individual needs.

The transition to mixed feeding is conducted similarly in method with that employed in breast-fed infants, in that the child is given cereal soup prepared with broth at the end of the sixth or seventh month, this food being soon followed by the use of vegetables.

In estimating the result of the feeding, increase in weight remains the most important index only during the first few weeks or months. The older the child, the more particular stress is to be laid upon other factors. This is to be especially emphasized because of the many errors that are made in this respect in the nursery since the practice of daily weighing has become popular. Of more consequence than the absolute gain in weight, which usually depends upon the size of the child and the rapidity of its growth, are the steadiness of this gain, the muscular tone, the turgor, color, bone development, the adequate sleep, the good disposition, the regularity of the bowels and bladder, etc. Excessive fattening should be especially avoided in artificially-fed children, because such constitutional diseases as rickets, spasmophilia and the exudative diathesis, take a more severe course in fat children than in moderately nourished infants.

During the second year, the child's protein requirement should still be largely met by the feeding of milk, of which ¾ to 1 litre (a pint and a half to one quart) a day is quite enough. During this period, indeed, even such quantity may be gradually reduced to half a litre (1 pint) and replaced by other foods. As in the first year, no milk should be given at noon either with the meal or after it. The menu should be selected according to the recommendations on page 51; but with greater latitude in the use of vegetables. For breakfast and in the afternoon, the child should receive 200 c.c., or at most 250 c.c. (7 or 8 ounces) of milk, with as much bread as it likes, but without butter. In the evening, a similar quantity of milk may be given with a cereal (rice, cornmeal, etc.). During the morning, a light lunch, consisting of 100 to 150 c.c. (3-5 ounces) of milk, may be served; to

which, after the middle of the second year, a thin slice of bread or a roll, with a little butter and fresh or stewed fruits, may be added.

At about the end of the second year, the quantity of milk should be gradually reduced further, until the child receives only a small supply with its breakfast, when the protein requirement may be met by giving a little meat and egg. In view of the much greater concentration of the protein in these foods, only small rations of them are required in addition to the vegetables, legumens and fruits necessary, not only to satisfy the appetite but for the normal mass formation of the feces and for the maintenance of a positive alkali balance. Thus the diet of a 3-year-old child gradually approaches that of the adult so closely that particular directions no longer seem necessary. Strong spices (mustard, pepper, etc.), do not appeal to the taste even of older children and naturally no one should attempt to accustom the child to them.

THE CARE OF THE CHILD

The care of the child during the first months of life and even throughout the entire first year, provided it be healthy and free from constitutional anomalies, is so far secondary to the problem of feeding that breast-fed infants, in poor and unhygienic surroundings, often thrive and develop better than do the artificially-fed who are cared for, according to all the rules of hygiene, in the homes of the wealthy. That despite of this warranted assertion, the value of proper hygienic care is not be under-estimated, is shown by the greater morbidity and mortality among artificially-fed infants of the poorer classes of society, as compared with the children of the educated and well-to-do. After the completion of the first year, when the feeding is no longer of preponderant importance, the general care in its broad applications is of larger influence upon the health of the child.

One of the first rules in the care of infancy and childhood is cleanliness. At the outset, it involves the asepsis and uneventful healing of the umbilical wound. The mummification of the umbilical stump is best accomplished by the use of such drying powders as bismuth subgallate, talcum with sodium salicylate, sterilized bolus alba and the like, and by wrapping the stump in sterile gauze or absorbent cotton. Ointments and moist dressings are undesirable. Certain authors advise that the daily bath be omitted after the first cleansing until the umbilical wound has healed.

Until the end of the first year, or until the child has acquired habits of cleanliness, it should be bathed daily. The temperature of the bath should be about 35 ° C. (95 ° F.), at first and may be decreased by 2 °-3 ° C. (3°-5° F.), as the child grows older. The bath should not be continued longer than is required for the careful cleansing with soap and water of the entire body and especially of the skin folds and the anal region. After the bath, the parts mentioned should be scrupulously dried and dusted with an inert powder (salicylic or zinc-oxide powder, or even rice powder or starch). The face and especially the eyes, should be washed with fresh luke-warm water either before or after the bath. All attempts at mouth cleansing should be forbidden. They are not only unnecessary but harmful

on account of the injury to the mucous membrane which may result (Bednar's aphthæ). After all the incisors have appeared, we may recommend that the teeth be cleansed, at first with a soft linen cloth and later with a soft brush. Early defects should be looked for, because the timely treatment of the milk teeth preserves them longer and gives better opportunity for the development of the permanent set.

The deplorable custom, so widespread, of permitting the child to form the habit of sucking a pacifier must be forcefully combated. The pacifier is not only unesthetic but dangerous, on account of the frequency with which it serves as the carrier of dirt and disease germs from the pockets of the clothing, from the mouth of the mother, who at times moistens it with saliva, and from the floor upon which it may fall, etc. Czerny's statement that there are constitutionally restless, neuropathic infants who are soothed most readily by the pacifier—and harmlessly, provided it be kept clean and does not contain sugar, is true; but the number of such children is small and does not justify the widespread abuse of an undesirable method of quieting a child.

The infant should be changed before each feeding and should be washed, dried and powdered carefully every time it is soiled by a fecal discharge. In cleansing the anal region in girls, the parts should always be cleaned from before backward, since particles of fecal matter may easily be carried into the gaping vulva and especially into the urinary meatus, eventually causing infection. At the slightest indication of chafing it is well to cover the parts with oil or petrolatum, or better with a thin layer of zinc oxide ointment, to protect them from the irritation of repeated wetting.

The healthy infant maintains his normal temperature when he is dressed in a shirt, gown and diaper and is covered with a light down quilt or with a linen-covered wool blanket. Applications of artificial heat, in the form of water bottles and the like are not necessary. The room temperature should be 19° to 20° C., (66° to 68° F.) as nearly as possible, and may be even a little less for older children. The child should, under all circumstances have its own bed.

The use of the cradle has rightfully gone out of fashion, for children are easily spoiled by becoming accustomed to the quieting rocking movement and are broken of the habit with difficulty. The unquestionably injurious effect of rocking, belief in which rests upon purely theoretic considerations, has yet never been demonstrated.

If care is taken to provide for the plentiful supply of fresh cool air to the nursery by the necessary oft-repeated and complete airing of the room, the initial attempt to take the infant out of the house in winter may be delayed without harm to the child. Older infants may be warmly dressed and taken into the open daily, even in winter when the temperature is mild. Later, when the child can go about, it is better to let it walk and run than to take it out in the go-cart. Even wet feet are not injurious so long as the child is in motion. Dry shoes and stockings should be put on, however, so soon as the child gets home.

Through the first half-year, the child should be taken up only for some such definite purpose as bathing or feeding; and otherwise should be left to itself even when it is awake. During the second half-year it may be held and allowed to sit up on the arm or in the lap. This gives it a free survey of its surroundings and exercise for its muscles while the natural tiring which results, produces sound sleep. The first attempts at standing and walking should not be hastened, nor should attempts be made to retard them, since no dangerous effect upon the back or legs is to be feared. The strengthening of the musculature, indeed, rather counteracts the tendency to deformities. In carrying an infant, care should be taken to hold the child first on the right arm and then on the left in order to prevent the development of scoliosis. If the child is permitted to sit up in bed, he should be laid down again as soon as he betrays by any abnormal posture the first sign of tiring. When the child is learning to walk, a pen gives him the best

Fig. 10.—Nursery pen (Feer).

outlet for his activities. The floor of the inclosure may be formed of a soft smooth cover, which should be kept scrupulously clean. The walls should permit the child to look freely out as in the nursery pen of Feer, shown in Fig. 10.

The habit of cleanliness is dependent upon the attention and care devoted to accustoming the child to the control of bowels and bladder. It may be earlier developed, therefore when an efficient nurse has the entire care of the infant and is relieved of other duties. At about the sixth month, at latest, the child should be taken to the toilet or placed upon a wide-rimmed vessel for the purpose of moving the bowels and emptying the bladder. Further, the child should be watched and waited upon when it expresses a desire. The rapidity with which results are obtained depends, in part, upon the skill of the nurse and in part upon the individuality of the child and it therefore varies greatly. In general it may be expected that with ordinary care the child of normal development will give evidence of its bodily needs by the end of the first year, and that within a short time later

it will learn to make known its desire. Even then, it may take several months before full control of its habits is established. If proper care is given to the child and success is not attained by the third or fourth year pathologic conditions must be suspected.

The habit of obedience in the child and of subordinating its wishes, tendencies and desires to the direction of its elders is of frequently underestimated importance in the entire future training of the child. This is secured the more readily by a quiet and purposeful demeanor upon the part of its instructors, whereas the child is confused and injured by moody treatment and may easily acquire an unpleasant and unreasonable disposition.

The training of the child should begin with the day of its birth, and the method by which this is best accomplished is classically described by Czerny in his lecture "The Rôle of the Physician in the Training of the Child." (Deuticke, Leipzic and Vienna; 1911, 3rd edition.)

III. GENERAL SYMPTOMATOLOGY AND TECHNIC OF EXAMINATION

REVISED BY

L. R. LeBUYS, B. S., M. D., F. A. C. P.,

Professor of Diseases of Children, School of Medicine, Tulane University of Louisiana, New Orleans.

THE technic of the examination of children can be acquired and the valuation of its findings appreciated only by practical demonstration in the clinics. The instructions in the following chapter presuppose that the student has mastered the methods of internal medicine. Only such methods of examination are described as are specific to the child and especially to the infant. A few suggestions as to variations in the interpretation of certain findings are offered.

The history of the case should be taken before the examination is made. In estimating the value of such history, careful distinction must be drawn between the parents' actual observations and their attempted explanations of events which often lead to misinterpretation, since they are based upon preformed ideas. Indefinite statements as to symptoms—as for instance in the matter of convulsions, the indefinite term applied to varying and heterogeneous symptom-complexes—should be cleared up or elaborated by precise questioning. If possible, the question should be asked so that the mother does not have to answer by a statement of opinion or by a description of events, but rather numerically or by yes or no. I have to thank my teacher, the late Richard Förster, the ophthalmologist, for the suggestion of this method. It was the result of his long medical experience. By this means a probable diagnosis will soon be foreseen, which later may be confirmed or modified by the complete history or by the examination findings. As compared with the experience of the adult, there is a monotony in the disease-picture in the infant which makes this method of diagnosis easier. This fact depends upon the comparative rarity of many disease conditions in infancy which many be excluded from the first.

The family history with its hereditary tendencies and contributing factors, as also the history of the previous illnesses, particularly those which may predispose to subsequent ailments, are frequently of considerable value. The history of the present illness with its mode of onset and the appearance of its symptoms is essential to a proper conclusion.

Even in taking the history, the physician is often able to form a certain impression of the child by casual observation, which is all the more valuable because in the young child self-consciousness and intentional deception do not play any confusing part. The posture and behavior of the child, the expression of his face, etc., may give valuable diagnostic hints. Soltmann, in a brilliant study of the facies and facial expression of the sick child has indicated many peculiarities which cannot, however, be discussed here.

The actual examination should be thorough and the beginner should proceed according to a definite plan. It is advisable to examine first that part of the body or that organ to which the history points. The parts of the examination which tend to excite the child, to cause it to cry or to resist the examiner, as for instance, the examination of the throat should be left to the last.

The color, respiration and pulse may be studied and an estimation of the temperature may be made by the hand before the child is undressed, an event which may, in a measure, disturb him. For complete examination the child should always be stripped; infants completely and at once, so that the entire body may be surveyed.

At this time observation should be made upon the skin, state of nutrition and development of the patient, condition of the muscular and glandular and bony systems. The existence of abnormalities should be looked for and a thorough inspection of the external genitalia and anus should be made. The motility of the neck and spine should not be overlooked.

After this he may be again covered as examination, part by part, permits.

In older children, the consideration of modesty may make it necessary to undress them little by little as the examination proceeds.

The examination should begin with the determination of the temperature which for accuracy in the infant should be taken by rectum. The child is laid upon his side and his flexed thighs are held with the left hand while with the right the thermometer is inserted into the rectum as far as the lower edge of the scale. Another position is one in which the patient is placed prone upon the lap of the nurse with his feet hanging over. The thermometer is inserted into the rectum as usual. This position is especially adapted to the resisting child. It should be held in place several minutes, the so-called "minute" thermometer requiring 2 or 3 minutes and others 5 minutes or longer until the column of mercury remains stationary for 1 minute. With an accurate minute thermometer the allowance of an extra half minute should be all that is required. It is unnecessary to lubricate the bulb of the thermometer. Even though the child lies perfectly still, the nurse should be warned against fastening the thermometer in place with a diaper or in any other fashion, and leaving it there, since an unexpected movement on the part of the child may break the thermometer and cause injury to the rectum.

During succeeding years the rectal temperature remains the most exact, although after infancy in very unruly children it may be taken in the inguinal fold, where the thermometer is placed between the strongly flexed thigh and the abdomen. After the eighth or tenth year, in children who are extremely emaciated, the axillary temperature may be accepted as fairly accurate. It should be remembered that in infants the cooling effect of a long journey, as for example to the physician's office, is often sufficient to conceal, for the time being, a high temperature.

The number of times the temperature should be taken and the hour of taking it should be prescribed by the physician. Usually it is taken in the

morning and in the afternoon. The normal range of temperature has been discussed on page 14.

In counting the respiration and the pulse, as in the adult, the attention of the child should be averted, if possible, so that satisfactory results may be obtained while he is quiet. The most accurate information is obtained while the patient is asleep. The only quality that can be definitely determined in the peripheral arterial pulse is the frequency. When awake the pulse may be considerably accelerated by excitement or upon the approach of the physician or a stranger.

Conclusions as to the force of the heart-beat must be determined by direct auscultation of the heart sounds.

The size, weight, growth and development should be carefully observed and compared with the normal average, inasmuch as nutritional disturbances may influence these factors considerably. These disturbances with their far-reaching effects should be constantly kept in mind for their influence upon the future of the individual.

Head and Neck.—In palpating the back of the cranium for craniotabes, the head is taken firmly between the palms of the hands which are applied flat to either side of it leaving the fingers free for palpating movements to discover soft spots. The same method is employed in palpating all other parts of the head and especially the great fontanelle. In estimating the tension of the latter, it must be remembered that crying and straining of the child may increase the pressure even under normal conditions. The size of the fontanelle should be measured diagonally, the results being stated either in centimeters, inches, or finger-breadths which latter usually is sufficiently accurate. The size of the cranium is commonly measured only in the greatest horizontal circumference and is considered in comparison with the circumference of the chest, measured with the arms extended, immediately below the scapular angles and over the nipples. In the newborn the circumference of the head is the greater, but at one and a half years the circumference of the chest becomes equal and never again falls below it.

The following table gives several average measurements:—

	Circumference of head.		*Circumference of chest.*	
	Centimeters	Inches	Centimeters	Inches
End of the 1st month	35.4	13.95	34.2	13.46
End of the 6th month	42.7	16.8	41.0	16.1
End of the 12th month	45.6	18.0	46.0	18.1
End of the 2nd year	48.0	18.9	47.3	18.6
End of the 3rd year	48.5	19.0	48.0	18.9
End of the 4th year	50.0	19.68	49.0	19.2
End of the 6th year	50.9	20.0	54.8	21.5
End of the 9th year	51.7	20.35	60.2	23.7
End of the 12th year	52.3	20.58	65.0	25.6

For the inspection of the throat the child is laid upon the bed or upon the nurse's lap, his hands being held firmly; the physician grasps the head from behind with his left hand and introduces the tongue-blade or spoon-handle with his right. This gives him full power to turn the head of the child so that the light will fall into the mouth. If the child bites the tongue-blade the physician waits patiently, pressing gently upon the blade until the

child lets go, and then pushes the instrument rapidly toward the posterior pharyngeal wall keeping the tongue well down. The touch of the instrument on the walls of the pharynx will stimulate the gagging reflex and force the child to open the mouth wide.

Probably the most satisfactory ways in which to hold the patient for the examination of the throat are as follows:—

(1) Hold the patient with his back against the chest of the nurse with the back of his head against her right shoulder. Her left arm is held firmly about the patient against her over the patient's lower abdomen. Her right arm held firmly also, is placed around the patient's arms at about the level

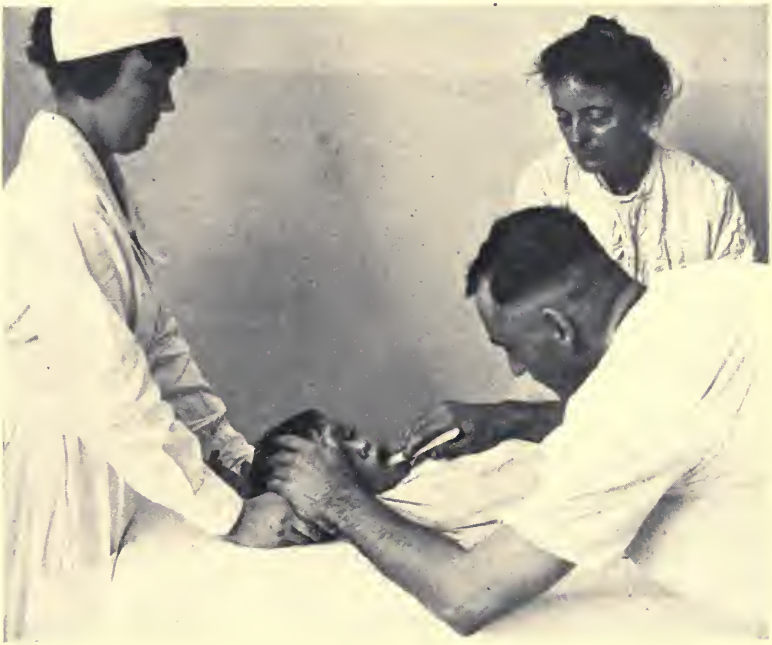


FIG. 11.—The depressor is introduced just far enough to hold the tongue down.

of his elbows, pinioning his arms tightly against his sides. The head is then grasped by the examiner's left hand and may be moved in any direction desired, when the tongue depressor is introduced into the mouth (Fig. 12). The depressor is introduced just far enough to hold the tongue down out of the way so the examination may be clearly made; in this way a definite idea is had of the conditions existing. If it is desired to cause gagging the tongue depressor may be passed further back so as to produce the desired effect. This method of holding the patient may be employed either with the nurse sitting or standing. The other method (2) is employed when the patient is too ill to be raised out of bed or if the examiner, for any reason, wishes to make the examination with the patient in the recumbent position. The patient is placed flat on his back, his arms are extended horizontally

above his head on a line with the bed. The nurse holds the arms tightly so that the head is secure between the arms (Fig. 11). The examiner proceeds as above. In both methods the feet may be readily controlled. Direct lighting or reflected light may be employed.

Holding the nose, or any other attempt at force is unnecessary and has a very bad effect on the parents. The tongue-blade to be used, in preference to the common thick glass form, is the smooth metal spatula best made of nickel which does not rust, or of nickeled wire according to the description of von Pirquet. Wooden blades, which may be thrown away after a single use, are very clean, but if not carefully prepared and the edges left rough may injure the mouth of the child who resists examination. When it is necessary for further inspection or digital examination a mouth gag may be used. The Dwyer, or the self-retaining Whitehead model will serve.

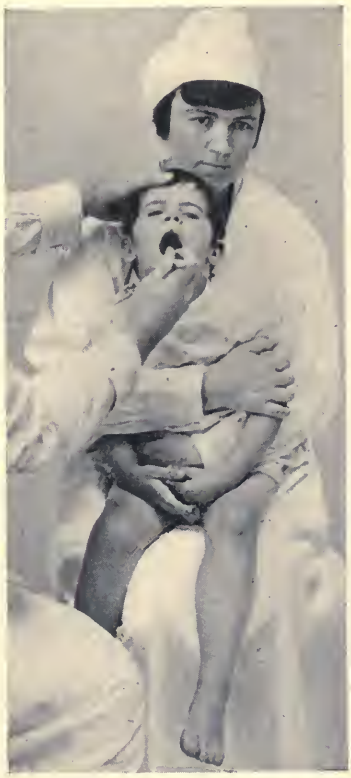


Fig. 12.

In making a digital examination for the determination of the existence of adenoids, the patient is held with his head pressed firmly against the left side of the examiner by the examiner's left hand; the forefinger of the left hand of the examiner presses the left cheek of the patient in between the teeth when the mouth is open. The right forefinger is then passed into the mouth and behind the soft palate making a thorough examination of the pharynx. The tendency of the patient is to close the mouth. In doing so, however, the teeth come in contact with the inner wall of the cheek and this affords protection to the examiner's right forefinger (Fig. 13.)

Laryngoscopic examination is difficult in young children. According to Roth, the mirror must be set at a more acute angle (100° instead of 135°) than in the adult. Many, however, prefer the latter. Anæsthesia of the throat with cocaine, novocaine or the like can hardly ever be avoided. Bronchoscopy according to Kirstein's method is possible only with extra small instruments and after much practice; even then it fails with some infants. In case of necessity, the examination must be done under complete anæsthesia.

According to Lynch, anæsthesia of the throat for laryngoscopic examination is never necessary, on the other hand, bronchoscopic examination should always be done under general anæsthesia and the latter examination should never fail, instruments smaller than those ordinarily employed in the adult, being used, but not extra small ones.

Anterior rhinoscopy and otoscopy is performed as in the adult, but of course with relatively small specula. It is usually necessary to remove all particles of cerumen from the infant's small auditory canal. The sharp angle of the tympanic membrane to the direction of the canal makes the picture less definite. However, by pulling the lobe of the ear downward, thereby straightening the external auditory canal, the examination of the tympanic membrane is considerably facilitated.

Pressure upon the tragus is usually sufficient to give indication of the presence of an acute painful inflammation of the external auditory canal, but at this point, as in all examinations to determine the presence of pain, one must be certain that the pain is local and is not stimulated by fear or excited by the condition of the child.

The examination of the eye does not offer any technical differences from that employed in the adult. The ophthalmoscopic examination is best made with the child lying upon his back on the bed or upon the nurse's lap. If necessary it should be done after dilatation of the pupil with homatropin. The lids may be separated by the retractor.

Fig. 13.—In making a digital examination for determination of adenoids hold the patient with head pressed firmly against left side of examiner.

Chest

The Examination of the Heart.—The apex beat in the infant is not normally visible and can be felt but slightly in the fourth intercostal space, half a centimeter outside of the nipple line. With increasing age, it gradually becomes visible and changes its position so that after the second or third year it is found in the fifth intercostal space, continually moving medially until puberty, when it has taken the position, normal to the adult, inside of the mammillary line.

Bulging of the precardia should require the exclusion of cardiac disease, as it is a frequent finding in such conditions in early life.

Percussion of the cardiac area of the infant gives but indifferent information as to the size of the heart and permits of definite conclusions only in the event of great enlargement. Mediate percussion with the fingers should always be employed and should be very light. It is best done with the child in a sitting posture, since when the child lies on his back the heart sinks away from the thoracic walls. In the infant, the relative dulness extends one-half centimeter to the left of the mammillary line to the second rib above,

slightly beyond the right sternal margin on the right and to the fourth intercostal space below. The absolute dulness covers only a small area to the left of the sternal margin. The relative dulness diminishes with increasing age, becoming narrower to the right and extending by one intercostal space in the downward direction, corresponding with the gradual sinking of the diaphragm after the second to the third year. The absolute dulness enlarges, especially to the left, and correspondingly downward.

Exploratory puncture of the pericardial cavity is seldom employed for diagnostic purposes, but may be done as a therapeutic measure.

A slight sternal dulness over the manubrium may be frequently demonstrated and is termed the thymus dulness. It is separated from the upper border of the heart dulness by a zone of clear lung resonance. In my experience the dulness of the thymus merges into the cardiac dulness. More intense sternal dulness is undoubtedly always pathologic and may be accompanied by a bulging of the upper segments of the sternum.

Auscultation gives more definite results. In order to localize the individual tones and sounds accurately, it is well to use an instrument rather than the naked ear. In applying a solid stethoscope to the soft thoracic wall any pressure should be avoided that may tend to make the child restless. In the use of a tube stethoscope the sounds are subdued and less distinct. With the phonendoscope, or any similar binauricular the sounds may be heard very distinctly, but the user must grow accustomed to the instrument in order to be able to exclude extrinsic sounds. The heart sounds of young children are as a rule relatively louder than in the adult and more sharply defined and sudden. At the apex the first sound is distinctly the more prominent while the predominance of the second sound at the base, physiologic in the adult, is scarcely or not at all demonstrable. On account of their volume, the heart-sounds may often be heard distinctly outside of the heart areas, and at times, even over the back. If the respirations are especially rapid, while auscultation of the heart is being attempted, and this can hardly be avoided in nervous children, the so-called heart-lung sounds may be mistaken for actual heart sounds. These complicating sounds are hardly ever present in infants. In children, who breathe during the auscultation of the heart a slight imperfection or splitting of the sounds, and especially of the first sound at the apex, is common, but is of no diagnostic significance. As a rule, endocarditic disease and resulting organic lesions are rare in infants. This is true, also, of accidental sounds. Every loud heart murmur should, therefore, arouse suspicion of a congenital lesion. This infantile peculiarity disappears progressively between the fourth and the sixth year.

Direct auscultation is most simply done with the naked ear and serves well in infants, during the first month, in determining the force of the heart. The examination with the stethoscope is much to be preferred. If the force is lowered, the sounds become duller and more subdued until finally only a dull tone, synchronous with the first or muscle sound, is heard at the apex. In the young infant, this method of examination takes the place of the examination of the radial pulse which, on account of its

smallness, does not permit definite determination of arterial tension volume, etc.

Blood-pressure, measured with the usual instruments, gives good results only with older children and should be interpreted with great caution. Its practical diagnostic value is slight.

During the examination of the lungs, as in the examination of the heart, the child should be as quiet as possible. This is usually best assured when the child remains in the arms or upon the lap of the mother; but this, again, makes it difficult to secure the complete symmetry of posture which is absolutely necessary to accurate comparisons. It is better, therefore, to have the child lying on his back upon the bed when percussing the anterior thoracic wall and to percuss the back with the child in a sitting position or while lying on his abdomen. If the child is

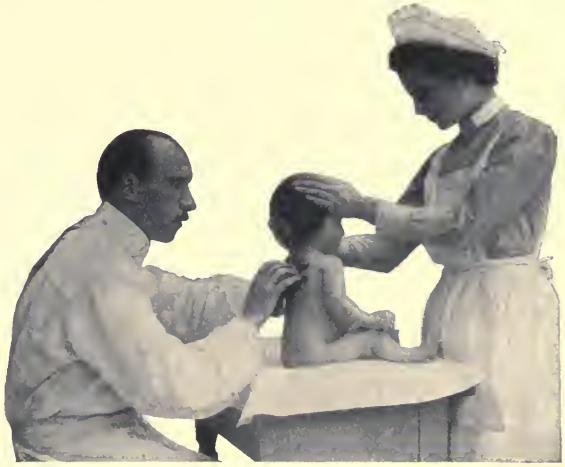


Fig. 14.—Percussion of chest in sitting position.

sitting it will be necessary to straighten the spine by gently pulling upward on the head, as is shown in Fig. 14, because the liver and diaphragm are pressed upward and to the right by the kyphotic posture resulting from the sinking down of the weak spine, and may thus cause dulness. In the examination with the child prone (Fig. 15) which is especially applicable to young infants, the breast of the child with adducted and flexed arms, rests on the hands of the nurse; then the back is stretched out and the examination may be made very comfortably. The percussion of the infantile thorax must be done very gently, finger to finger, on account of the great flexibility of the thoracic walls. When dulness or increased sense of resistance is encountered by this gentle percussion, the force of the stroke may be increased in order to arrive at a conclusion as to the intensity of the dulness. In the infant, small pneumonic foci either do not change the percussion sounds at all or may even impart a tympanic quality to it on account of the surrounding lung tissue. For this reason, any marked dulness which remains with even deeper percussion should arouse a suspicion,

at least, of exudate. In percussing children who are screaming, two things are to be noted; first, a loud "cracked pot" resonance, which often sounds like the clink of coins is frequently heard over areas of normal lung tissue and especially over the anterior of the thorax. Secondly, the increased tension of the intercostal muscles during crying causes a reduction of the sounds and increases the sense of resistance, both of which disappear in the phase of inspiration. This fact makes it possible to distinguish between the dulness due to the increased tension and the dulness due to actual diseased conditions of the lung. In thoracic exudation the percussion sounds of the normal side also frequently show changes.

In the auscultation of the lungs in the young infant the stethoscope has always given the most reliable information. In direct auscultation the

Fig. 15.—Percussion of back, infant lying face down supported by hands of nurse.

child may very properly be held in the position shown in Fig. 16, which gives the physician great freedom of motion.

In auscultation the crying of the child does not usually disturb the examiner as it does in percussion. In fact the cries rather give enlarged opportunity by bringing out indistinct sounds which can be heard clearly only at the height of the inspiration. It may be necessary, therefore, to excite crying at times. The breath sounds of the young child are much louder and more distinct than in older children or in adults, the so-called "puerile" breathing; even normal expiration is distinctly audible. The auscultation phenomena in the infant are not essentially different from those of the adult, aside from the fact that a certain amount of practice is necessary in order to distinguish the normal breath sounds from the transmitted sounds of the upper respiratory passages by which they are accompanied and sometimes concealed.

D'Espine's sign should be looked for while examining the thorax. When present it is indicative of enlarged bronchial lymph glands. Finally, it is necessary only to mention that at this age bronchophony is often clearer, and appears earlier than bronchial breathing in pneumonia.

Exploratory thoracentesis should be performed wherever flatness exists. The point of selection when the flatness is general should be in the posterior axillary line at the fifth interspace on the right and at the sixth interspace on the left. If, however, the flatness is more localized, the point of introduction of the needle should be at that of greatest flatness.

A needle sufficiently large so as not to become blocked by particles of pus should be used, preferably one of about one millimeter in diameter and it should be introduced from one to two centimeters. The procedure should be performed under aseptic conditions. In order that the pus should not be missed in the path of the puncture, the piston of the syringe should be slightly withdrawn, thereby creating a little suction immediately after the needle is introduced beyond the skin layer. The needle should pass between the ribs nearer to the upper than the lower border, so as to avoid the intercostal artery. The child should be in a sitting position and held firmly; the hand on the affected side should be brought over the opposite shoulder.

FIG. 16.—Direct auscultation.

The Abdomen.—In the infant the condition of the abdominal wall offers important points in diagnosis. Abnormal laxity of the walls always indicates that the diseased condition has gone on for a considerable length of time or that the attack is serious. If, at the same time, there be extreme emaciation, it is possible to see the peristaltic movements of the intestine through the abdominal wall.

The peristaltic waves of the stomach in the instance of pyloric obstruction are readily seen and are more pronounced if much loss of weight has occurred.

The size of the liver and spleen can be determined definitely only by palpation, while percussion gives uncertain results. In the infant the liver is relatively larger than in the adult. Its anterior border extends further downward and under normal conditions may even be felt distinctly below the costal margin.

It is not true that every spleen that is easily palpated is enlarged, since it may have a normally greater mobility.

The kidneys are palpable from above only when the abdominal wall is very thin and lax. More often it is possible to locate and feel the lower pole from the rectum, as they are extremely movable. This condition can hardly be said to have any pathological significance.

It is often quite difficult to demonstrate any localized tenderness to pressure in the abdomen of the child during the first year. The close observation of the facial expression during palpation, and of other reactions, as for instance, the position of the legs, the gait, the maintenance of a certain posture upon one side or upon the abdomen, are more important

than the indefinite and unreliable subjective symptoms of these small patients. Otherwise the topographical conclusions which may be drawn from the determination of a circumscribed area of pain are the same as in later life. The same methods are used as in adults for determining the position and size of the stomach or of a certain portion of the intestines. The technic of the use of the stomach tube is much easier. The method is described under lavage of the stomach on page 113.

The methods of examination of the gastric contents and gastric motility are the same as in the adult. The size of the catheter should be 14 to 18 French scale.

Abdominal paracenteses is done, diagnostically only to secure the fluid for bacteriologic or cytodiagnostic examination. The point of selection for the introduction of the needle is midway between the pubes and the umbilicus. The procedure is done under aseptic precautions. The needle

FIG. 17.—Method of collecting urine from a male infant. Test-tube fastened over penis by means of adhesive plaster.

should be about one millimeter in diameter. The syringe should be tested before using.

Catheterization of the urinary bladder is possible in the infant, but requires some practice. In boys it is done with a metal or elastic catheter, the caliber of which should correspond with that of the urinary meatus, and in girls it is more easily done with a metal catheter of about the thickness of knitting-needle. When necessary, a cystoscope of the smallest calibre may be introduced under anæsthesia in girls at the end of the first year. A paralysis of the sphincter, however, will remain for several days, so that this method of examination should be used in cases of extremity alone. Catheterization of the ureters is technically impossible during the first year.

When urine is to be collected for examination, the danger of bladder infection by the use of the instrument, even under proper precautions may be easily avoided by employing instead a strong test-tube fastened over the penis with adhesive plaster, as is shown in Fig. 17. In girls, a small Erlenmeyer flask (Fig. 18) may be placed over the labia which have been separated and carefully cleansed.

These methods of collecting urine suffice for the ordinary routine exam-

ination, but where it becomes necessary to determine the existence of a pyelitis or for the culturing and examination of the urine for bacteria, catheterized specimens are essential except possibly in the male patient, with no phimosis and an easily cleansed glans and the urine voided under direction. The first several streams of urine should not be collected, as they may be contaminated with washings from the urethra. When properly done the possibility of infection from catheterization is practically nil.

Because of the relatively high position of the bladder in the infant catheterization may become necessary at times to differentiate between a distended bladder and ascites or a new growth.

Rectal examination should be made whenever an intestinal obstruction, intussusception or an obscure abdominal condition may exist. The examination is best made with the patient lying on his back, the oiled finger being inserted into the rectum.

The Nervous System.—In order to test the passive motion and the tendon reflexes, it may be necessary to distract the attention of the child

FIG. 18.—Method of collecting urine from female infant. Small Erlenmeyer flask or large test-tube fastened over vulva.

and thus diminish the increased muscle tone, since voluntary tension of the musculature in nervous children often makes it impossible to bring out these reflexes. In the infant this is most easily done if the child is examined while taking his food. The absence of the patellar reflex when these precautions are taken, is always pathologic. Ankle clonus is common during the first year, especially in feverish or excited children, without having any diagnostic significance.

Because of the frequency and the clinical importance of spasmophilia in infancy, attention must be called to its symptoms and especially to the facial and peroneal phenomena. Special discussion of these is to be found in the section on nervous diseases.

Of the various tests for the quality of sensation, only that for the pain sense can be applied in children who have not learned to talk, and even this test must be done very carefully because the infant is often so alarmed by his first experience that he will cry out to non-painful stimulation, and often at the mere approach of the examining hand or when he is but gently touched. The final findings must always be controlled by comparison with normal parts.

Electrical tests are applied as in the older person. Despite the smallness of the child the stimulating electrode should be of the same size as that used in the adult (a Stintzing normal electrode one cm. square, see Diseases of the Nervous System) so that the results of the reactions at all ages may be directly compared. A plate, fifty centimeters square, may be used for the indifferent electrode which should be placed over the breast or abdomen. The technic of the examination requires not only a certain amount of practice but also close attention, if voluntary muscular contractions, independent of the electric stimulus, are not to lead the observer into error.

Lumbar puncture is of extraordinary diagnostic value in children as it is in later life, but it is done much more frequently on the infant because, on the one hand, of its simplicity of technic, and on the other hand, because the greater elasticity of the cranial wall entirely relieves it of danger. The most satisfactory instrument is a trocar of about the size of a knitting-needle. Only in case of necessity should a heavy hypodermic needle be used, because in the use of the trocar all danger of injury is avoided after the stylette has been withdrawn. Many prefer needles to trocars. The size of the needle depends upon the size of the patient.

Aspiration of the fluid by means of a syringe is frequently not only useless but dangerous and, therefore, should never be attempted.

The pressure is measured either by means of a long tube of small calibre, according to Quincke, which is attached to the trocar by means of rubber tubing; or by the method of Pfaundler, with a manometer filled with mercury or salt solution. By the first method we actually measure the pressure after a certain amount of the fluid has been withdrawn; and, as a result, lower figures are obtained than by the second means—the result of which can be calibrated only by comparison with those obtained with similar apparatus. It is to be further noted that the pressure is naturally lower when the patient is lying down than when he is sitting up. Sahli gives from 69 to 100 or 150 mm. water pressure (=5 to 7 or 11 mm. mercury), measured, according to Quincke's method, with the patient lying down; while Pfaundler, with the iris mercury manometer, has found 20 mm. in the infant and 25 mm. in children of from two to twelve years, in an upright position. In comparing Pfaundler's figures with those taken by the hydrostatic method, values of from 12 to 19 mm. mercury are obtained in children lying down. It is superfluous to say that the mensuration, as well as the puncture itself, should be done under absolute asepsis.

Whenever tumors of the brain are suspected the lumbar puncture should be carefully performed, with the patient in the prone position, and preferably with the head lower than the feet, so as to avoid the danger of a possible damage to the vital centres of the medulla from pressure from above. If after the spinal canal has been reached the fluid does not flow through the aspirating needle the position of the patient should be gradually changed to approach the sitting posture or to such an angle that will allow the fluid to flow through the needle.

In making the puncture with the child lying on his side, which position is to be greatly preferred because the child may be held more firmly,

the back is arched as much as possible by bending the head forward on to the breast and flexing the thighs upon the abdomen, as is shown in Fig. 19. The point of entry is then marked by connecting the iliac crests by a line and by locating with the under finger of the left hand, the inner-vertebral space lying nearest this line. The level so located will usually be the fourth or more surely the third lumbar space. In young children it is not well to go above this point, on account of the fact that the spinal cord extends lower than in adults and may therefore be injured by higher puncture. The entry is best made in the median line and the needle should not deviate

Fig. 19.—Position for lumbar puncture.

from the middle plane, but should follow the direction of the spinous process inclined slightly upward. A slight decrease of resistance may be felt the moment the needle enters the canal, 2-4 cm. ($\frac{4}{5}$-$1\frac{3}{5}$ inches) beneath the skin. At this point the trocar should not be pushed any further on account of injuring the large venous plexes on the anterior wall of the canal. Even though the hemorrhage caused by such an injury is not at all dangerous, the admixture of blood with the spinal fluid is very disturbing and may even make the specimen entirely useless for examination. The cerebrospinal fluid begins to drop from the trocar immediately after the stylette is withdrawn, if the puncture has been properly performed. If the pressure is increased it may flow out in a steady stream. If the opening of the trocar

is blocked by a root of the cauda equina a slight movement of the needle will often cause the flow to begin. If no outflow appears, in spite of the fact that the needle has been properly introduced (a dry puncture) it may be due to the abnormal viscosity of the highly fibrinous cerebrospinal fluid, which cannot pass the fine calibre of the needle. In such an event the injection of a few cubic centimeters of sterilized physiologic salt solution will often dilute the fluid sufficiently to permit enough to flow off for purposes of examination. Great caution should be exercised so as not to produce additional pressure when there is already an increased intercranial pressure.

A few cubic centimeters (5-10) of the fluid will suffice for examination. In cases where the pressure is increased larger quantities may, of course, be removed, continuing so long as there is a free flow. After the needle has been withdrawn, the puncture wound is covered with sterilized gauze fastened with adhesive. The method of examination of the fluid and the conclusions to be drawn from the results of examination are given in a special chapter.

Cranial puncture, a diagnostic measure which in the adult is rapidly gaining importance, may be done all the more easily in the infant because it is unnecessary to trephine. It is usually possible to puncture the thin cranial wall, or rather the membranous fontanelle, very readily with the trocar. The puncture is made 1-2 cm. (0.4-0.8 inch) to one side of the sagittal suture at the top of the head or slightly anterior to it and preferably in the area of the fontanelle. The trocar is inserted perpendicularly to the tangential planes for a short distance only and the stylette is withdrawn to note whether fluid is present at this level. Many prefer the use of a needle to that of a trocar.

Fluid at this point indicates an external hydrocephalus. If there is no fluid the needle is pushed in a few centimeters further in the direction of the lateral ventricles. In older children with a completely ossified cranium, it is necessary to trephine. The removal of the hair, which is necessary before preparing the field of operation, may be done much more easily in young children by using a depilatory, applied to the skin for a few minutes in the form of a freshly prepared paste, than by shaving the scalp.

Stools.—The physician usually sees the bowel movements of infants upon the diaper and often they are not very fresh. It is necessary to remember that, on account of the drying, thin stools may appear more solid than they were when fresh and that the green color so frequently seen may be reasonably due to the oxidizing influence of the air, and, therefore, is of no clinical significance.

Sputum.—On occasion, and especially where there is an open tuberculous lesion, the examination of the sputum may be of great clinical interest even in children of early years who always swallow their sputum. In these cases it may be secured for examination or for culture, by passing a tongue-blade far back in the throat and catching the sputum thrown up by the coughing on the blade or on an applicator covered with cotton. This method is much more simple and more successful than the difficult search in the feces for tubercle bacilli swallowed with the sputum.

Blood.—The blood of young children, to be used for microscopic examination is preferably taken from a toe, usually the great toe, or from the ear rather than the finger-tip. When larger quantities are required (2-5 cc.) for serum diagnosis, as for instance in the Wassermann reaction, a superficial vein may be punctured. For this purpose one of the superficial veins of the head is better than the veins of the arm. A cupping glass may be used. For this purpose, one or two long incisions (1-1½ cm.) are made over the back and a cupping glass placed over them is permitted to draw out the required amount of blood.

Sinus Puncture.—The puncture of the longitudinal sinus for the purpose of obtaining blood and for the injection of therapeutic fluids has proved very useful. The technic is quite simple and free from danger. A Luer syringe with an 18 or 20 gauge needle, about 1¼ inches in length is used. By a series of measurements it has been determined that the sinus lies 1/16 to ⅛ inch beneath the surface of the scalp and is about 5/32 of an inch in calibre at the posterior angle of the fontanelle. For this reason it is convenient to have a guard firmly fixed about ¼ inch above the point of the needle to prevent deeper penetration. Reports tend to show that even though the sinus be transfixed no special injury results.

Roentgen Rays.—The diagnostic use of the Roentgen rays is hampered by the fact that many parts of the skeleton which are not completely ossified permit the passage of the rays fully as well as the surrounding soft parts. The comparison, therefore, with radiograms of skeletons of normal infants in the same stage of ossification is always unreliable in the matter of pathologic findings. In the recognition of alterations in the internal organs, for instance of enlarged bronchial lymph nodes, pneumonia areas, pleuritic exudates, enlargement of the heart, etc., examination with the Roentgen rays has gained in importance in pediatrics as well as in internal medicine. The Roentgen rays are of great value in the determination of stenoses and atresia, as for example, those of the œsophagus, stomach and intestines, also for obstructions due to intussusception, etc. With good apparatus and careful technic, changes which cannot be determined by other means may be demonstrated in the same degree as in the adult.

IV. GENERAL PATHOGENESIS

MORTALITY AND MORBIDITY

REVISED BY

B. RAYMOND HOOBLER, M. D.,

Professor of Pediatrics, Detroit College of Medicine and Surgery, Detroit.

THE mortality of infants and children is of great significance because it exerts a pronounced influence upon the growth and quality of the population upon which depends the expansion of a Nation. In fact the infant mortality rates should be a matter of national concern.

Table I. shows in outline form the relation between the birth and death-rate, also the relative high mortality of the first year.

TABLE I.

	No. of Children Born Alive Per 1000 Inhabitants	No. of Deaths Per 1000 Inhabitants	Deaths in First Year Per 1000 Children Born Alive	No. of Children Born Alive Exceeds No. of Deaths Per 1000 Inhabitants
France 1906	19.6	19.3	143	0.3
Belgium.....1908	24.9	16.5	147	8.4
Sweden 1908	25.6	13.7	85	11.9
England (Wales)..1909	25.6	14.5	109	11.1
Norway.....1908	26.2	13.5	76	12.6
Switzerland.....1908	26.3	16.6	108	9.7
Denmark.....1908	28.3	13.3	123	15.0
Holland.....1908	29.1	13.7	99	15.5
German Empire...1909	31.0	17.1	170	13.8
Japan.....1908	32.3	20.0	158	12.3
Italy.....1908	32.4	21.4	148	11.0
Austria.....1907	33.6	22.4	209	11.2
Hungaria.....1909	37.0	25.1	212	11.9
Russia 1901	48.0	29.5	272	18.5

The comparison of the four columns shows that the number by which the birth-rate exceeds the death-rate per thousand inhabitants is quite variable ranging between 0.3 in France to 18.5 in Russia. Germany has an excess birth-rate of 13.8 which is exceeded not only by Russia, but also by Holland and Denmark. Most of the other civilized countries have the same number of excess births as Germany.

In Table I. it can readily be seen that a growth in population can take place in two ways. First, by a birth-rate so large that even after deducting a large death-rate we have an excess; second, by a lowered mortality rate such that even a small birth-rate would give an increase in the population. Russia is an example of the first instance; the Northern European countries of the second.

Each people pursue their own course in this matter depending upon their many circumstances. The second way seems at first glance to be the most rational, but the experience of the civilized people seems to show that not only is it very difficult to reduce the death-rate, but that a decrease in the number of births due to prevention of conception is, in itself, a sign

TABLE II. (GERMAN EMPIRE.)

To Each 1000 Inhabitants.

Year	Were Born Alive	Deaths Including Still-Births	Excess No. of Births Over Deaths
1901	35.7	21.8	15.1
1902	35.1	20.6	15.6
1903	33.8	21.1	13.9
1904	34.0	20.7	14.5
1905	33.0	20.8	13.2
1906	33.1	19.2	14.9
1907	32.3	19.9	14.2
1908	32.1	19.0	14.0
1909	31.0	18.1	13.9
1910	29.8	17.1	13.6
1911	28.6	18.2	11.3
1912	28.3	16.4	12.7
1913	27.5	15.8	12.4

of degeneration. It seems, therefore, that when we try to understand what would be rational sex life in the peoples of all classes in the various nations, there is no safe means by which to stop the growth of a people. Unfortunately even in Germany, which up to the beginning of the World War had a greater birth excess than most civilized countries, there has been a definite tendency to a decrease in the birth excess.

Table II. shows not only what was pointed out in Table I., namely the

TABLE III.

Births Not Including Still-Births	Deaths Not Including Still-Births	Illegitimate Births Including Still-Births	Deaths in 1st Year Not Including Still-Births			Deaths Per 100 Born Alive			Yrs.
			Legitimate	Illegit.	Totals	Average	Legitimate	Illegit.	
2,032,313	1,174,489	179,683	361,745	58,478	420,223	20.7	19.4	33.9	1901
2,022,477	1,112,202	177,060	324,592	50,044	374,636	18.5	17.5	29.4	1906
1,924,778	1,045,665	179,584	267,171	44,291	311,462	16.2	15.2	25.7	1910
1,838,750	1,004,950	183,977	235,272	41,924	277,196	15.1	13.5	21.8	1913

great influence of infant mortality on the general mortality rates, but teaches us above all, the fact that since about the year 1900, the number of births have decreased more rapidly than the number of deaths; also, that if there is further decrease in frequency of births, Germany will soon present a condition like that of France.

Table III. shows for the German Empire the exact *number of those living*, those born and those that die in a population of over sixty million.

Also, the unfavorable position of the illegitimate child, the cause of which will be elucidated further on.

As has already been mentioned, the danger to life is not the same at all times during childhood but by far the greatest in the first year and proportionately great, during the second year. From then on the mortality curve sinks rapidly.

TABLE IV.

Deaths according to age per 1000 deaths (in German Empire).

Between ages of	In the Year 1911		In the Year 1913	
0–1 yr.	317.1		275.1	
1–2 yr.	48.8		45.1	
2–3 yr.	17.0		16.0	
3–4 yr.	10.7	84.5	10.4	79.3
4–5 yr.	7.9		7.8	
5–6 yr.	6.0		6.1	
6–7 yr.	4.9		5.1	
7–8 yr.	4.0	21.6	4.2	22.1
8–9 yr.	3.4		3.5	
9–10 yr.	3.2		3.0	
10–11 yrs.	2.8		2.9	
11–12 yrs.	2.8		2.7	
12–13 yrs.	2.4	13.9	2.7	14.0
13–14 yrs.	2.7		2.7	
14–15 yrs.	3.0		2.9	

In Table IV. the author has purposely contrasted the years 1911 and 1913, one a very warm, the other a very cool year. It can be seen by noticing

TABLE V.

Number of deaths per 1000 at various times according to mortality tables for years 1901 to 1908.

Still births	33.47	Previous mo.	165.53	Previous mo.	219.39
1st. mo.	59.07	5th. mo.	18.39	9th. mo.	8.53
2nd. mo.	26.77	6th. mo.	13.73	10th. mo.	7.41
3rd. mo.	25.55	7th. mo.	12.31	11th. mo.	5.79
4th. mo.	20.67	8th. mo.	9.43	12th. mo.	5.45
Total	165.53	Total	219.39	Total	246.57

the five year periods as well as the single years, that a difference exists mainly in the first and to a lesser extent in the second year.

The infant mortality is not uniform during the first year but is the greatest during the first part of infancy. This fact is shown by a large number of statistics, and in Table V. by the Magdeburg Statistics.

During 1918 there were 193,855 deaths of infants under one year of age in the registration area of the United States. They were distributed through the year as follows:

Death occurring at	*Number of Deaths.*	*Percentage of deaths.*
Less than 1 day	29,106	15
1st day	9,554	4.9
2nd day	6,829	3.6
3rd to 6th day	12,645	6.5
Less than 1 week	58,134	30
1st week	11,750	6
2nd week	8,292	4.25
3 weeks but less than 1 month	6,572	3.39
Less than one month	84,748	43.64
1st month	16,278	8.39
2nd month	13,238	6.8
3rd to 5th month	31,503	16.25
6th to 8th month	26,111	13.66
9th to 11th month	21,977	11.26

Within the first month, which is the most critical one as far as the life of an infant is concerned, we note that the first day and week are of greatest importance. For example, according to carefully collected statistics in Berlin, there are two and one-half times as many deaths in the first week, as in the second week of life and one-half as many deaths during the last half of the first month, as during the first two weeks.

Two other definite factors are to be noted in the statistics given thus far, namely the very much higher mortality of the illegitimate as compared to the legitimate child in the first year (Tables III and IV) and secondly, the rise in infant mortality in the summer months (Table VIII).

Many of the diverse factors influencing the expectation of life in infants, which a physician meets in daily routine but has not learned how to evaluate, can only be expressed truthfully if the statistician is allowed absolute liberty in choice of material. This is possible, however, in only more or less limited way and for only a short space of time.

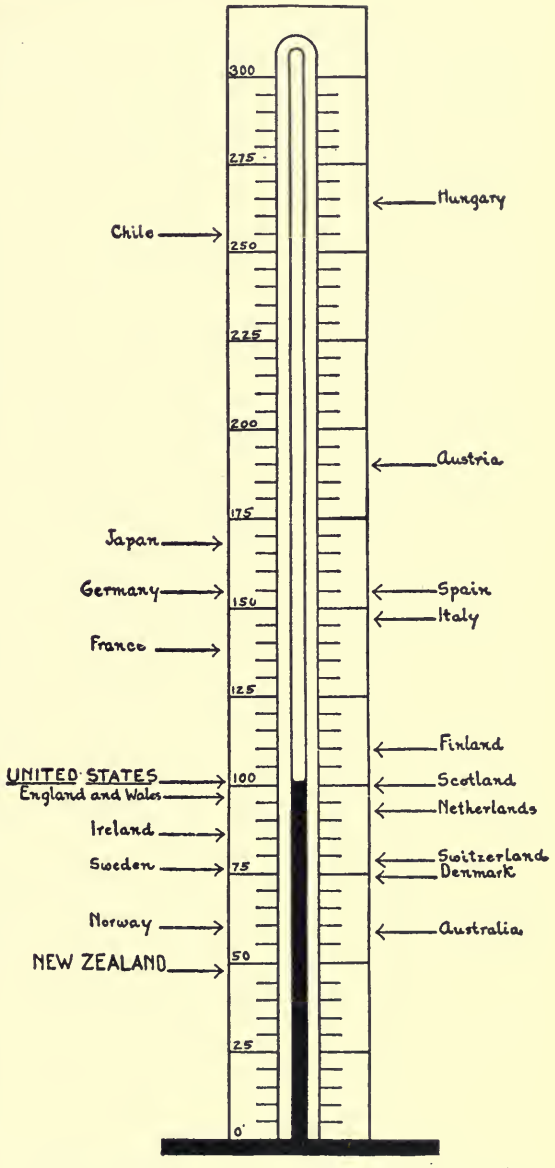


FIG. 20.—Deaths under 1 year of age per 1000 births. Rates are for latest available years up to 1918 as compiled by the Children's Bureau, U. S. Dept. of Labor. Within the first year after birth the U. S. loses 1 in 10 of all babies born. It ranks 11th among the principal countries of the world. New Zealand loses fewer babies than any other country.

The high mortality of infants is, according to Malthus, a natural protection against an excessive increase in population so that individuals may be enabled to obtain the necessities for life. Later under the influence of the Darwinian theories

this was explained on the basis of natural selection. Thus only the strong survive, the weaker, being worthless to the race, are cast out. Many statistically established facts, as for example, the infant mortality among the different classes of people (which is firmly established and noticeable in the German Empire) have been quoted as proof of the Darwinian Theory by its supporters. As also, the higher mortality of the illegitimates. How-

TABLE VI.

Infant Mortality in Berlin 1900 to 1902.

IN FAMILIES OF	Per cent.
1. Officers, officials and professional men	11
2. Business men	15
3. Skilled artisans	16
4. Unskilled laborers	18

ever, any one who will look at the facts as a true physician and scientist, will soon see that the explanation is not to be made on biological grounds but on the basis of social status. Much more important is the effect of the social conditions under which the child is born and those under which he develops, as for example, the financial condition of the parents. According to statistics, if we choose the calling of the parents or the size of dwelling

TABLE VII.

Nutrition and Infant Mortality in Berlin. Deaths per 1000 infants.

Month of life	Breast-fed			Artificially-fed		
	1885–86	1895–96	1906	1885–86	1895–96	1906
1	22.4	19.6	22.4	142.0	111.9	58.1
2	9.	7.3	7.9	82.7	58.7	31.3
3	6.8	4.3	4.3	72.2	49.7	27.3
4	6.4	3.6	2.4	61.8	46.6	22.1
5	5.3	2.6	1.7	57.1	37.0	18.5
6	4.9	2.5	2.2	50.7	31.0	16.1
7	4.7	2.5	1.4	46.5	27.7	14.1
8	4.5	2.3	1.8	40.8	24.1	12.2
9	5.3	2.0	2.1	33.3	21.3	10.2
10	5.4	3.8	1.5	29.5	19.1	9.2
11	6.3	3.1	1.3	24.9	16.7	8.0
12	——	3.6	1.5	——	14.6	8.0
Average per m.o.	8.4	6.0	6.3	54.1	35.8	23.6

as an indication of social status, we can see the increased danger of life to infants born in unfavorable surroundings.

A fundamental law, substantiated not only in every day medical practice but also by a large number of statistics, is the high mortality of the artificially-fed as compared to the low mortality of the breast-fed infant. This fact does not coincide with the idea of natural selection. If it were true that children do well *only* when nursed by their own mothers we could say that the nursing ability of the mother would give us prognostic knowledge as to health of the child. But we know that children do well on breast-milk

of other women beside their mother, and also that observation has firmly established the fact that children that are ill and on artificial food recover most rapidly on breast-milk. Thus even though a very small per cent. of breast-fed children may not do well on the breast, we should notice more particularly the problem of nutrition rather than that of hereditary defects, thus keeping down the infant mortality and assuring good health to most infants.

The infant mortality rate in many of the cities of the United States has been reduced much below the average, due to the intensive Infant Welfare work done through many agencies and especially through breast feeding propaganda. The American Child Hygiene Association is the leading National agency working for the reduction of Infant Mortality.

The figures for the ten largest American cities taken from publication of American Child Hygiene Association is given below.

	1916	1917	1918	1919
New York	93	89	92	82
Philadelphia	105	108	124	90
Detroit	112	103	101	97
Cleveland	109	109	98	91
St. Louis	89	85	93	75
Boston	105	99	115	97
Baltimore	122	118	149	97
Pittsburgh	115	120	139	115
Los Angeles	69	71	77	67
San Francisco	67	63	67	65

The following table shows the lowest and highest infant mortality rates in cities of various sizes in the United States as published by the American Child Hygiene Association, 1920.

CITIES WITH HIGHEST AND LOWEST RATES

Population over 250,000

Lowest		Highest	
Seattle, Wash.	54	Pittsburgh, Pa.	115
Minneapolis, Minn.	61	Buffalo, N. Y.	107
San Francisco	65	Kansas City, Mo.	103

Population 100,000–250,000

Lowest		Highest	
Houston, Tex.	61	New Bedford	124
Oakland, Calif.	62	Camden, N. J.	121
Cambridge, Mass.	64	Nashville	116

Population 50,000–100,000

Lowest		Highest	
Berkeley, Calif.	44	El Paso, Tex.	245
Fort Wayne, Ind.	51	Knoxville, Tenn.	135
Topeka, Kan.	59	Racine, Wis.	123

Population under 50,000

Lowest		Highest	
Brookline, Mass.	40	Burlington, Vt.	150
Marinette, Wis.	45	Paducah, Ky.	146
Aberdeen, Wash.	45	Hannibal, Mo.	145

Aside from the evidence given by statistics that the breast-fed infant has a lower mortality and morbidity rate, statistics can be adduced to show the increased mortality as resulting from variations of temperature. In Table VIII., the years 1911 and 1913 were chosen because the summer months in the latter were not excessively hot while in the former there was persistent high temperatures during the months of July, August and September.

TABLE VIII.

Deaths in infants during first year of life.

	1911	1913		1911	1913
January	20.99	21.50	July	49.38	27.41
February	22.60	22.83	August	62.11	27.36
March	21.04	24.84	September	45.04	29.15
April	25.04	20.99	October	23.36	26.01
May	25.37	23.33	November	18.28	25.08
June	27.08	24.52	December	21.71	17.60

The editors add here a table by Graham consisting of statistics from the city of Philadelphia showing again the increased mortality in infants on artificial foods. This table further brings out the increased mortality coincident with the summer heat.

	Breast-fed	Artificially-fed	Feeding not known
January	8	21	9
February	9	14	18
March	13	15	21
April	9	13	17
May	10	16	27
June	7	4	50
July	8	155	138
August	28	148	161
September	22	104	71
October	18	56	73
November	11	36	36
December	10	24	34
	153	606	655

Of the 759 deaths with known type of feeding, 20 per cent. were breast-fed and 80 per cent. were artificially-fed. This is a strong appeal for maternal nursing. Maternal nursing is considerably reduced because of the very high maternal mortality rate prevailing in the United States. According to the Children's Bureau the United States lost over 23,000 women in 1918 from Childbirth. We have a higher maternal mortality rate than any other of the principal countries.

In the United States the deaths from diarrhœa and enteritis under 2 years of age increase greatly during the summer months. The total deaths (47,753) from these causes in 1918 is divided among the months as follows:

January	1775	May	2712	September	8128
February	1712	June	4027	October	4458
March	2040	July	6811	November	2394
April	2192	August	9822	December	1682

Broncho-pneumonia which occurs more often in children has a much higher death-rate in the colder months. The 54,697 deaths from broncho-pneumonia in 1918 were divided among the various months as follows:

January 5256	May 3005	September 1853
February 4901	June 1507	October 13078*
March 5515	July 1309	November 6208
April.....5194	August 988	December 5883

* During influenza epidemic.

The following circumstances acting together influence the mortality rate of infants namely, financial and social status of the parents, the character of their dwelling place, the kind of nourishment, and also the time of the year, but, the most striking factor is the kind of nourishment. H. Neuman's statistics of Berlin are of interest.

TABLE IX.

Deaths per 100 children according to housing conditions.

	1-2 rooms and kitchen Percentage	3 rooms and kitchen Percentage	4 rooms and kitchen Percentage
Total	17.7	12.8	7.3
Bottle-fed	12.8	10.9	4.7
Breast-fed	4.9	2.6	2.6

Besides the factors mentioned above, others worthy of mention but closely related are, the unfavorable prognosis for the illegitimate child because of the lack of breast-milk and a mother's care and the influence of a large or small number of children in individual families.

Statistics covering illegitimacy in the United States are meagre. The latest authoritative statement is from the Eighth Annual Report of the Children's Bureau 1920. Miss Lothrop states that, "Each year in the United States at least 32,000 white children are born out of wedlock." This is proportionately fewer than in most foreign countries. The death-rate of illegitimate birth, according to the studies made by the bureau, is three times as high as that of other children.

The above statistics cannot be discussed fully, except that a lesson is given in the realm of Pediatrics which is familiar to all physicians who understand social conditions, namely, that families in which the children are breast-fed and in which there is a pause of one and one-half to two years between children have a minimum infant mortality and that the later children do not labor under any disadvantage—(especially when we have large families of eight, ten or more).

In order to understand the above mentioned facts a knowledge of the causes of death and the proportion of each is necessary. However, it is with reluctance that one presents statistics of the causes of death, even though there are a great number available, both of the civilized countries and great urban communities The value of statistics is depreciated because of poor raw material on which they are based, giving conclusions at variance with the facts noticed by the physician in every day medical

practice. All these statistics agree, however, that gastro-intestinal disease causes one-third or more of the deaths in the first year and that during this same time the acute infections and inflammation of the respiratory tract cause but a small part of the total number of deaths. This great number of gastro-intestinal infections tells only half the truth, because there are many indefinite diagnoses made, in which the artificial feeding was the principal fault. Thus the diagnosis of congenital weakness, debility, etc., have been shown many times to be clinically only the manifestation of artificial feeding. Even congenital lues which is given as a cause of death, very often means that death was due to artificial feeding because many luetic infants gain well on the breast. Again the cause of death may be ascribed to pneumonia, furunculosis or heart lesions, all of which are but terminal affairs following severe nutritional disturbances.

Close examination of the high death-rate in summer, with due consideration of the mode of feeding, reveals the fact that only the partially or entirely artificially-fed infants contribute to the increased death-rate.

The possible explanations of this fact are given in detail in the Chapter on The Disturbances of Nutrition in Infants.

All the injuries of artificial feeding react most severely upon the children of the poorer classes. H. Neumann says, as a result of his experiences in Berlin, "The results of natural feeding are very good in the various strata of society. In artificial feeding the expectation of life is reduced in proportion to the level of the social stratum." Special proof of this general rule is seen in the high mortality of illegitimate infants.

The death-rate decreases markedly from the second year and this is probably due to the fact that after the second year disturbances of nutrition, as a cause of death, become more and more uncommon. In their place, however, the acute exanthemata, diphtheria and tuberculosis appear more frequently.

Even though vital statistics give us exact information as to mortality in its many sided relationship to age, season, social welfare, methods of feeding, etc., it is impossible for obvious reasons to draw from them even an approximate picture of the morbidity.

The statistician obtains reliable figures for only one group of diseases—the acute infections of childhood which must be reported to the health authorities, and then, of course, only of those cases which are seen by a physician. In some classes of society and in some parts of the country, this is only a negligible number. All the statistics based upon the material of various dispensaries and clinics suffer from similar errors and, therefore, we forego citing any of them.

All these arduous compilations, among which the work of Escherich must, at least, be mentioned, have so far succeeded only in proving to every observing physician the fact that every period of childhood has its peculiar disease groups, which are notable for their frequency, rarity, or complete absence; or for the definite combinations they present of seemingly unconnected disorders and anomalies which indicate close pathogenetic relationships between them. It would not seem that the time has arrived

when a general pathology of the diseases of childhood may be written, since pediatrics is still a young science and must concern itself for a long time with the collection of data. But the attempt to examine the disease groups of the various stages of development more closely and to draw briefly such conclusions as appear, after careful study, to have a basis in fact, is justified. These, however, are very few if one does not wish to enter deeply into the etiology of the various diseases; a topic for which the reader is referred to the literature at large.

These factors are especially concerned in the startlingly high morbidity which is revealed by the enormous mortality of the first few days of life. They are: (1) *Congenital Malformations*, in so far, as they affect extra-uterine life; (2) *Birth Injuries*, and (3) *Congenital Debility*.

The first group is probably the least important, while birth injuries are frequently the cause of more or less serious illness, which often results in the death of the child. Omitting the danger of infection from the birth canal, hemorrhages from vital organs (the central nervous system and its coverings, the adrenals, etc.), either as a result of congestion or injury following the use of force in difficult labor, are apparently common to both of the first two groups.

Congenital debility is not confined to premature infants alone, although it is much more common among them than in others. Under congenital debility is included functionally retarded development of a degree which imperils the life or development from birth, even though it be given the same care, in the way of suitable temperature, breast feeding and freedom from infection, under which children normally develop. Space will not permit us to enter into the pathology of congenital debility. We can only call attention to the fact that its diagnosis must be confined to those cases in which an abnormally labile temperature, a reduction of the respiratory rhythm and often a marked disturbance of reaction to all physiologic stimuli can be shown. The early death of a new-born infant as a result of artificial feeding, or of infection and the like, cannot be laid to congenital debility, if we do not wish to make this diagnosis the "catch all" of diagnostic uncertainties and imperfect observations.

That the morbidity of infancy is governed by artificial or unnatural feeding cannot be repeated too often. The experience of physicians teaches this incontrovertibly, and not only that the more or less severe disturbances of nutrition or gastro-intestinal diseases occur almost exclusively in artificially-fed infants, but that this is true also of the development and the severity of the numerous parenteral infections of the skin, the respiratory system, the uro-genital tract, etc.

As examples, which might be multiplied without end, it suffices to mention furunculosis and numerous other purulent infections of the skin, lobular and hypostatic pneumonia, cystitis and pyelonephritis. The same relationship appears in the cachectic types. Spasmophilia, the exudative diathesis, rickets, etc., exhibit much more serious manifestations in the artificially-fed infant than in the breast-fed child.

The frailty and low resistance of the infant, as compared with older

children, have always been proper subjects of special attention. Emphasis should be put, however, upon the fact that the normal healthy child with suitable food and care, beginning with a sufficiently long period of breast feeding, does not exhibit nearly so great morbidity as one might be led to suppose by the high mortality statistics.

Why artificial feeding, even when the weight curve shows that it is conducted to good results, should increase the morbidity of the new-born and of older infants so markedly and should lower their immunity so distinctly has not yet been determined. Whether feeding with human milk produces a sort of passive immunity by the constant supply of antibodies from the mother's milk, or whether the artificial food prejudices the chemical integrity and consequently the functional resistance of the infant organism, are questions still under discussion, the solution of which, however, will not materially alter clinical methods.

The infant escapes a certain number of diseases, because even with the poorest of care he is not exposed to certain of the dangerous injuries of later life. Among these may be mentioned traumatic diseases, the results of exposure to inclement weather and, particularly, the lesser opportunity of contact with a variety of infections, so long as the child lies quietly in his bed. After the first year, in fact as soon as the child begins to creep and comes more frequently into contact with his surroundings, all this is changed.

Dirt and contact infections, in fact, reach their maximum in the second and third years. Of these, diphtheria, pertussis, contagious impetigo, aphthus stomatitis, various forms of angina, and tuberculosis, may be mentioned. Certainly it is not an accidental thing that most of these diseases have their primary localization or rather their port of entry in the mucous membranes of the mouth and upper air passages, to which the grimy hands and unclean toys or eatables passing from floor to mouth first bring their load of disease germs. Later another opportunity is freely given among the crowds of children in the kindergarten and the schools where they come into contact with one another in their play at a time when the prodromata of the acute contagious exanthemata may be present.

It will be seen that there are all-important external influences which give a peculiar stamp to the morbidity of the run-about-age and of the early school years. Undoubtedly the school age, when the so-called school diseases appear, broadens the circle of infections which threaten the child. At this period, whether we have to deal with such diseases as scoliosis, myopia and other visual disorders, with headache, anorexia, disturbed sleep and the like; or with such distinct neuroses as hysteria, migraine, neurasthenia, psychopathy, etc., it is always possible to find beneath the actual injury arising from school attendance, an existing and recognizable predisposition, or the influence of injuries traceable to home surroundings and training, which have played an important part.

As the child approaches puberty, the disease groups of both sexes come to resemble more and more those of the adult. Early puberty shows a slight increase of morbidity as compared with the low morbidity of late

boyhood and girlhood. Aside from those disturbances which stand in direct relationship to the development of the genital organs, an increasing frequency of tuberculous manifestations, of certain infections, of functional heart diseases and psychic anomalies, are especially to be noted. These years are characterized, as we have already seen, by a marked increase in the rapidity of growth in height and by the development of various organs and their systemic relations.

The relational study of the increased rate of growth and the greater morbidity of the years of development has suggested the theory that this rapid growth and the more rapid metabolism associated with it serve to increase the vulnerability of the tissues and organs to disease. The hypothesis is a very probable one and affords a possible explanation of many other observations; *e. g.*, to cite but one instance, the localization of rachitic changes at the epiphyses, the point of the entire bone of by far the greatest measure of growth (Kassowitz). Much is still wanting by way of acceptable proof of this hypothesis to permit its use even as a working basis for the study of the relational operation of these factors in the living mechanism.

V. GENERAL PROPHYLAXIS AND THERAPY

REVISED BY

ALBERT H. BYFIELD, M.D.,

Professor of Pediatrics, State University of Iowa, College of Medicine, Iowa City.

(*a*) GENERAL PROPHYLAXIS

THE protection of the child from disease should begin before birth. The influence the physician can exercise in the prevention of congenital disease is usually very slight, even in the families of the educated classes; and among the uneducated there is a general want of responsibility for their progeny. It must be admitted, however, that rarely does the present incomplete understanding of the laws of heredity permit prediction, in a given case or with any degree of certainty, of children sound in mind and body. Particular attention should be called to the fact that the marriage of close relatives does not, in itself, endanger their offspring, and that ill results are observed only in cases of converging hereditary taint (Feer). Thus the prenatal protection of the child practically begins only after conception, and then only in so far as instruction and circumstances permit the mother to take such measures to safeguard her own health as are necessary to the well-being of her expected child. This volume is not the place in which such teaching may be given in detail. Mention might be made here of the possible relationship between the nutrition of the parents and that of the offspring, such as occurs in the animals.

During delivery the child is in actual danger of injury by trauma and from infection. Further, it may be said that an injury during birth or from puerperal disease may so injure the mother that nursing becomes impossible and that child loses the mother's care. It is, therefore, desirable that infant welfare organizations consider the hygiene of maternity and especially of the puerperium, as well as the direct care of the infant.

It has been clearly shown in Chapter IV., that the greatest danger which threatens the life and health of the infant arises, directly or indirectly, from artificial feeding. Hence the most important prophylactic measure is a sufficiently long period of feeding with mother's milk.

The methods of breast feeding, even in cases where difficulties or dangers arise, are fully discussed in the second chapter. The spread of the propaganda for breast feeding by institutions for infant welfare will be fully described later.

The avoidance of infection is an important feature of prophylaxis even in children. It must begin in the new-born. Even though purulent infections of the umbilicus do not have the importance which was formerly assigned to them, tetanus of the new-born, which is always avoidable, undoubtedly has its origin in the umbilical wound. Ophthalmia neonatorum is equally preventable and it should therefore be made the duty of the

physician and of the midwife to use the Crede instillation in all cases even where gonorrheal disease of the mother can be absolutely excluded. The physician should be careful to see that this order is carried out.

The organisms of infection to which the infant is exposed find entry through the mucous membranes of the upper air passages and mouth. Among these infections we may mention rhinitis, tonsillitis, pharyngitis, bronchitis and influenza.

Generally speaking, all of these diseases run a more severe course in young infants, and especially in children artificially-fed or suffering with disturbances of nutrition. It is therefore necessary to guard such children against these infections in every possible way. This is measurably and readily possible because these diseases are favored or induced by climatic influences. Obviously, this method of contagion is not of major importance. Usually, these infections are carried to the child by the adults of the family. Therefore, the most effective measure would be to keep persons suffering with any of these diseases entirely away from the child. This is practical so far as other sick children of the family are concerned and should be achieved as thoroughly as the circumstances of the home will permit. Of course it is not always possible to replace adults to whom the care of the infant is entrusted. Nevertheless, the danger of the spread of contagion by adults may be definitely reduced by proper precautions prescribed by the physician. Since the infection in the above diseases is spread either by the hands of affected persons soiled with infectious material or by particles sprayed into the air, not only by coughing and sneezing, but even by speaking, laughing, etc., it may be avoided by the exercise of due care. This, of course, implies that infected material must be kept not only from the child's person, but also from all such objects as clothing, pacifiers, etc., with which the child comes in contact.

In normal children the predisposition to this group of diseases tends to diminish with increasing age; so that we may fairly say that children who are infected every time they come in contact with an adult suffering from any of these respiratory disorders have a pathologic predisposition or are so-called susceptible children. This class will be more fully discussed later.

Even with careful training the majority of children do not learn to use mouth-washes and gargles correctly before the fourth or fifth year. The most important prophylactic measure against infection is, therefore, to keep children away from any source of trouble. To carry out this measure successfully it will be found necessary to accustom the child, even when well, to refrain from unnecessary caresses and the like.

All this is equally true of the so-called diseases of childhood; the acute exanthemata, diphtheria and pertussis. These diseases, likewise are transferred only by close contact with those suffering with them or by contamination from their secretions. Since, with the exception of diphtheria, one attack usually confers immunity, it is readily seen that adults spread infection only as carriers and that the greatest danger to the child lies in contact with diseased children. Day nurseries, kindergartens and schools offer the most frequent opportunities for contagion. The spread of

measles and pertussis is increased by the fact that these diseases are contagious in their prodromal stages. Danger in diphtheria lies in the fact that in many cases there are no characteristic symptoms. It is the duty, therefore, of parents, a duty which the physician should impress upon them, to keep children at home when they are ill or even under suspicion of disease in order to protect their playmates and school fellows.[1]

If any such disease makes its appearance in a family in which all of the children are not immunized by previous attacks, every possible precaution, of course, must be taken by the complete isolation of the patient, and this isolation should extend to the objects used by him and should include adults who nurse him. Under such circumstances infection is carried much more readily by the adult because of the frequency of exposure, than it is if contact is only occasional and for a brief period. For this reason, the physician is less often a carrier than the mother. When the question arises of removing other children from the home to place them in the care of relatives, we must consider not only any obstacles which may exist to their effective care and isolation in a new abode, but also the possibility that they themselves are in the incubation period of the disease. It is always best to keep such children away from other families where there are children who might be endangered by exposure to disease. As it is practically impossible to guard children, in a large city, at least, from the acute exanthemata, it may be as well to resort to extreme prophylactic measures only for those whose health would be especially jeopardized as a reason of their age or because of latent tuberculous foci (or other influences tending to lower resistance).

A peculiar method of disease conveyance is exemplified in the so-called dirt infections which are most common during the second and third years. Children creeping about the floor and putting soiled toys and dirty hands in their mouths, may contract diphtheria, pertussis, impetigo, aphthous stomatitis, angina, and occasionally tuberculosis, in this way. The only means of combating this mode of infection is by training the child and, while the child is still too young for this, by the most scrupulous cleanliness of the room, toys, and utensils. This may be accomplished most successfully by the use of pen shown in Fig. 10.

The prevention of tuberculosis during childhood is largely a question of a sanitary home and the proper hygienic care of the child and of tuberculous adults with whom the child comes in contact. Since this can be but partially achieved in a large population and then only with considerable difficulty, the most reliable method of prophylaxis lies in the absolute isolation of the tuberculous member of the family. This is best accomplished by placing the infected individual in a sanatorium. If this is not possible and if the hygienic conditions in the home are not good, the danger of contagion is very great, as is evidenced by the high incidence of the infection in the children of the proletariat of the larger cities. The danger of tuberculosis for the child in the school is but slight. The experience of

[1] The problem of diphtheria carriers and the detection of them is more fully discussed under "Diphtheria" in the chapter on Infectious Diseases.

school physicians has shown that the number of children of school age with open tuberculosis is extremely small. The danger that the teacher may be the source of infection must be considered as much more likely. It should be remembered, furthermore, that the danger of infection comes not alone from members of the immediate family, but also from such other residents in the house as servants, governesses, tutors, lodgers, etc. The necessity of a more careful survey of the health control of these people than is usually made will be readily recognized. Parents have been known to actually take their children to visit friends or relatives suffering with advanced tuberculosis. It is equally evident that healthy children may be infected with syphilis by a luetic wet-nurse or any other servant, a danger to be safeguarded by careful medical examination of those employed.

Besides these prophylactic measures, the lessening of the predisposition to disease by improvement of the general constitutional resistance offers the largest possibility of success. As already shown, children are not alike in their susceptibility either to tuberculosis or to the ordinary diseases which arise from common "colds;" an evident tendency to disease may entirely disappear in some children during early childhood, whereas in others it remains almost unchanged even through the school age. These latter not infrequently have parents who themselves as children suffered with similar catarrhal infection. It would seem that this heightened susceptibility of children to these respiratory troubles can be traced back to some unusually severe or long drawn out acute infection which occurred early in the child's life, which left a *locus minoris resistentiæ* which endures for many years. That chronic infection may reside in the nasal accessory sinuses seems to have been established beyond question. In such cases it is necessary to try to avoid those conditions which will stir up these respiratory infections or cause new ones. The method of feeding, plays a very important part even beyond infancy. As overfeeding is a cause of great danger to the infant, so diets containing an excess of protein or fat tend, in children with a special predisposition to catarrh, to aggravate and to lengthen the duration of the attacks. Czerny has laid particular emphasis upon this factor, often under-estimated even by the physician, in his treatise on exudative diathesis. He notes that such children do best upon a diet largely vegetarian. [It should not be forgotten that a sufficient quantity of milk, preferably poor in fat must be included in order to supply the calcium phosphate and other salts necessary for growth.]

The aim with such children should be to develop good resistive powers by methods which need adaptation to the individual case. As a result of the theory that most of these catarrhal conditions are the result of "catching cold," it has been thought necessary merely to accustom the patient to cold or rather to temperature changes and this has led to an unbalanced and excessive use of cold water treatment. We know now that this method should be employed sparingly in susceptible children, if it is to be useful rather than harmful, and that any rapid cooling of the body-surface by douches, baths, cold sponging, etc., which is not immediately followed by a vasomotor reaction and a feeling of warmth, may directly provoke or

aggravate catarrh and may exercise a bad influence upon the nervous system. The child may be hardened more safely and more easily by accustoming it to remain in the open, even in variable and cool weather. Here, also, extreme and continued cooling of the body is to be prevented by sufficient clothing and exercise. The clothing should not be so heavy as to be impermeable to air, and the head and neck, in particular, should be covered but lightly. It is not wise to send children out to play with bare legs and in thin clothing in cold weather.

The unquestionably favorable influence of fresh air depends not alone upon the direct action of the cold, but also very largely upon the effect of air currents and the greater amount of sunlight. Both of these agencies produce effects just opposite to that of cold, in that they serve as stimuli to the peripheral circulation. A happy combination of these factors is the probable cause of the favorable effect of sea air even in children for whom cold-sea-bathing should be prohibited. Doubtless, the much greater outdoor activity, the resultant muscular exercise and an increased metabolism play a combined part, scarcely to be over-estimated, in this process of hardening.

In exudative children, training plays an even more important part than it does with children in general; because the frequent and inconsequential illnesses of these individuals may in themselves lead to errors of management which in their turn develop those neuropathic tendencies which are often latent in them. It is absolutely necessary that such training should be continuous. It is very important that they be not permitted to look upon illness as a pleasant and desirable experience because of the excessive care and tenderness that are lavished upon them in such events. Malingering to avoid school attendance and other unpleasant and even hysterical manifestations, may result from grave errors in training. If this viewpoint cannot be sufficiently impressed upon the child's parents it may become necessary in extreme instances to advise a change of environment.

Not only exudative and neuropathic but rachitic children as well, require special prophylactic measures to guard them against serious complications. Of these, scoliosis is the most important because of the difficulties of its later treatment. Since this condition is due in the majority of cases to long continued and unchanging position, special emphasis should be put upon the frequent shifting of position, carrying the infant first on one arm and then upon the other, and supporting it so that no bending of the vertebral column can occur. For children in their second year, who are accustomed to sit without support, the rocking-chair designed by Epstein (Fig. 21) is very useful, because it forces the child to employ the muscles of the back to keep its balance while rocking and by keeping the arms high, to hold the back straight while at the same time the weight of the body is taken from the legs—which may at times be necessary—and it is impossible for the child to sit with them doubled under him. For older children, a rocking-horse or a swing may serve the same purpose. Systematic massage may be used successfully.

The child welfare movement is of recent origin and has for its purpose the

prophylactic protection of children. Interest in infant welfare has been aroused, on the one hand, by the well-established fact that infant morbidity and mortality did not show the distinct decrease recorded during the last few decades and, on the other hand, by the absence, at least in European countries, of a normal increase in population resultant upon the diminished birth rate. (See Chapter IV.)

There are two problems, in particular, with which infant welfare work is chiefly concerned. It has been shown that the high infant mortality is found largely among the artificially-fed. In the course of the last decades a reduction both of the number of nursing mothers and of the length of the nursing period has occurred almost everywhere. This is due not so much to a lessened interest in children and their welfare as it is to a false estimate, among all classes of the people, of the effectiveness of artificial feeding and of the extent to which it may replace breast feeding.

Fig. 21.—Epstein's rocking-chair.

It should not be forgotten that an overvaluation of sterilized infant foods and the development of a huge industry for the manufacture of artificial foods (preserved, sterilized, and evaporated milk and infant foods, flours, etc.), almost all of them heralded in the advertisements as the "best" or "perfect substitute for mother's milk," have contributed their harmful influences in lessening breast feeding.

Those artificially-fed infants who are denied not only their natural food but a mother's care as well are in greatest danger. The increased participation of women in the various industries, in so far as it leads to the mother's absence from home, has developed a serious menace which threatens the health and life of infancy and the normal growth and training of older children. Not only does the alarmingly high mortality among infants and the neglect of older children stand in direct relation to this great question, but domestic poverty, the abuse of alcohol, the development of tuberculosis and social evil in general are closely associated with the necessity of wage-earning by the mother with its consequent disruption of the family.

Accordingly, the work attempted by infant welfare organizations should include first of all an efficient propaganda for breast feeding aided by the teachings of physicians in private practice, free dispensaries, the activities of visiting nurses in the homes of the people, the distribution of pamphlets, public lectures, as well as direct appeal, as for example, by the offer of prizes to nursing mothers. It would be well worth while to offer special training to physicians and nurses who after all are the most efficient agents in this propaganda in order that they may be best fitted to carry out their work in this new and special field of social endeavor.

The second and, in the interim, an equally important task lies in the improvement of the methods of artificial feeding or, at least, in the decrease of its dangers. In this matter, individual and popular teaching are little less important than the provision of certified, bacterially pure milk for the poorer classes, either by milk laboratories or dispensaries, and the improvement of the general market supply of milk by careful inspection. The advisability of relying upon milk which has not been boiled is open to serious question. The actual work in this line is the more difficult because it must be so conducted that it will not discourage breast feeding, the attainment of which alone will insure the lasting benefits of infant welfare work in the economics of the nation.

Finally, the third and most difficult task is the expansion and regulation of actual economic assistance in so far as it leads to the complete protection of the infant. Special emphasis must be laid upon the fact that the separation of the mother and infant must be prevented by adequate aid to the mother and that when this is impossible the child should be placed in an institution where it will receive proper and continuing care. The law fixing the responsibility of the father for at least the provision of food for his illegitimate children has proved successful. Lying-in-hospitals where the nursing mother and child are cared for and housed for a sufficiently long period serve beneficently the welfare of both. Physicians experienced in infant welfare work should see to it that the laws provide generous financial assistance, enough to amply cover all the needs of the infants and that the best of care be given those who are placed either in such institutions as day nurseries or infant asyla. Wherever there is overcrowding, babies are exposed to the dangers of respiratory infection. In estimating the value of any of these modern attempts at infant welfare work, which often represent great pecuniary sacrifices, alike on the part of the charitable giver and the taxpayer, we must always remember that their aim is not only to preserve the lives of a certain number of infants, but also to guard the survivors from serious and irreparable physical and mental injury. This latter is probably the more important result and fraught with larger consequences to the nation.

The care of children of the run-about-age has been provided for by welfare organizations, lay associations and large industries by means of day nurseries, schools and the like; but this aid is still inadequate in many respects. As we have already suggested, the lack of proper physical care during this period may not cause any marked increase of mortality but it does produce a high morbidity. The after-results of disease and of errors in feeding during infancy are brought out with particular prominence during this period. Rickets especially becomes aggravated and results in more or less irremediable deformities of the extremities, the spine, the thorax, etc. Perhaps there should be included here "bony cavities of the face"—deformities which may play a part in causing chronic nasal infection. It is at this age that careless training produces its most injurious consequences, because it is a time in which the foundations of character are laid and when the counter influences of the school are lacking.

Children of school age are provided for in many ways. In the building of the newer type of school-houses much more consideration is given than formerly to air space, heating, ventilation and lighting. Light should not only be adequate but should fall over the child's shoulder. Many new schools are indeed models from a hygienic point of view. By providing abundant daylight, proper fixtures for artificial lighting and large print in school books, myopia, a school disease, is measurably avoided. The hygienically correct arrangement of the desk, with proper distance between seats, with suitable backs, and writing surface should prevent incorrect postures from which scoliosis may arise. It should be emphasized that permanent scoliosis is a definite deformity, generally rickitic in origin, which may still be corrected at the period when the child enters school.

To safeguard the school from the dangers of infection it is sufficient to prohibit the return of convalescents until the period of contagion has entirely passed and to exclude early all children suspected of communicable disease. The school physician should be the advisor of teachers and parents upon questions of hygiene. He should call their attention to any diseases or abnormalities which he may discover in the child. Doubtless by this means a large number of children will receive timely medical attention which, but for the school examination, they might have lacked. School inspection is an important prophylactic measure.

Children with defects of the sense organs and the seriously crippled should be cared for in special institutions. Subnormal children should receive training graduated to their ability in separate schools. For physically weak school children and particularly for those who show indications of tuberculosis, open-air schools, or classes, have been established in many communities. Philanthropic associations provide for children in summer vacation camps in the country, in the mountains or by the sea.

An old question, frequently leading to heated discussions, based more on sentiment than on actual knowledge, is that of overwork in the schools. It is not to be denied that children who are weak, sickly or neuropathic are easily tired, but it is hardly to be expected that schools for normal children can reduce their standards or change their methods out of consideration for a few abnormal individuals. The selection and classification of subjects taught, on the one hand, and, on the other, the grading of school children and the demonstration of their degrees of educational progress are naturally and must remain the problem of the pedagogue or of the school authorities. The attempt has often been made to prove overwork in the schools by an experimental demonstration of the children's weariness. The results so obtained cannot be accepted without further corroboration. Careful medical observation, which alone may competently decide the question, records no signs of overwork in the healthy child. The real cause of the difficulty, commonly laid to the school, is generally a matter of improper home training. Improper feeding of the child may also be a not insignificant cause of school fatigue. The rôle of actual underfeeding, unbalanced diets, bad eating habits is just at present being realized. When this is not true and when the child cannot keep up his school work in spite of suitable

home conditions, there appears no other alternative but to give him more time to do the required task, or to put him in lower grades where the work is easier. For the children of the well-to-do, who for obvious reasons decline these alternatives, there are numerous training institutions or schools which, while costly, secure good educational results by wise individualization and scrupulous physical care.

With legal safeguards against abuse and industrial exploitation and with the work of humane societies in caring for neglected children, the list of agencies for the protection of the child is complete. The delinquent child is also coming in for his share of study and treatment.

(*b*) GENERAL THERAPY

If the well-known axiom of all therapy that we must treat the patient and not the disease is recognized, it means that not only do numerous variations in the course of disease present indications for differing therapeutic measures, but that over and above all other considerations, the individuality of the patient must be studied. It will be readily understood that the therapy applicable to childhood and more particularly to infancy, presents special problems incident to the peculiarities of the infant organism and to the pathology of childhood.

In the child certain symptoms or symptom complexes are characterized by their frequency or by their unusual clinical importance; and these, with the therapeutic methods adapted to them, will be discussed. The question whether and to what extent, in a given case, a symptom may be combated symptomatically must be left for inquiry to the special chapters of the book.

Fever.—If fever cannot be treated locally, as by incision of abscesses, by paracentesis in purulent otitis media, or by the water diet in alimentary fever, it may be treated symptomatically by the avoidance of food to prevent heat production or by the more commonly practiced attempts to increase heat dissipation.

The fact that the child is so much more easily handled than the heavy adult and that the bath for the child is more simply and easily prepared, makes it possible to use this cooling agent more freely than in later life. A bath at 30° C. (86° F.) or warmer, continued for a long period, is more efficacious than one in which the water is at 20° C. (68° F.), or colder, which while it produces a rapid cooling of the surface, yet on account of the contraction of the skin capillaries to the point of pallor, or even cyanosis, secures only a minimal cooling of the deeper tissues. Even baths at 35° C. (95° F.) the temperature of the cleansing bath, without the addition of colder water, will have a cooling effect, the difference between this temperature and that of the fevered body of the child being great enough to produce sufficient reduction of the body-heat in five to ten minutes, a reduction moreover, which is lasting. Even a large clinical experience does not enable one to predict in a given instance the amount of the cooling effect of a bath; so that a definite time limit is not justified. Since the skin temperature while in the bath is misleading, it may be well to interrupt its application in order to take the actual (rectal) temperature and deter-

mine whether the bath should be continued. Frequently the general condition of the child, the clearing of the sensorium, the improved heart action, or on the other hand, the appearance of chilliness, will suggest that the temperature has been sufficiently lowered.

The desired effect may be secured by moist packs or may be attained in a simpler manner, which in many cases is quite sufficient. With the moist pack it is not advisable to prescribe the exact temperature of the water because it is impossible to control its changes during preparation. For an infant it suffices to have the water at room temperature or better still, somewhat warmer. Only with older children should water below 20° C. (68° F.) be used and then only rarely. As a rule, cooling packs are applied only to the trunk, leaving the arms free; but, with high fever, when a full bath is impracticable or is contraindicated, the extremities may also be included in the pack.

The sheet intended for the pack should be of six or eight thicknesses and should be wrung dry enough to prevent dripping. It may be covered with a loosely woven woolen or knitted blanket so that evaporation from the pack may occur gradually. The cooling effect depends chiefly upon this evaporation. So soon as the pack becomes dry it prevents the radiation of heat and therefore it should be changed at once or, if the child cannot be moved, it may be moistened again by carefully pouring water over it. If the pack is to remain in place for a long time it is well to protect the skin from irritation or infection by its inunction with a bland ointment.

A very harmless, but less active form of cooling is achieved by the use of moist compresses to the thorax, to the abdomen, to the head, or to the arms and legs. Cold sponging of the skin also comes under this head. It is especially valuable in those cases in which the child, on account of the nature of its disease, must be kept as quiet as possible. In the use of the ice-cap, which is best avoided with infants, care must be taken that too great cooling does not take place. Cold water enemata have sometimes been recommended in the treatment of hyperpyrexia.

In comparison with these hydrotherapeutic measures, medicinal agents for the treatment of fever in young children occupy a less important place. Such antipyretics as acetylsalicylic acid, antipyrin, dimethyl-amido-antipyrin, etc., are of advantage only when their use is dictated by their more or less specific action in certain infections, such as influenza, rheumatism, some cases of tonsillitis, etc., or when they are simply used to provoke perspiration. Preferably physicians prescribe quinine or one of its less bitter derivatives, a practice justified by the fact that quinine not only increases heat radiation but also decreases heat production. These latter drugs are little used in this country to control fever.

Antipyretics are more widely used for older children, but even then only in those who suffer from severe subjective symptoms consequent upon fever. In these cases it is often possible to relieve the sleeplessness, anorexia, headache, pain in the limbs, etc., more completely and more easily by an antipyretic than by the use of baths or packs.

The use of alcohol, even though it has a cooling effect through its dila-

tion of the peripheral circulation, and although it produces a certain euphoria by its narcotic action, should be scrupulously avoided in children of every age. It may be used as a stimulant in threatened collapse and may be countenanced in those cases in which an increase in the volume of the respiration is desired; but even in these conditions, other remedies may well be used. It is necessary only to remind the reader that besides combating the fever, good care and proper feeding must be given due attention.

Most of the medicinal agents noted, as well as the hydrotherapeutic measures employed against fever, have a tendency to cause sweating whenever the loss of heat is prevented by the clothing covering the patient. This is a therapeutic effect often sought in children, but with respect to it, two precautions must be taken. First, it is not permissible to give infants large quantities of milk to produce perspiration. Some form of hot tea, possibly sweetened with benzosulphinidum (saccharin) may be given instead. Plain or sweetened water is the American equivalent for the commonly used tea of German authors. Second, all methods of inducing heat production or preventing heat radiation are contraindicated in spasmophilic and lymphatic children, since these measures may readily cause hyperpyrexia and heart lesions. Pilocarpin is used for children as a last resort and then only in those whose heart action is strong.

For combating subnormal temperatures, the same methods are used as are designed for their prevention, *viz.*, the incubator, the warming tub, hot water bottles, etc. The use of the incubator and of Crede's warming tub is, of course, limited to suitably equipped institutions. In the home any large bottle which can be securely corked may be used. The attention of the mother or the nurse must be called to the danger of scalding the infant from a leaking bottle or of burning it by direct contact with the hot bottle insufficiently wrapped. This accident may also occur with the various forms of electric heating pads. When it is necessary to warm the child rapidly the hot bath is to be preferred. Such a bath may be begun at a temperature of 35° or 36° C. (95°-97° F.) gradually increased by the careful addition of hot water to 40° C. (104° F.), or even more. During this bath the child should be supported with the left hand while it is rubbed energetically with the right.

Since a subnormal temperature is usually accompanied by other manifestations of collapse, and particularly by cardiac weakness and a diminished reaction to stimuli, it is customary to counteract the analeptic action of the hot bath by dashing cold water over the chest or back. Slapping the skin with a cloth dipped in cold water serves the purpose equally well. These methods, like the use of the mustard bath or pack, do not lessen the beneficial effect of the hot bath, but on the contrary they stimulate the cardiac and respiratory activity. Subnormal temperature frequently indicates dehydration, demineralization or both. Saline or Ringer's solution by mouth or per rectum will often cause a return to normal of the temperature.

A mustard bath is prepared by placing four or five tablespoonfuls of ground black mustard in a muslin sack and steeping this in hot water for several minutes. By this means strong mustard vapors are given off which

are irritating to the mucous membranes and may increase an existing bronchitis. For this reason the mustard bath has been almost entirely replaced by the mustard pack, the use of which is described under capillary bronchitis. The mustard pack cannot be used with lymphatic children or for those with widespread eczema. In mild cases or in emergency, when neither the hot bath nor the mustard pack can be prepared quickly enough, the warmth of the skin and the resulting stimulation of the circulation may be promoted by rubbing the body energetically with the bare hand or with a dry cloth or, even better, by rubbing the surface with spirits of mustard, camphor, a volatile liniment, or a mixture of equal parts of alcohol and oil. As internal remedies, alcoholic stimulants and black coffee are readily obtainable. What has already been said about alcohol applies here as well. Its stimulating action is transitory and since it is certainly not beneficial to the gastric mucosa it were better avoided altogether. There is no danger in using coffee, as hot as possible, in the form of an enema. It warms the patient and at the same time has a stimulating effect. Even when given in large quantities, it is not followed by paralysis or by any other symptoms of poisoning. Free use of it, even in infancy, may be strongly recommended. In comparison with coffee, the stimulating action of black or green tea is too slight to justify its therapeutic use.

Medicinally the circulation may be stimulated most quickly and effectively by the subcutaneous injection of camphorated oil. Even in infants not less than 0.5 c.c. (7 minims) and even 1-2 c.c. (15-30 minims) may be used. If necessary, these doses may be repeated every hour for days at a time. Care must be taken, however, when so frequent medication is employed, that the injections are not made too closely together lest they cause necrosis and, in cases of existing bacteriemia, abscesses.

Caffein, with sodium benzoate, may be given to infants of six months, or over, in doses of 0.03 gm. (½ gr.); in doses of 0.06 gm. (1 gr.), during the second and third years; or 0.1 gm. (2 grs.), during the fourth to the sixth year; and in children of school age, 0.15-0.20 gm. (2-3 grs.), three to four times a day. Such doses may be prescribed in a ten to twenty per cent. solution in sterile water. Its action is quick but not lasting.

For very rapid results, epinephrin, is very useful. From 0.2-0.3 c.c. (3-5 minims) of a 1:1000 solution may be used for one intramuscular injection in infants. Still larger doses may be given older children. As an adjuvant to the intravenous transfusion of saline solution it meets with insuperable technical difficulties in young children, but Pospischill has succeeded in the subcutaneous injection of as much as 60 drops, in 150 c.c. of physiologic salt solution, as often as two to four times a day, in older children.

Digitalis acts more slowly and, therefore, for a longer time. An infusion of digitalis 0.3-0.5 gm. (5 to 8 grs.) in 100 c.c. (3 ounces), for infants and even more for older children may be used within a period of three days. On account of the cumulative action of digitalis it is necessary, if the drug is used for a long period, to alternate its exhibition with intervals of withdrawal. During these intervals the tincture of strophanthus (1-5 drops, every three

hours), or caffein may be given. Instead of the infusion of digitalis, digalen, digitoxine soluble or a solution of digipuratum may be used with good results. Of either of these preparations two or three drops may be given to infants, and ten or twelve to older children, three times daily. Sterile digalen and digipuratum, or similar preparations, are obtainable in ampules and may be injected intravenously or intramuscularly if more rapid absorption is desired. Their influence upon the circulation is decidedly less marked than that of the infusion but it is equally necessary to watch for its appearance and especially for the possible retardation of the pulse. The effect of drugs of the digitalis group is often difficult to estimate, especially in young children and the dosage therefore will depend on conditions. The quantities given above are to be regarded as average doses and more may be freely given—usually in vain—where severe cardiac collapse is apparent.

All of the so-called heart stimulants act, also, as stimulants of respiration, but they fail in cases in which paralysis of respiration is the most prominent symptom, as indicated by the alternation of respiratory pauses and periodic breathing while the heart action remains actually or relatively strong. In such cases the inhalation of three litres of oxygen a minute, for five or ten minutes, repeated two or three times in an hour, may relieve the exhausted expiratory centre and by relieving it and thus increasing the oxygenation of the blood may interrupt a dangerous vicious cycle. With the exception of laryngeal stenosis, I have, however, scarcely ever observed a permanent result even if oxygen is given in large quantities although temporary benefit was achieved.

If the disturbance of respiration is due to obstruction of the nasal breathing, often occurring in young children who experience some difficulty in learning to breath through the mouth, relief may be obtained by the mechanical cleansing of the nose with a dry cotton applicator, or with cotton saturated with glycerin, or, if there is much swelling, with epinephrin. Cotton tampons, moistened with a fresh solution of epinephrin 1:3,000 may be placed in the nostril after it has been cleaned. Serious obstruction of respiration in the larynx may necessitate intubation or tracheotomy.

In infancy the problem of preventing a dangerous loss of water from the body or of stopping such loss as quickly as possible often presents itself. The most simple method, that of permitting the child to drink large quantities of physiologic salt solution,[2] or of slightly alkaline mineral water, or of weak tea containing salt, is often thwarted because the seriously sick child refuses fluids or because of persistent vomiting. In these cases, it is often possible to obtain the same results by repeated rectal injections of these solutions, in quantities of 50-100 c.c. (1½-3 ounces) at body temperature, by means of a Nelaton catheter introduced as far as possible. To prevent the immediate expulsion of the fluid, the nates should be gently pressed together for several minutes. If it proves impossible to secure the absorption of sufficient fluid from the bowel by this method, enteroclysis, a more elegant method of administration, may often be employed successfully.

[2] For the explanation of the reason for the addition of salt, see Chapter I, page 18.

The apparatus for enteroclysis consists of an irrigator with a long rubber tube, controlled by a glass stop-cock with a catheter attached to it. The catheter is passed as high as possible into the rectum and held in place by adhesive straps. The flow is regulated by means of the stop-cock, so that 30-40 drops a minute, or 90-120 c.c. (3-4 ounces) an hour are instilled. By this method it is possible, without causing distension, to give 200 c.c. (6 ounces) several times a day. Any of the solutions mentioned above may be used or, to avoid the pyrogenetic action of the sodium chloride, Ringer's solution (sodium chloride 7.5 g.; potassium chloride 0.42 g.; calcium chloride 0.24 g. per litre) may be instituted. The rectal application of many other drugs may be practiced to the advantage of the patient. Sodium bicarbonate in 2-5 per cent. solution and glucose are frequently used. The latter substance is tolerated even by the infant in surprisingly high concentrations. I have given up to 25 per cent. glucose solutions to infants without the least sign of irritation.

The most rapid and certain method of increasing the fluids of the body is by hypodermoclysis, in which either sterile physiologic salt solution (0.7 per cent.) or, preferably, Ringer's solution may be used.

A large serum syringe or a canula, attached to a funnel by means of rubber tubing may be employed for the purpose. In infants who have lost much water, from 50 c.c. to 100 c.c. (1½-3 ounces) may be introduced. This quantity may be injected gradually through a single puncture of the skin over the chest, abdomen or back by occasionally moving the needle. Its painfulness has reserved this method for extreme cases in which, however, it is often the only means of saving the patient, when it may be repeated as often as four times a day. The intraperitoneal injection of salt solutions is also now being practiced more than in the past.

Frequently and under varying circumstances, it is found necessary to induce rapid and complete emptying of the intestinal tract. Enemata or colonic flushings empty the large intestines only and are especially efficient when it is necessary to remove hardened fecal masses from the lower bowel.

In using the small rectal syringe, which carries 30-50 c.c. (1-1¼ ounces), the hard rubber tip should not be directly introduced into the bowel, because by any unexpected movement of the child serious injury may be produced. The tip should be armed with a Nelaton catheter or a rectal tube. This is also true of the hard rubber irrigator tip.. A rectal tube, which, even for the infant may have the circumference of the little finger, should be inserted as far as possible, the pelvis being propped upon a pillow with the child lying on its back or side. The nates should be pressed gently together after the injection, in order to hold the enema in the bowel for a little while to permit it to dissolve the scybala.

The enema may also serve the purposes of a flushing. For this purpose it is better to use a funnel attached to the tube rather than the irrigator. The tube should be somewhat larger than that used in gastric lavage (*q. v.*). A tepid physiologic salt solution, or astringent, laxative, antiparasitic antiseptic solutions, etc., may be used according to the desired effect. Enemata of 30-100 c.c. (1-3 ounces) of oil will serve to soften scybala.

Glycerin, one or two teaspoonfuls with an equal quantity of water, is said to act as a stimulus to the peristalsis of the lower bowel. An identical result may be had by the insertion in the rectum of the well-known glycerin suppository, small forms of which, suitable for children, are obtainable; or by the similar use of the soap stick, a common domestic device, of about the thickness of the little finger and from 3-5 cm., (1-2 inches) long, prepared from any ordinary laundry soap.

The medicinal cathartics are much more satisfactory for the complete evacuation of the whole intestinal tract. For decades, calomel has undeservedly ranked as the most popular cathartic. A dose of 0.01-0.05 gm. (⅙-1 gr.) may be given to the infant, and from 0.05-0.1 gm. (1-2 grs.) to the child of three years, or more may be given every two hours until results are obtained. That calomel has the obstipating effect ascribed to it when it is given in small and infrequent doses is a very questionable matter, since it is not an intestinal antiseptic. There is, however, no doubt that it frequently produces an intestinal irritation far in excess of its desired result and that it is injurious at least in so far as it increases the intestinal secretion which is very prone to decomposition and putrefaction. For this reason, it is hardly ever given to infants by the modern pediatrist. Its place has been taken by castor oil, a much less harmful remedy which young children take readily, especially if it has been thinned by warming it in the spoon. It should be given in sufficiently large doses of one or two teaspoonfuls at a time. Older children will take it without objection if the tongue is first covered with sweet chocolate or if the oil is given in the form of an emulsion with equal parts of the aromatic syrup of rhubarb as recommended by Henoch. Milk of magnesia can be used for those infants and children in whom the mildest laxative action is desired.

Phenolphthalein, in tablet or candy form, is very efficacious in children of three years or more. It produces one complete evacuation of the bowels and is entirely painless in action. In younger children castor oil or, if necessary, the compound rhubarb powder will usually be found adequate.

In older children, the saline laxatives may be considered, particularly in that degree of habitual obstipation which demands medicinal treatment. It is best to begin with one teaspoonful of artificial Carlsbad salts in a wineglassful of warm water, increasing or diminishing the dose as required. Syrup of senna may be used, but it is somewhat objectionable on account of the colic which it is apt to cause.

For the forced emptying of the stomach emetics are seldom used nowa-days; since the result may be attained with less injury and more completely by gastric lavage, which is technically simple in young children. The child is laid upon its side, with its face slightly downward, as shown in Figure 22, so that any vomited matter passing along the side of the tube may run off and escape aspiration—the only caution that needs to be observed. The apparatus employed consists of a funnel of 150-200 c.c. (5-8 ounces) capacity, to which a catheter of about the diameter of a lead pencil is attached by means of a piece of tubing about a yard long. This should have glass connections but no stop-cock. In the infant, the catheter serves well

as a stomach tube. Being passed directly to the posterior pharyngeal wall it causes slight gagging and then swallowing movements. The catheter is passed very readily. There is no danger of forcing the tube into the larynx and the first slight gagging soon stops if the tube is quietly held in place for a moment. The rest of the maneuver, the extraction of the stomach contents, and the washing with water or with physiologic salt solution at body temperature is carried out as it is in the adult. The tube must be moved up or down a few centimeters so that its eye dips into the fluid, instead of lying

Fig. 22.—Gastric lavage.

in the air space above, the so-called "gastric bubble," in order to remove the last remnant of fluid used. A few large curds, which occasionally form in the atonic stomach, may escape the lavage, but for its therapeutic purposes this is of no great importance. The addition of various medicinal agents in lavage has not been found practical. The use of the stomach tube for purpose of feeding, where food cannot be taken or where it is refused, deserves especial emphasis.

Of the various astringents employed in the treatment of the diseased intestine, tannin is still in most common use. In irrigation of the bowels a .25 per cent. or .33 per cent. solution is preferred, but it reaches only the

lower portion of the large intestine. To affect the entire tract, tannin must be given by mouth. Many modern preparations which pass the stomach more or less unchanged and which gradually liberate tannic acid in the intestine, such as diacetylic tannic acid, tannigen, tannalbin, tannoform, tannismut and others are available. Since any excess passes through unchanged it is not necessary to gauge the prescribed amount carefully. Bulk powder is usually ordered with directions to give a small quantity several times a day. As the powder is insoluble and rather bulky it is best given stirred into a thick gruel.

A two per cent. of aluminum acetate may be used for irrigation instead of the tannic acid solution. Starch enemata (1 teaspoonful of starch, in 150-200 c.c. (5-7 ounces water), water heated to form a paste. Of this solution 30-50 c.c. (1-2 ounces) may be injected at body temperature. Although frequently recommended, it has no astringent action but may be used as a vehicle. The starch is supposed to act as a protection to the mucous membrane but this effect seems to be rather doubtful.

Beside the tannin preparations, bismuth is often prescribed as a medicinal astringent. Bismuth subnitrate or subsalicylate suspended in mucilage of acacia (20 gms. to 100 c.c.) may be given to infants in doses of 0.2-0.3 gm. (3-5 grs.) every three hours. The stools become grayish-black from the bismuth and sulphur compound which is formed in the intestine. Tannismuth, colloidal bismuth oxide (Birmon), etc., are new preparations of bismuth which may be used.

The anti-diarrhœic action of these preparations depends upon their astringent action and their diminution of the secretions. Kaolin (bolus alba) and animal charcoal (carbo animalis) given in large doses [5-10 gms. (45 grs.—1 dram) in 100-150 cc. (3-5 ounces) of boiled water] three times daily has a different effect. It is of purely mechanical influence, due to the fact that the fine grains of the powder, thoroughly mixed with the intestinal content, prevent the access of the intestinal bacteria to their nutritive media and thus limit their fermentative action. Unless the intestinal canal is at least in part cleaned of its contents or unless no food is being given, little is to be expected of the bolus or charcoal therapy.

Opium as an anti-diarrhœic measure in children demands special discussion. Its danger to the infant organism has been generally over-estimated and evidences of disorder which really belong to the picture of certain severe alimentary disturbances have been erroneously regarded as the result of small doses of opium. For this reason the use of opium has been generally condemned in young children. It is true that opium is not often indicated for its anti-peristaltic effect, but while it diminishes peristalsis and to a certain extent absorption, it does not in any way diminish the decomposition going on in the intestinal contents. Just as its use is justified for the control of a spasmodic cough, so its symptomatic employment is permissible to arrest too frequent evacuations, excessive loss of water, severe colic or repeated rectal prolapse. It is clear, however, that this symptomatic treatment may mask a serious basic disease, which in young infants clinical experience teaches is often a very dangerous thing.

As to the dosage of opium, H. Neumann advises, perhaps quite too conservatively, the addition of one drop of the tincture or wine of opium to 50 c.c. (1⅔ ounces) of water of which solution infants under six months of age receive one teaspoonful and in the second half-year a dessertspoonful, (1½ drams), every hour, until the bowel movements have been stopped for three hours. With increasing age the dosage is advanced, until children of three to six or eight years receive three to six drops not oftener than every three hours. These doses are exceeded only in exceptional cases, as for example, in appendicitis or peritonitis. In such cases it is better to use the drug in the form of extract of opium given in suppositories. So prescribed, 0.01-0.02 gm. (⅙-⅓ gr.) may be used for children from three to six years old; and 0.02-0.03 gm. (⅓-½ gr.) for older children according to necessity, three or four times a day.

Hot compresses, very hot dry packs, or poultices applied to the abdomen to relieve severe abdominal pains, may be used instead of opium. While their action is less certain they are entirely harmless. Dry applications must be changed frequently and at least every half or three-quarters of an hour; they should be very hot and covered with dry cloths. The favorable influence ascribed by mothers and practical nurses to the various kinds of herb-tea in the relief of colic is probably not specific. They are permissible, since when they are being given the child usually receives no other food—a most important indication in such cases.

Aside from the drugs noted, the use of narcotics in children should be confined to the relief of troublesome cough and the induction of sleep. The first of these objects, the more complete discussion of which is left to special chapters, we will treat but briefly here. The most useful narcotic is codein sulphate, dissolved in sweetened water. It may be given to infants in initial doses of 0.002 gm. (1/30 gr.) three or four times a day. In some cases it may be necessary to increase the dose materially, 0.003-0.005 gm. (1/20-1/12 gr.) in order to obtain results. In children of the run-about-age, a further increase to 0.01-0.015 gm. (⅙-¼ gr.) may be required. If codein in these doses does not produce the desired effect, the use of morphin sulphate, which in children is fully as objectionable as opium, is permissible, but requires careful observation. The doses should be about one-third as large as those of codein.

The soporifics require more detailed consideration. Habitual insomnia, either a difficulty in falling asleep, which may continue for a period of hours or an abnormally light sleep is not at all uncommon during childhood. Since in insomnia we have to deal with a general neuropathic symptom, therapeutic measures must be aimed at the basic disorder, or rather, in a given case, must be directed to the training and environment which have led to the disturbance of sleep. It is better to treat the case with tepid packs or long continued hot baths, etc., than to employ sleep-producing drugs, which should be reserved for exceptional cases. This, is not true, however, of a temporary sleeplessness, caused by severe acute disease, which in itself weakens the patient. The uncontrollable restlessness of certain infants suffering with disturbances of nutrition or the sleeplessness

which occurs with older children in the course of septic fevers, typhoid, miliary tuberculosis, etc., is of this order. Under such circumstances, the indications for procuring sound sleep are no doubt pressing. In infants the most common remedy employed for this purpose is chloral hydrate. Since this drug acts rapidly, it is well to give one teaspoonful of a one to two per cent. solution, every fifteen minutes or half an hour until sleep is produced. By this method of administration no injury is ever done and even though the patient be especially resistant to the drug we are usually quite certain to obtain the desired effect. Of course the doses required will vary greatly, several decigrams being necessary in some cases. Even a single dose of 0.5-1.0 gm. (7½-15 grs.) given in a two per cent. solution per rectum in a case of frequent eclamptic convulsions in infancy, is wholly without injurious effect upon the heart or the blood-pressure and will stop the attacks within ten or fifteen minutes and produce several hours of sleep.

Chloral hydrate is a very good soporific for older children, but is often refused because of its acrid taste. As a substitute diethyl-barbituric-acid [veronal 0.1-0.3 gms. (2-5 grs.), for children from three to twelve years old] or sodium diethyl-barbiturate (sodium-veronal) which is more soluble and therefore acts more rapidly, is excellent.

Since soporifics are only required temporarily, and often for a single dose, there is no necessity for change; these remedies, therefore will be found sufficient.

The discussion of the expectorants, indicated in congestion of the smaller air passages, due either to the absence of the cough stimulus or to the viscosity of the secretion; of the narcotics, as employed for the relief of cough; of the diuretics, hemostatics, anthelmintics, and of the entire armamentarium of serum therapy may be referred to special chapters of this work.

The group of tonics, however, requires general discussion, since children who are subject to such treatment are very common in every physicians' practice. A child is often brought to the pediatrist with a lay diagnosis, sanctioned actively or passively by the physician of "watery blood" or anemia, based upon the patient's faulty nutrition, pallor and apparent weariness, or upon a series of such functional disturbances as headache, anorexia, etc. The pediatrist is then expected to relieve the condition by means of tonic remedies.

The number of preparations offered for this purpose is so great that no physician need hesitate for a choice if he is willing to prescribe untried remedies without reference to their effect in any particular case. A large field is open to him who wishes to make more or less aimless trial of these innumerable combinations of nutrients and alterants, into which iron, arsenic, iodine, quinine and phosphorus enter in indiscriminate variation. It goes without saying that such methods are unworthy of the thinking physician and are not to be excused by the ignorance of anxious parents. Even if the physician wishes to employ a medicinal agent for its purely suggestive influence, he must nevertheless determine, through physicial examination and careful inquiry into the manner of life and the training

of the child, the basic cause of the given disease and must attempt, as a physician and an educator, the removal of such cause. A large share of the disturbances, which lead to a demand for this sort of therapy, are of functionally nervous origin in children, as they are in adults. In early childhood, suggestive therapeutics of a medicinal character have not the same justification as they bear when they are addressed to the adult, because young persons are not responsive to such measures. The empiric use of the so-called alterants is the more narrowly restricted on this account.

Information concerning blood-forming remedies, with reference to their usefulness in hemic disease, may be found in the chapter on Anemia.

The artificial food preparations of which, also, an extremely large number are on the market, are used very extensively for children who are either well or ill. While extended study of the pathology of infancy has taught the pediatrist definite indications for the increase or decrease of the several elements of the selected dietary, older children are carelessly given these manufactured preparations upon the strength of preformed conclusions of merit or of accidental successful experience of their use.

The feeding of artificial protein preparations is not justified for the purpose of increasing the deposit of fat in a lean child. Only when an exhausting disease has reduced the protein content of the organism and it is desired to increase the nitrogen retention, by an increase of the nitrogen intake, is such a procedure indicated. Under any other circumstances high intake results in an increased nitrogenous output which cannot be considered desirable. Of food preparations consisting largely of fats or carbohydrates, the former, if they are well borne, must be given the preference. A justly high value has been placed upon cod-liver oil and its emulsions; but this cannot be extended to the substitutes for cod-liver oil, since these are of entirely different chemical composition. Among the carbohydrate preparations the numerous malt extracts, often combined with iron, iodine, etc., enjoy some popularity. Several of these have a mildly laxative action, which dry maltose and dextrin preparations do not possess. A favorite food of high carbohydrate content and always in favor with children is cocoa or chocolate.

Even physicians who permit the temporary use of alcoholics or consider them necessary in cases of acute weakness, unamimously condemn the daily use of alcoholic tonics or of wine, heavy beer, etc. Alcohol should be used only as a vehicle for other drugs and especially for the stomachics, as in the wine of pepsin or the aromatic tinctures (compound tincture of cinchona, etc.), which are given by the teaspoonful or by drops. The action of agents which stimulate the appetite chiefly by their taste is naturally very slight in young children; but in older ones is more definite. Pepsin and dilute hydrochloric acid, in teaspoonful doses, immediately before or after feeding, are occasionally useful; but it should be remembered that a temporary loss of appetite may dictate a wise refusal of food which may not be combated without harm.

In the habitual anorexia of children of the run-about or school age, such remedies as ichthyol albuminate and the like, give much less satisfactory

results than does a careful regulation of the diet. Feeding should never be urged. It is especially necessary to treat the frequently coexisting habit of obstipation. These and other influences, not infrequently chronic infection of the nose and throat, affecting the psychic well-being of the small patient should be removed. The child is often of a nervous temperament and surrounded by neurotic adults and it is sometimes necessary to place it in entirely new and more favorable surroundings. The surprisingly good results often attaching to its removal to a children's home in the mountains or by the seaside are largely due to a change of environment.

While the psychologic factors of treatment are particularly emphasized, it is not to be denied that such hygienic influences as light, air, exercise, bathing, etc., are largely involved in bringing about a good result. Nevertheless, it remains true that the reputed benefits of a change of air are achieved less by climatic than by psychic agencies. Especial weight is to be given to intensive exercise in the open, which, with its variable qualities, does not become tedious and does much to alleviate the illness of the child. The action of light is only second in value, while bathing is a less important matter. This conclusion seems justified by the fact that sea-baths given at home, in the patient's habitual surroundings, have little or no effect. Furthermore, the strong stimulation of the skin and the nervous system induced by cold sea-bathing, by-the-sea, must be more carefully watched and adapted to the conditions of the child than is necessary in the enjoyment of the less active influences of light, air and pleasurable exercise. In every case the attendant should see to it that cold sea-baths are of very short duration, not exceeding one or two minutes, that the child moves about actively in the water and is warmed up as rapidly as possible after the bath.

When a number of children are bathing together it is usually possible to make the timid among them go into the water without persuasive force. If this is not possible, and skilful and repeated attempts at persuasion fail, the repetition of force in giving the child the cold bath only increases fear and becomes an injury to the nervous system.

Sea-salt and sun-baths have become widely popular in the effort to build up feeble and particularly scrofulous children. Experience corroborates the conclusion already reached that at best they are but adjuvants of treatment and, as given in the home, are inefficient, whereas, when combined with other therapeutic measures at the sea-side, they seem more effective.

In the matter of technique, the following may be noted: Enough salt, (table salt, sea-salt) is used in the bath to make a 1.5-2 per cent. solution, using 30-40 litres, or 7-8 gallons, of water for infants and 100-200 litres, or 25-50 gallons, for older children. If there is much insoluble sediment, the salt may be first dissolved in boiling water and then strained into the bath. The bath temperature should be 32-33° C. (88°-90° F.) for the babe, and about 30° C. (86° F.) for the older child, a little less than that of the ordinary bath. It should be given for ten or fifteen minutes. Frequently the bath is followed by a cool rub down in the sun, to increase the reaction of the skin.

It is well to begin with two baths a week and to increase the number only if the patient reacts well.

Convenient as it would be for the physician, it is impossible to give any definite directions for the dosage of various drugs in childhood. A very indefinite approximation may be made by giving that fraction of the adult dose which corresponds to the fraction represented by the child's ascertained body-weight, as compared to that of the adult, estimated at 60-70 kilos, or 120-150 pounds. By this method, the permissible dose for the young infant will hardly ever be exceeded and it may more often fall below the amount required for a desired effect. Since the administration of drugs to children is usually confined to those of definite, more or less rapid and readily recognized action, and without dangerous qualities, it is often better to prescribe medicine in divided doses, repeating the small dose until the required result by way of bowel movement, sleep production, disappearance of fever, or active perspiration is obtained. The choice of intervals between doses must, of course, depend upon the rapidity with which the result is to be secured. With chloral, or acetylsalicylic acid, for instance, it may vary between fifteen and thirty minutes; with cathartics it may range to two or three hours. With certain drugs, it may develop only after a much longer period, when smaller or less frequent doses than those required to produce the initial effect should follow. In the use of drugs having a cumulative effect, as with digitalis, or of those which show a diminishing strength of reaction or which lead to the development of tolerance, the same practice should be adopted with children as in adults. A generally increased sensitiveness to only a few drugs exists in childhood. Opium belongs in this class, as shown by recent experiments upon animals. It has been already said that its injurious action is in part, at least, due to a misunderstanding of the indications for its use or to a failure to recognize the supposed symptoms of poisoning (see page 115). Infants show an actual intolerance to phenol; and it is well, therefore, to avoid its use altogether wherever there is a possibility of its absorption in perceptible quantities. Chloroform, also, is dangerous for infants and it is better to use ether exclusively for purposes of general anesthesia. It is hardly necessary to add that such poisonous drugs as cocain should be especially avoided in favor of non-poisonous substitutes.

The exhibition of medicinal substances necessitates deviations in other respects from the rules which govern their prescription for adults. The young infant, even if he be not entirely without the sense of taste, has that function so poorly developed that it is hardly ever necessary to disguise the taste of drugs. Even in the second or third year, children with disturbances of nutrition or rickets have a sense of taste so slight that they will take cod-liver oil without objection. This statement needs the more emphasis because advertisements of cod-liver oil preparations, wrongfully claim that children can be made to take the oil only by force. If at all necessary, only sweet substances are required to disguise its taste. The taste for other substances, as the aromatics, is normally wanting. A peppermint lozenge or a piece of chocolate, given before a medicine will cover an objectionable taste better than anything else.

Powders may be given in milk or stirred into gruels. Children of five or six years will usually learn to take them in wafers. Tablets, pills and capsules, or their contents, must be dissolved or crushed before the child can swallow them.

Subcutaneous or intramuscular injections are made precisely as in the adult. In the infant it is best to make a subcutaneous injection over the breast and preferably in the skin over the large thoracic muscles, rather than in the extremities. Intramuscular injections are usually in the gluteal region.

Intravenous infusion is very difficult or almost impossible in infants and young children because the veins are not apparent beneath the skin and are of too small calibre to permit the entrance of the canula. For injections of small amounts of fluid one may with practice use the large veins of the scalp (temporal) and the longitudinal sinus may also be used. The method of subcutaneous infusion is described on page 111.

Among medicinal agents for external use in infancy, as applications or ointments, of purely local action, and but an indeterminable fraction of which is absorbed, concentration must be relatively high. It should be at least half as great as for the adult and may be increased according to need. The greater delicacy of the infant's skin makes greater care necessary in the use of counterirritants. This applies especially to the frequently prescribed tincture of iodine.

SPECIAL PART.

I.

DISEASES OF THE NEW-BORN

BY

H. FINKELSTEIN and L. F. MEYER,

Berlin.

REVISED BY

N. O. PEARCE, M. D.,

Assistant Professor of Pediatrics, University of Minnesota, Minneapolis.

The viability of a premature infant depends upon the degree of its development and the cause of its prematurity. While disease in the mother greatly reduces the chances of life in the prematurely born, even though carried almost to term, the number and the marked prematurity of the viable children of healthy parents is surprisingly great. It has proved possible to bring children born in the sixth month of pregnancy, weighing only 750 gms. (26 ounces), and measuring 35 cm. (14 inches) in length, to normal development. It is usually possible to maintain life in the premature infant who weighs 1200-1500 gms. (4-5 pounds), and is born in the twenty-seventh or twenty-eighth week of fetal life.

Syphilis of the parent is the most common cause of premature birth and impaired vitality. In syphilitic children serious structural changes of the visceral organs reduce the prospect of life to a minimum. Next to syphilis, such organic diseases as nephritis, diabetes, eclampsia, tuberculosis, etc., in the mother are causally important.

Many difficulties are encountered in rearing premature infants. Their relatively great body surface permits an increased loss of heat by radiation, which must be met by a larger intake of energy-producing food. This necessity is especially embarrassing, for the weak and torpid infant, lying almost lifeless and without appreciable respiration, can be roused to active nursing at the breast with great difficulty. Consequently, not a few premature babies die of starvation. A greater number, no doubt, die of disturbances of nutrition and intercurrent infections to which they can offer but feeble resistance.

1. The Care of the Premature Infant.—In the treatment of the premature child the following points must be considered. All chilling of the body-surface must be avoided, especially in the first few days of life, when, in face of the insufficiency of the temperature-regulating mechanism, it may cause marked lowering of the body temperature. The changing of clothing or diapers, the bathing and feeding must all be done rapidly. The bath should be warmer than usual, 43 ° C. (110° F.) or, better, may be omitted entirely at first. Artificial heat should be applied by means of the hot bath, as soon as possible, if the premature infant has been chilled. A persisting

subnormal temperature narrows the prospect of life. The clothing should be of the usual character. Wrapping in cotton is unnecessary.

Provision should be made first of all for continuous and uniform heat supply. The cheapest and most successful method, applied with proper care, is by the use of hot water bottles.

A large roomy clothes basket lined with flannel and in which hot water bottles, sufficient to maintain the necessary basket temperature, are placed, is a very efficient and easily prepared receptacle for the premature baby. The temperature should be maintained at between 80 and 90 degrees as indicated by the temperature of the baby. This heat may be regulated best by partially covering over the top of the basket with a light blanket. It is important that the thermometer in the basket be placed on the baby just under the outer clothing so that the temperature recorded will be approximately that of the air immediately surrounding the baby.

Four ordinary beer or mineral water bottles are filled with boiling water and after being well wrapped are laid in the crib so that there is one bottle on each side, lying parallel to the body and one at each end. Over these, heavy layers of bedding are placed. One of the bottles should be renewed every four hours, excessive heat being prevented by placing new layers of bedding around it. The commercial U-shaped containers or ordinary rubber hot water bottles are more easily handled (O. Rommel). The double-walled warming tubs of metal (Crede's tubs) which are filled every four hours with water at 50° C. (120° F.) may be used.

Any application of artificial heat requires careful handling, for injuries due to either excessive or insufficient heat occur very easily. The incubator is a more certain device and less dependent upon the intelligence of attendants. Various models are in use and some of these are very simple in construction. It is possible to improvise a satisfactory incubator from a packing case. In the most simply constructed model, the source of heat, provided by hot bottles or stones, is in a lower compartment of the box, which is divided horizontally; the air entering through several openings and rising, warming the feet of the child in the upper compartment and passing out through openings near the head.

For use in clinics, automatically regulated incubators, susceptible of very ready adjustment, are designed. To-day, in modern infants' hospitals, entire rooms well ventilated and in which a constant temperature may be maintained are reserved for the new-born. As a rule, the incubator should be regulated so that the temperature on the inside is 30° C. (86° F.). In individual cases, however, the heating must be regulated according to the degree of heat demanded by the body-temperature of the child. Therefore, the child's temperature must be taken very frequently. Over-heating is to be guarded against with as much care as is chilling; for aside from the fever, serious disturbances, (diarrhœa, convulsions, collapse), may arise as a result of excessive heat. An observing nurse will note at once, from the perspiration and the child's restlessness, that it is too warm.

With increasing age, the functional capacity of the heat regulating mechanism improves, until the child is able to maintain an equal body-tem-

perature outside of the incubator. By this time, the body-weight is usually 2200-2400 gms. (4-5 pounds). Naturally, no definite limit to the length of time for the use of the incubator can be set. External heat may be necessary for a shorter period for one child than for another. Too great dependence upon the heat-regulating ability of the premature infant frequently results in an arrest or loss of body-weight after the child has been removed from the incubator. Continuation of the external heat is still essential to its development.

Likewise, it is not possible to state accurately when the child may be taken into the open air for the first time, despite of the fact that sun, light and air are important to the infant, it is better to wait until a normal standard of gain for its age has been reached.

2. Feeding.—The premature infant has a great task to perform during its first year of life, a task which predicates vitality rather than weakness. While the normal infant only doubles his body-weight 3410 to 6714 gms. (7 to 14 pounds), by the end of the twentieth week, the premature child, according to the observations of Camerer, nearly triples his weight, 1740 to 5180 gms. (3½ to 10 pounds), in the same length of time. This he can accomplish only with sufficient nourishment; in fact, all observers have determined that such children elect to nurse very large quantities. They require about 120 to 130 calories per kilogram of body-weight (54 to 60 calories per pound), which is from one-fifth to one-third more than normal children demand. Hence, the conclusion has been reached that there is a greater need for nourishment, conforming to Rubner's law of the ratio of food requirement to the greater heat radiation resulting from the relatively large surface of the small infant. More recently, this assumption has been denied and it is asserted that the premature infant receives a greater quantity than is necessary and that his normal development can be secured with a much smaller supply.

Most excellent results are obtained in the premature infant by the early feeding of relatively small amounts of breast-milk at four hour intervals. It is especially true of small and weak prematures that they have a limited ability to digest and assimilate food and that their tolerance for food is easily destroyed by overfeeding. An adequate gain in weight will take place on amounts of breast-milk as small as from fifteen to twenty cubic centimeters given every four hours. The amount should be increased only when the weight remains stationary for forty-eight hours.

Breast feeding is more imperative for the premature infant than for any other; yet certain technical difficulties are met in the attempt at natural feeding which depend upon the limited power of the child to nurse and to swallow.

These difficulties react upon the child itself, in that it gets too little nourishment from the breast. Weak prematures often cannot be put to the breast at all and must be given the expressed milk through mouth or nose, by spoon, medicine dropper, tube or other instruments. Even stronger children may overexert themselves at the breast and become faint; and therefore are better fed in the beginning, wholly or in part, with expressed milk.

On the other hand, arises the difficulty of establishing and maintaining lactation. The stimulation of powerful suckling which serves physiologically to this end is absent or inadequate. Even though the mother is willing to nurse the infant, the secretion does not appear or appears only in small quantities and soon dries up. If a wet-nurse is employed, a congestion of milk in the mother's breast is threatened and again, on this account, the secretion may fail. The breast-pump is of some assistance; but it is better to substitute a strong healthy child at the breast until the supply increases. The wet-nurse should be allowed to nurse her own child at times, or her excess output should be expressed or pumped. It may be well for the mother and the nurse to exchange children several times a day. Under these circumstances, of course, we must be absolutely certain that syphilis is not present in either the mother, the wet-nurse or one of the children. A child with lues or suspected of lues, on account of the history of the parents, may always nurse its own mother, even when she has remained free of syphilitic manifestations. But such a child must never be put to the breast of the wet-nurse, although nothing can be said against feeding the infant with expressed milk provided by her.

Persistent demand on the breast is a most important factor in the establishment and maintenance of the breast-milk supply and even where suckling is for a time impossible, failure of the breast-milk supply is unnecessary, if the breasts are properly stimulated by correct and thorough manual expression of both breasts at least five or six times a day. The best method is to grasp the breast one or two centimeters back of the colored areola and carry the milking motion toward the nipple, but without massage. This is to be continued until the last drop is obtained. Mothers and attendants soon become very expert in draining the breasts in this manner, and the supply of milk will be made to increase rather than be allowed to decrease.

In regulating the number of feedings, we must not be influenced, as we are in the healthy child, by the fact that the physiologic food requirement of the child must be met, with due respect to the periods of waking and sleeping. Premature infants sleep almost constantly and must be aroused to be fed. The artificial feeding of premature infants should be adopted only in cases of extreme necessity; it is always a risky undertaking.

The life of such a child, possessed of so little reserve power, is seriously endangered by a disturbance of nutrition. Every diarrhœa that occurs in the first few weeks of its life should serve as an absolute indication for the interruption of artificial feeding and the substitution of breast-milk.

It is difficult to say what method of artificial feeding should be chosen in any given case. All food mixtures have occasionally given good, but far more frequently bad results. The usual milk dilutions do not give the acquired caloric value. It seems that buttermilk, with the addition of maltose and dextrin in carefully regulated quantities, has great advantages over the usual mixtures recommended. Yet, even with this food, the danger of disturbance is great.

Prognosis.—A fairly large percentage of premature infants readily overcome all of the obstacles to their development and evidence greater vitality than could be anticipated at birth. Even by the end of the first year, and certainly by the close of the second or third year, their differences from the normal child are equalized. There are peculiarities, however, which even later must be considered in relation to premature birth; such as the tendency, greater than in the full-term child, to rickets, spasmophilia and, especially to certain anemic conditions. Toward the end of the first year of premature life, if not earlier, a noticeable pallor appears, which is, at least in part, due to the insufficient deposit of iron; which, as we well know, accumulates in large store during the later months of fetal life. In this event, the early addition of iron-containing foods frequently produces favorable results. When this is unsuccessful, we have to conclude that there is a congenital insufficiency of function in the blood-forming tissues.

It has been said that idiocy, hydrocephalus, Little's disease, and other central spastic paralyses, are more common in premature children than in others. This seems to be true, but their causation is not to be charged to the fact of premature birth in itself, but rather to the conditions of disease in the parents which caused the premature birth and are also responsible for these anomalies of the nervous system. Premature children of healthy parents can hardly be said to have a poorer prognosis,[1] in regard to these complications, than children born at term.

ASPHYXIA

By asphyxia, is understood a condition produced by interference with the oxygen intake and the carbon dioxide output of the child. This interference may have developed while the child is still within the uterus, so that it is born asphyctic, or it may arise after birth.

1. Congenital Asphyxia.—It is a recognized fact that the stimulus to the first inspiration is given to the respiratory centre by an excess of carbon dioxide and an insufficiency of oxygen in the blood, as a twin result of interruption of the placental circulation. If this interruption happens before the delivery of the child's head, respiratory movements will occur too early, and may cause the asphyxia. Disturbances of the circulation between the mother and child may be occasioned by premature separation of the placenta, by compression of the umbilical cord, etc.

Symptoms.—Two types of asphyxia are clinically distinguishable. 1. The slightly asphyctic child is born with a deep bluish-red color (asphyxia livida) with slow heart action, developing slight respiratory movements or even apnœa, but of good muscular tone. Active respiration is easily produced in this child by stimulation of the skin reflexes.

2. The severely asphyctic child with a skin color resembling death-pallor, (asphyxia pallida), in whom all reflexes and all muscle tone have disappeared. A very weak heart action alone shows that life is still present.

[1] The bulging of the fontanelle, which frequently occurs in premature children during the second half year, is of no particular prognostic value. As a rule, the slight hydrocephalus, which appears in connection with the rapid growth of the brain, disappears in a few months.

The attempted respiration has ceased and cannot be awakened by the usual skin stimulation.

At autopsy the signs of suffocation are seen in congestion of the viscera, hemorrhage from the serous membranes and, as result of the premature breathing, meconium and amnionic fluid in the air passages.

Prognosis.—The prognosis is favorable in the milder form and doubtful in the severe form. Even in the latter, the seemingly impossible may sometimes be accomplished by proper methods. The claim has been made that the survivors of asphyxia are predisposed to diseases of the central nervous system, (Little's disease and idiocy), but this has not been proved. Doubtless, some inherent predisposition of the central nervous system must be present when asphyxia has such marked sequelæ.

The recent work of Rodda and others on the hemorrhagic disease of the new-born throws much light upon this subject. It is now generally recognized that severely asphyxiated infants are most likely to suffer from cerebral hemorrhage, which we believe to be largely responsible for the subsequent paralysis and mental deficiency.

Therapy.—The most efficient means of stimulating the respiration is through frequent and powerful irritation of the skin. After the aspirated material has been removed from the mouth and pharynx by means of a catheter, the new-born should be quickly bathed in warm water and rubbed thoroughly dry. If this does not produce results, the child should be plunged into cold and then immediately into warm water; a double bowl bath. These methods are usually successful in the milder form of asphyxia.

In the severer form, more energetic measures must be adopted. First of all, the Schultze's method of resuscitation, described in obstetrical works, the great value of which is generally recognized, is advised. In addition, compression of the thorax, heart-massage, at the rate of thirty strokes a minute, and rhythmic traction and release of the tongue, some forty or fifty times a minute, may be tried.

2. Postnatal Acquired Asphyxia.—When asphyxia develops after birth, it is due either to disease of the central nervous system or to some disorder of the circulatory or respiratory mechanism which interferes with the normal supply of oxygen.

Such a condition actually develops in pulmonary disease, in the white pneumonia of syphilis and enteritis; in heart lesions; in malformations, (pulmonary aplasia, diaphragmatic hernia, compression of the trachea by goitre or hyperplastic thymus); in general diseases of the new-born, (lues, sepsis); in diseases or injuries of the brain (encephalitis, congenital hydrocephalus, brain hemorrhage from birth trauma); and in weak premature infants in whom it is usually combined with pulmonary atelectasis dependant upon imperfect thoracic expansion. Such asphyxia occurs soon after birth. If a child who has developed well during the early weeks, suddenly falls subject to asphyctic attacks, the disturbance of the respiration is usually due to a grippe-like infection. In premature children and in those with severe disturbance of nutrition, spontaneous asphyctic seizures occur, the cause of which has never been definitely explained.

Symptoms.—The clinical picture differs with the disease which causes the asphyxia. The inspirations are infrequent, gasping, occur in irregular sequence or are interspersed with long pauses during which they are entirely absent. The skin is blue and cyanotic, the body-temperature is subnormal, the extremities are cold and occasionally a slight edema is seen. Fine crackling râles are heard, from time to time, over the lungs as a result of the atelectatic process.

In forms of asphyxia which develop late, symptoms are usually present, such as fever, cough, retraction of the epigastrium and intercostal spaces, which even with negative lung findings indicate a catarrhal pulmonary disease.

Diagnosis.—In the diagnosis the cause must always be looked for first, because the asphyxia itself is no more than a symptom.

Very frequently some developmental error which was not and could not be discovered during life is first seen at autopsy. In other cases, the findings are those of atelectasis, with the conditions described in the intra-uterine form of asphyxia.

Prognosis.—The prognosis depends entirely upon the cause. The anomalies named naturally give but a poor outlook. Lacking these, however, the prognosis depends largely upon the possibilities of treatment and care and even then the desired result may not be attained.

Treatment.—As in the congenital form, rapid alternation from the hot to the cold bath, friction of the skin and Schultze's method may be tried. The best method of all is the inhalation of oxygen.

In premature infants born with asphyctic tendencies, all causes of rapid cooling of the body must be avoided and special attention must be given to the supply of a sufficient quantity of food.

BIRTH TRAUMATA

External Cephalhematoma

Symptoms.—Cephalhematoma is the term applied to a hemorrhage occurring on the second or third day and occasionally later, over the parietal bone or, more rarely, over other cranial bones. Now and then it is bilateral and reaches its maximal size during the fifth or sixth day (Figure 23). The swelling rarely exceeds the size of an egg; larger tumors are exceptional. It is not sensitive to pressure. Upon palpation, a hard boundary line is felt inside of which the palpating finger seems to touch upon a soft dough-like hollow.

During the following weeks, the extravasate is gradually resorbed and the periosteum again becomes adherent to the bone and presents a thickened surface which remains for a long time. If the resorption occurs slowly, a thin plate of bone is formed over the tumor from the lifted periosteum, which is recognized upon palpation by its peculiar parchment-like rattle. The general well-being of the infant is not disturbed and the body-temperature is not influenced, excepting as a result of interference or of spontaneous infection, inflammation and pus formation.

Etiology.—The probable and generally accepted theory is that the scalp is displaced by pressure against the walls of the parturient canal during

delivery, so that the periosteum, which is not so closely adherent to the bone in the new-born as in later life, is loosened. On account of its delicate vascularity, hemorrhage between the periosteum and the bone occurs. The loosening of the periosteum at the sutures is impossible, because here the pericranium is more closely adherent to the surface of the skull, a circumstance which limits the hematoma to the site of the one cranial bone. Other attempts at explanation, such as defects of ossification, decompression of the head with a resulting hyperemia and rupture of vessels, are less plausible.

One must not fail to investigate the coagulation and bleeding time at

FIG. 23.—Cephalhematoma over left parietal bone (Gisela Children's Hospital, Munich, Prof. Ibrahim).

frequent intervals when a cephalhematoma is present as it has been repeatedly shown to be coincident with the development of general hemorrhagic disease of the new-born.

Diagnosis.—The diagnosis should give no difficulty. The inexperienced observer will occasionally be deceived by the surrounding bony wall and will mistakenly suppose that he has to do with a defect of the cranial vault with a resulting meningocele. A cephalhematoma can always be distinguished from a meningocele by its location. While the meningocele usually appears at a suture or a fontanelle, the hematoma commonly covers the arch of the bone. Pulsation of the tumor, its increase in size when the child cries, and the possibility of its replacement, are all indicative of meningocelle rather than of hematoma.

From the unimportant swelling of the scalp, the *caput succedaneum*, a diffuse edema of the soft parts which may be present even at birth and which soon disappears, the cephalhematoma is distinguished by its limitation to one cranial bone.

Prognosis.—The prognosis is good provided that infection is avoided. In the latter case, it becomes doubtful; for bone caries, meningitis and sepsis may threaten the life of the child if the pus is not immediately evacuated.

Treatment.—Usually treatment is not necessary when the process runs its natural course. Because of the danger of infection, it is best to await spontaneous recovery. Only when the resorption of the extravasate takes too long, may aseptic puncture be required. When symptoms of an infection occur, *i. e.*, redness of the skin, fever, etc., surgical interference is necessary.

Internal Cephalhematoma

In very rare cases, hemorrhage between the cranial bones and the dura mater occurs simultaneously with the external hemorrhage. This is termed internal cephalhematoma. This will occur, however, only when a fracture, from birth trauma, is present or when an external extravasate passes through the skull by way of some ossification defect in the frontal or occipital bone. Symptoms of cerebral pressure indicate the nature of the trouble. It may be distinguished from those manifestations of cerebral pressure which are caused by hemorrhage in the meninges or in the brain by the absence of blood in the fluid obtained by lumbar puncture.

The prognosis of an epidural hemorrhage is doubtful. With symptoms of cerebral pressure, the aspiration of the extravasate may be necessary after its location has been determined by exploratory puncture.

Cerebral Hemorrhage

In difficult instrumental labors and rarely in normal labors so great a compression may be produced that evidences of cerebral pressure occur. Such children are born asphyctic, the respiratory action is weak and irregular, the pulse is slow. If the asphyxia does not yield to proper treatment and if convulsions and paralyses also appear, it is improbable that the condition is caused by pressure alone and cerebral hemorrhages must be considered.

Etiology.—The trauma which causes the hemorrhage does not necessarily cause severe deformities, fissures, or fractures of the cranium. Frequently hemorrhage occurs without external injury and this even when the delivery was not particularly difficult. The cause is rather to be found in an overlapping of the cranial bones with a resulting congestion, rupture of the vessels, etc.

Recent work on hemorrhagic disease of the new-born leads us to believe that a very large percentage of cases of cerebral hemorrhage are due to this disease rather than to traumatism as indicated by the author.

Symptoms.—The clinical picture is caused by the excessive intradural pressure. Sopor, slow pulse, irregular or intermittent respiration, a bulging

of very tense fontanelle, strabismus, paralyses and especially convulsions will be observed. The deep and continuous sleep, the sopor, and the imperfect respiration, in cases in which postnatal asphyxia is increased rather than improved by the usual methods of treatment, justify the suspicion of cerebral hemorrhage.

In favorable cases, even serious cerebral symptoms disappear, after several days, without leaving a trace of a brain lesion. Quite frequently, however, epileptiform convulsions or paralyses develop earlier or later as evidences of permanent cerebral injury.

At autopsy, in those children who die with symptoms of severe asphyxia, we find subdural or subarachnoid hematoma on the convexity of the brain. Hemorrhages over the cerebellum are more rare, as are those at the base and in the ventricles. Hemorrhages into the brain substance are exceptional but probably occur more frequently than is generally believed. They are usually very minute in character and only discovered at autopsy by microscopical examination, or, in case of recovery, are either completely absorbed—giving no subsequent symptoms or are a large factor in the later development of so-called "Little's Disease."

Diagnosis.—The differentiation of hemorrhage from malformation of the brain, especially when visible injuries are absent, is very difficult. In doubtful cases lumbar puncture is advisable. Cerebrospinal fluid containing blood, with a large number of broken down or crenated red blood-cells, is evidence favoring a diagnosis of cerebral hemorrhage. Much information may be obtained from a test of the coagulation and bleeding time in cases of suspected cerebral hemorrhage. A delayed coagulation and bleeding time combined with the symptoms of cerebral hemorrhage makes the diagnosis almost certain, and points to the immediate necessity of subcutaneous injections of whole blood.

Prognosis.—The prognosis should always be made reservedly, even if all manifestations of intracranial pressure disappear within a few days. The danger of some permanent effect upon cerebral function remains. Paralyses, idiocy and epilepsy are not infrequently the late sequelæ of such a lesion. With symptoms of great pressure, the attempt to puncture and to aspirate the extravasate may be considered.

Hematoma of the Sternocleidomastoid

As a result of rupture of the fibres of the neck muscles, especially the sternomastoid, the trapezius, or the scaleni, hemorrhage into the muscular sheath occurs. Such ruptures may occur as easily in spontaneous labor as in artificial birth. An extreme rotation of the head is enough to cause such an injury to the muscle. Symptomatically we notice, soon after birth, a small swelling of about the size of a pigeon's egg on the neck. This swelling is hard, is not painful and is covered by intact skin.

The hematoma of the muscle and the scar formation which follows the absorption of the blood must be distinguished from a form of cicatrization acquired in intra-uterine life, which is probably a more common cause of wry-neck than this injury.

Therapy.—Since the hematoma is, as a rule, resorbed spontaneously within a few weeks, gentle massage and passive motion are to be advised. However, the possibility of a contracture of the muscle developing as an after-result of the hematoma with the resultant wry-neck (*caput obstipum*) should not be overlooked.

Paralyses

(*a*) **Paralysis of the Brachial Plexus.**—This form of paralysis in the newborn is the result of pressure upon or rupture of the nerve plexus in normal labor and more frequently in instrumental delivery.

It will be remembered that the brachial plexus consists of a combination of branches of the cervical nerves from the fifth to the eighth, with the first thoracic. It supplies the muscles of the shoulder, the arm and the forearm. Injury to the plexus occurs most frequently at Erb's point, which lies two to three centimeters above the clavicle. Electric stimulation at this point produces contraction of the deltoid, the triceps, the biceps, the brachioradialis, the supinators and the infraspinatus muscles. Accordingly, the paralysis is usually of the so-called Erb's type, affecting the muscle groups named, but not disturbing sensation.

Fig. 24.—Birth paralysis of the arm, upper arm type.

Symptoms. — Immediately after birth, the arm lies immovable and lax and is rotated inward. The palm of the hand is directed outward and backward. Reflex movement, by flexion of the arm cannot be elicited by pin pricks, to which the normal arm readily responds. The paralysis is especially noticeable when the child is lifted up, the paralyzed member hanging relaxed by its side (Figure 24).

A second type of birth paralysis, the so-called paralysis of the forearm, is more rare. In this form, the muscles supplied by the seventh and eighth cervical and the first thoracic nerves are affected. This involves a paralysis of the forearm, the smaller muscles of the hand, and the flexors and extensors of the fingers. It may be accompanied by disturbances of sensation. Some cases also show manifestations of Klumpke's paralysis, oculopupillary symptoms, contracted pupil, narrowed palpebral fissure and retraction of the bulb. Frequently the muscles concerned in the first type are involved also in these cases and the paralysis may be equally distributed in the arm and forearm (Figure 25).

After the paralysis has existed for some time, the muscles give the reaction of degeneration. Atrophy, flail-joint and contractures ensue.

The pathologic findings in children dying from concurrent diseases, who have come to autopsy, show hematomatas, tears, and the presence of scar-tissue in the brachial plexus.

Diagnosis.—The diagnosis of birth paralyses is difficult, especially in their differentiation from fractures and luxations occurring during delivery which are often characterized by similar disturbances of mobility. Separation of the epiphysis at the head of the humerus is especially important.

FIG. 25.—Birth paralysis, rupture of chords of cervial plexus.

FIG. 26.—Left sided unilateral birth paralysis of the facial nerve (Berlin Children's Asylum).

Paralysis and fractures occasionally occur together. Error in diagnosis may be avoided by careful palpation and by an X-ray picture of the fracture or dislocation. Changes in the contour of the shoulder, as seen from above, should lead to a suspicion of dislocation. Hyperpronation and early contractures are similarly indicative. Careful general examination and the Wassermann reaction will prevent confusion with luetic pseudoparalysis (Parrot's paralysis; see Syphilis).

Recovery usually occurs in paralysis of the arm. Complete restoration of function is very frequent and in mild cases results within a few weeks; but it must not be expected with any too great certainty. In the forearm type and in complete paralysis, the prognosis is much less favorable. If no

improvement or but partial recovery takes place by the fourth month, there is nothing more to be hoped for.

Treatment.—The treatment should attempt to prevent atrophy and contracture. At first, gentle massage and passive movements are indicated. Later, faradization and galvanization may be used two or three times a week. If paralysis remains, recent experience has shown that operative interference (suture of nerve trunks, separation of adhesions, or tendon transplantation), may be efficacious.

(*b*) **Facial Paralysis.**—Facial paralysis is frequently a result of birth injury.

The cause must be sought in trauma to the peripheral route of the facial nerve. Paralysis of the facialis is three times as common in cases of forceps delivery as in spontaneous birth, doubtless because of the direct pressure of the forceps upon the nerve. However, the pressure upon irregularities of the pelvic outlet, especially in contracted pelvis, must be considered.

The symptoms are those generally seen in facial paralysis. In the act of crying, the mouth is drawn toward the normal side (Fig. 26) and frequently the eye on the palsied side cannot be completely closed. The paralysis is commonly unilateral. In diagnosis, a congenital nerve defect or peripheral or central site must be excluded. A double paralysis is peculiarly suggestive of this cause.

The prognosis is generally favorable. In most cases the palsy disappears within a few days; or, in slower process, by the end of six weeks. Continued paralysis is rare and always arouses a suspicion of a more remote etiology.

The treatment consists of faradization with weak currents. It should be used only if the paralysis shows no improvement within two or three weeks.

DISEASES OF THE UMBILICUS

During fetal life, the umbilical cord forms the connection between the mother and the fetus. It contains, besides the remnants of embryonic organs (the yolk-stalk and the allantois), two arteries and a vein covered by the amnion and imbedded in Wharton's jelly. The anatomic structure of these channels differs from that of other vessels. The walls are thick, and well supplied with muscle fibres and elastic tissue. They contain no vasa vasorum, so important for the nutrition of the vessels themselves. The blood from the placenta passes through the umbilical vein to the body of the child. Here the vein divides into two branches, one of which passed into the portal vein, while the other, the ductus venosus arantii, opens directly into the inferior vena cava. The blood passes through the fetal circulation and is finally carried back to the placenta by the umbilical arteries which arise from the hypogastric vessels.

The circulation of the umbilical vessels stops when the first respirations occur. On account of the opening up of the lungs the umbilical blood-pressure falls, the arteries and the veins contract by means of the large amount of elastic tissue present in their walls and force out the remaining blood.

Because of the absence of the vasa vasorum and therefore of all nutritive supply, the umbilical cord dries up. This mummification cannot be considered a vital phenomenon, as it was once believed to be. It follows even when the stump is separated from the body of the child and is kept warm and dry. Warmth and moisture, on the other hand, produce decay of the stump. The physiologic drying is usually completed by the fourth day. Coincident with it, a circumscribed inflammation at the navel occurs which causes the separation of the stump, usually on the fifth or sixth day. The umbilical wound lies deeper than the walls of the abdomen, in a small funnel-shaped depression formed by the retraction of the intra-abdominal portion of the umbilical vessels. A period of about three weeks is necessary for the healing of the wound.

As results of the cessation of the blood flow, thrombi immediately form in the intra-abdominal portion of the umbilical vessels. These thrombi are gradually organized by the growth of connective tissue from the intima until the vessels are completely closed (*endarteritis obliterans*). The umbilical vein becomes the ligamentum teres extending from the umbilicus to the liver; the arteries become the lateral vesico umbilical ligaments, which pass from the navel to the urinary bladder.

Congenital Anomalies

The skin navel (*cutical navel*) is formed by the extension of the skin from the body over the cord, so that after the cord has fallen off the usual funnel-shaped depression is absent. The umbilical wound lies at the apex of a protrusion which, in most cases, is gradually retracted, but which may remain permanently.

The amnion navel, on the contrary, is caused by the spreading of the amnionic sheath of the cord which is normally connected to the skin at the base of the cord. This may spread to the size of a dollar over the abdominal surface. The mummification and falling off of the cord leaves a skin defect over this area, which usually heals by granulation. Both these anomalies are harmless and require no special treatment.

Hernia at the umbilical cord (*funicular umbilical hernia*), is more important. Its origin is to be traced to the normal umbilical hernia which persists to the second month of fetal life. A loop of intestine which should normally return to the abdominal cavity has remained enclosed within the sheath of the umbilical cord. A large tumor forms at the umbilical site. The covering of the tumor is so transparent that the contents may be seen through it. The hernia often contains other organs than the intestine.

The stomach, liver or spleen have occasionally been seen through it. Only rarely is the rupture spontaneously healed. If the condition is not treated, a fatal peritonitis may set in after the cord has separated.

Error in diagnosis is possible in the case of small ruptures only. It is especially dangerous, because where the intestine has been tied off, gangrene and ileus cannot be avoided. It is necessary, therefore, to establish the content of the umbilical hernia by careful palpation.

The treatment consists, generally, in prompt laparotomy, reposition and suture of the edges of the skin. Only in rare cases should any dependence be placed upon spontaneous healing; it has rarely been observed.

Persistence of the Omphalomesenteric Duct.—The omphalomesenteric duct, which passes from the intestine to the yolk sac, is normally obliterated by the end of the second month of fetal life. If this does not occur, a communication between the ileum and the umbilicus remains—a patent Meckel's diverticulum.

This rare anomaly prevents the healing of the umbilical wound after the cord has separated. A secretion appears and close examination reveals a fistula from which a turbid fluid exudes. This fluid, upon investigation proves, by its odor and its microscopic and chemical features, to be intestinal content. Diverticulæ also occur, which have been obliterated upon the intestinal side, but remain open at the umbilicus and which secrete small amounts of an alkalin fluid resembling intestinal juice.

The treatment consists in the surgical removal of the diverticulum.

Fistula due to persistence of the urachus. Normally the urinary bladder is developed from the urachus, while the remainder of the organ obliterates and forms the median vesical ligament. If this latter portion remains patent as it does in rare cases, a communication between the bladder and the umbilicus is established with consequent leakage of urine from the fistula at the navel. The fluid may be recognized by the presence of uric acid.

Etiologically this malformation depends upon obstruction to the passage of the urine from the bladder, by normal means such as phimosis, preputial adhesions, etc.

In the treatment, the existence of obstructions must be sought for. The fistula should be cauterized after the natural urinary passages have been opened. In some cases, the edges must be freshened and sutured.

Infections of the Umbilicus

Any delay in the separation of the umbilical stump and in the healing of the wound (see page 133) should direct our suspicion to a possible infection of the wound, complicating the physiologic process. The distinction between the normal and the diseased umbilical wound cannot be too sharply drawn. Practically, the wound should be considered infected whenever inflammation appears and the temperature rises after the stump has sloughed. The infection which is usually due to cocci, more rarely to bacilli, is localized in the stump or in the umbilical wound or may extend from either. Apparently normal healing does not predicate the absence of infection; for serious extensive infections often occur without the usual visible signs of inflammation.

According to location, we may classify the diseases of the umbilical stump, *e. g.*, irregularity of physiologic mummification, gangrene, etc.; localized inflammation of the umbilicus, as blennorrhea, ulcer, fungus, omphalitis, gangrene of the umbilicus; and finally general infections starting

from this focus as arterial thrombosis, periarteritis, venous thrombosis, and periphlebitis.

Another classification in common use is dictated by the severity of the disease. It distinguishes:

Mild Diseases of the Umbilical Wound.

1. Delayed healing.
2. Blennorrhea.
3. Ulceration and fungus.
4. Circumscribed omphalitis.
5. Gangrene of the stump.

Severe Diseases of the Umbilical Wound.

1. Umbilical gangrene.
2. Arterial and venous thrombosis.
3. Periarteritis and periphlebitis.

Infections of the umbilical wound, particularly of the more severe forms, have decreased continually during the last few years, thanks to the great progress in our knowledge of asepsis. The obstetrician has learned to use proper measures of prophylactic asepsis, especially in the care of the umbilical stump. The dry method of treatment has proved especially useful. The stump of the cord is wrapped in clean sterile linen or gauze rather than absorbent cotton, which adheres too readily, and is then held by the usual abdominal binder. All antiseptic fluids and ointments are avoided because the moist warmth delays mummification and fosters decay. The drying process is usefully hastened by means of such dusting powder as bolus alba,[1] or bismuth subgallate in small quantity. As soon as the dressing becomes moist or soiled it should be changed, great care being taken to avoid tearing the cord.

This same dry method of treatment is applicable to the umbilical wound after the cord separates. Especially is it necessary, if sloughing occurs early and there is much exudation from the wound, to avoid anything that will obstruct free drainage.

In private practice the daily bath presents no dangers but in crowded institutions, where the possibility of local infection exists, it is safer not to bathe the child until the wound is healed.

Gangrene of the Stump

Gangrene of the stump, or *sphacelus*, recognized by the strong odor of decay, the discolored appearance and the moisture of the stump of the cord, and causing resorption fever, develops only when the cord is not properly cared for and when dressing materials are used which prevent drying.

Treatment consists in the removal of the decayed tissue by means of the actual cautery. If this is done soon enough rapid healing results; if

[1] Because of the danger of the presence of tetanus bacilli in the bolus alba it must be sterilized before use. Stearate of zinc is very successfully used as a drying powder.

not, the possibility of septic infection or of toxemia from the gangrenous area threatens.

Blennorrhea

Excoriation of the Umbilicus.—Blennorrhea is that condition in which a seropurulent or purulent secretion occurs from the umbilical wound after the stump has been sloughed, the depression in the skin being slightly reddened and indolent granulations at the base of the wound being covered with a mucopurulent exudate. The flow of the secretion is obstructed by the anatomic relations, the retraction of the wound beneath the level and within the overlapping folds of the skin. In consequence and often by aid of constricting bandages, the inflammation frequently spreads to the neighboring periarterial tissue and to the newly formed thrombi in the arteries, which become involved in a purulent decomposition for some distance. In this way, something resembling a fistula is formed into which a probe may be introduced for several inches and from which large quantities of pus may be expressed.

The diagnosis depends upon the continuous flow of the secretion from the wound. It must be remembered that the mere formation of pus in the navel, even though it be extremely abundant, is insufficient basis for a diagnosis of general umbilical sepsis. It is necessary, therefore, when pus formation in the navel is associated with serious disturbance of the general health to look for other causes of the major illness.

The prognosis is favorable. The infection rarely spreads to the hypogastric arteries because of the complete destruction of the thrombi.

By way of treatment it is of first importance to provide free drainage. It must be remembered that strips of gauze and the free use of antiseptic powders rather favor the continuance of the infective process than promote free drainage and granulation. Sometimes the pus may be carefully expressed from the fistula several times daily. Occasionally, it may be necessary to open the fistula by means of incision made over a grooved director to insure proper drainage. If this is done and if the child is protected from disturbances of nutrition, the blennorrhea heals rapidly. Slight dusting with antiseptic powders may aid in the formation of granulations. The development of pyodermia around the umbilicus may be prevented by the use of ointments.

Umbilical Ulcer

Umbilical ulcer may occur primarily or in connection with blennorrhea. In this condition there is loss of tissue, varying from one-half to one and one-half inches in diameter of circular or irregular area surrounded by a sharply defined infiltrated edge, the base of the lesion being covered with pus or a thick white exudate. In some cases, it resembles the primary lesion of syphilis. There are no general symptoms, excepting fever. The prognosis is favorable, although local or general infection of the wound may arise (Fig. 27).

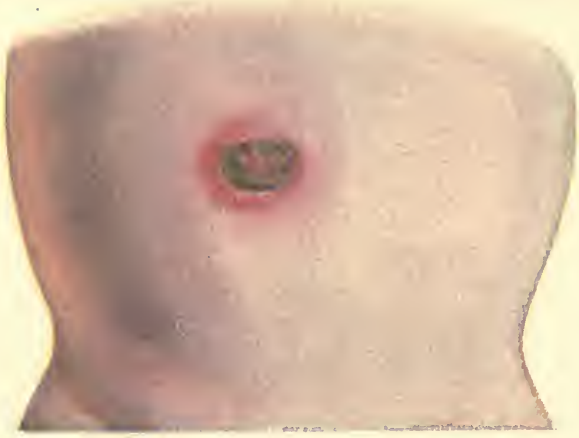


FIG. 27.—Small umbilical ulcer.

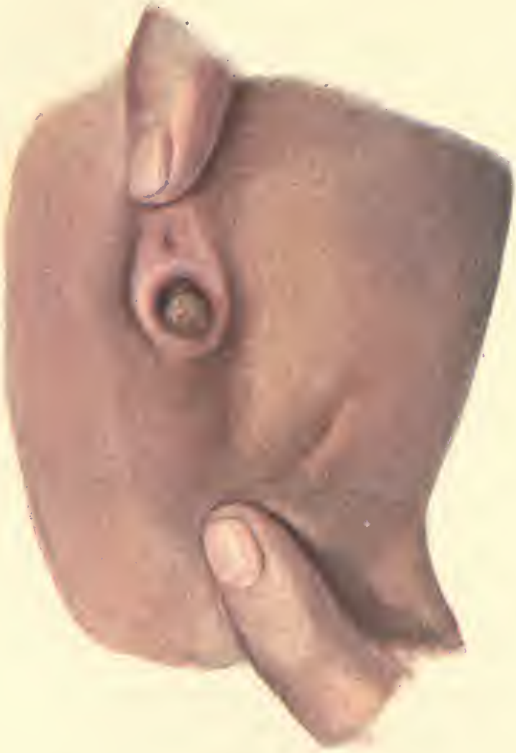


FIG 28.—Granuloma (Fungus) of the umbilicus.

The treatment consists in cleanliness, with the application of moist or dry heat, the surrounding skin being protected with petrolatum. Peroxide of hydrogen and antiseptic powders and, in some cases, the salts of mercury used in ointment or powder form give excellent results. Rapid healing is often had upon the application of a little calomel.

Diphtheria of the umbilicus appears as a flat ulceration covered by a heavy membrane, around which there is a hard and often a widespread inflammatory deposit. The diagnosis should always be confirmed by bacteriologic examination. The prognosis in cases treated early is not bad. Later cases, unrecognized, may die of cardiac complications. Antitoxin, in combination with local applications, is indicated.

Umbilical Fungus (Granuloma of the Umbilicus)

If healing is long delayed, the excessive secretion and pus formation may produce a granulomatous tumor, the granuloma or fungus of the umbilicus. This growth appears as a small red tumor the size of a pea or a hazel-nut, arising from the base of the umbilicus. At times the growth is hidden, so that it can be seen only when the apposed surfaces are forcibly separated (Fig. 28).

Besides the granulomata, enteroteratomata and adenomata rarely occur. These arise from inverted rests of the omphalomesenteric duct which have been separated by a process of constriction from their point of origin. They are to be distinguished from fungus by their smooth surface, the latter being rough or papilliform. In diagnosis it must be remembered that a protruding fistula of the urachus or a prolapse of the persisting omphalomesenteric duct may simulate a tumor. (See page 135.) A mistake of the one for the other may have serious results, since the treatment indicated for granuloma may cause perforation of the peritoneum and the bowel.

In the treatment of smaller granulomata, the application of the silver nitrate pencil or of chloracetic acid is usually sufficient to shrink the granulation tissue. Larger tumors must be ligated and cut off with scissors or removed by cauterization.

Omphalitis (Acute Inflammation of the Umbilical Ring)

Omphalitis, an inflammation of the umbilical ring and the surrounding skin and subcutaneous cellular tissue may occur secondarily to blennorrhea and ulcer, as well as primarily in the normal healing of the umbilical wound. In the latter event it arises from small fissures at the edge of the ring. The skin about the umbilicus becomes reddened and infiltrated. The abdominal walls are tense, while to avoid pain, which is increased by every movement, the diaphragmatic breathing is extremely shallow or entirely absent and the more active costal breathing is compensatory. To relieve the tension of the abdomen, the legs are flexed. The temperature rises and with the development of symptoms of more intense inflammation the general health of the patient is affected.

The prognosis is favorable if the affected area is small. In cases of extensive infiltration it must be carefully made, for there is danger that the inflammation may spread into the deeper parts and that a complicating peritonitis may ensue.

The treatment consists in the application of moist or dry heat. If an abscess forms, it must be incised at the proper time. In obstinate and wide-spreading infiltration, it may be necessary to open the infected area freely.

Gangrene of the Umbilicus

Umbilical gangrene, so frequent in bygone days is now, happily, hardly ever seen. It is usually secondary to some one of the above mentioned conditions. It may be primary in cachectic infants. Discolored, vile-smelling tissue necroses spread over large areas of the abdominal skin. In especially severe cases, the inflammatory process extends to the deeper tissues, causing peritonitis and even intestinal perforation. With fever, collapse, and extremely severe general symptoms, this disease, the prognosis of which is difficult, goes on to death. But few cases are recorded in which healing by granulation has occurred after sloughing of the phlegmonous areas.

Treatment is very unsatisfactory. Beyond the local treatment advised for gangrene, attention must be given to the conditions of feeding and nutrition in order that the patient's resistance may be raised.

Migratory Infection

A general tendency to spread and to lead to septic and pyemic infections is characteristic of this class of umbilical diseases. The general infection may be brought about by a purulent necrosis of the vessel thrombi or through the lymph channels as an extending lymphadenitis.

A migratory infection of the umbilical arteries is the more common form. In this condition, we have to deal chiefly with a periarteritis, recognized at autopsy as a seropurulent infiltration of the perivascular connective tissue. This directly causes an inflammation in the preperitoneal space and later of the peritoneum, with a consequent general peritonitis (Fig. 29). In rare cases, the process descends in the preperitoneal space and breaks externally through the inguinal ring, causing funiculitis, orchitis and phlegmon of the inguinal region, the so-called preperitoneal phlegmon. Thrombo-arteritis is more rare. It differs from periarteritis, which more often follows the normal healing of the umbilical wound, in that it arises from pus formation at the terminus of the umbilical arteries and frequently accompanies blennorrhea. It is probable that, favored by improper treatment which tends to keep the wound open, the purulent necrosis of the thrombus spreads, until it finally reaches the hypogastric arteries and thence causes a general pyemia. Arterial thrombosis and periarteritis may occur together.

In disease of the umbilical vein, lymphangitis alone is hardly ever observed, because of the slight development of the perivascular tissue. In this vessel, thrombophlebitis, or a combination of thrombo- and peri-

phlebitis is more frequent. The disease of the vein leads to peritonitis or hepatitis or both, resulting frequently in multiple abscesses.

Of the symptoms of this disease, fever, only, is first observed; the cause of which is the more obscure because the umbilical wound, in even cases of venous infection, is usually completely healed. But as soon as the peritonitis or pyemia develops, the picture is one of severe general septic

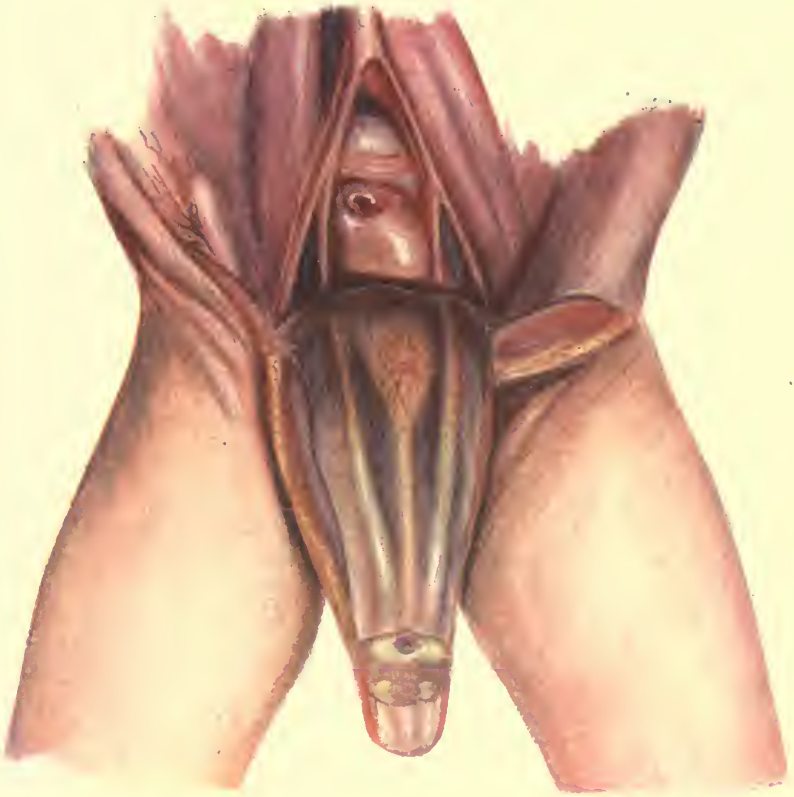


Fig. 29.—Arteritis and periarteritis of the umbilical vessels. Mucopurulent infiltration of the arterial walls and the periarterial connective tissue extending to the urinary bladder. Beginning peritonitis.

infection, often showing pyemic metastases. Icterus, as is well known, is common in venous disease. This complication, if the disease spreads slowly, may occasionally occur during the second, third or even the fourth month. Abscesses of the liver may also be postponed to so late a period.

The prognosis is bad in almost all cases. Exceptionally the purulent process may be arrested and encapsulation may ensue before the appearance of general infection.

The treatment is that generally indicated in septic infection.

Tetanus Neonatorum

Etiology.—Tetanus in the new-born is distinguished from that in the adult only by the peculiarity of its port of entry. Almost invariably the umbilical wound is responsible for the invasion of the micro-organism. The soiled hands of those who care for the child and especially hands soiled with garden earth or floor dust, the normal medium for the tetanus bacillus, carry the infection. Occasionally the materials for the dressings (*bolus alba*) may come under suspicion as carriers of the infection.

The demonstration of the bacillus is possible. For this purpose, the umbilical wound is scraped with a sharp curette and the material so obtained is injected into a mouse. The organism does not cause local inflammatory changes in the wound. If inflammation does occur, we have to deal with a mixed infection (pyogenic organisms).

The pathologic findings are those of congestion in the central nervous system. Occasionally, extensive hemorrhages are present.

The incubation period is sometimes very short, symptoms appearing

Fig. 30.—Tetanus neonatorum. Typical facial expression.

as early as the first or second day. Most cases appear by the end of the first week; but a few in the second and third week.

Symptoms.—The earliest symptom is trismus, first recognized by the difficulty in introducing the nipple. The rigidity soon spreads to the face and gradually appears in the muscles of the trunk and of the arms and legs, so that the whole body eventually becomes stiff. The child's face is characteristic; the tetanic facies, with wrinkled forehead, closed eyelids, taut cheeks, hardened by the contraction of the masseters, slightly puckered and firmly compressed lips, with the angles of the mouth drawn down producing the risus sardonicus (Fig. 30), completing the picture. In the further course of the disease, lightning-like tetanic spasms passing over the whole body like a flash, occur. In the interval, the patient lies silent, perfectly rigid, frequently in opisthotonos, with distended legs and rigidly flexed arms. With the extension of the tetanus to the respiratory muscles, disturbances of respiration and cyanosis appear. The more severe the type, the more frequent the spasms. In extreme cases rises in temperature are observed, which occasionally show most peculiar variations and may reach a stage of hyperpyrexia.

Diagnosis.—The diagnosis may always be made from the clinical picture. The demonstration of the bacillus is not necessary, since a negative result is of no value. Of course, the differentiation from similar diseases is not always easy. Tonic contractions in the new-born are also seen in such birth injuries as cerebral and meningeal hemorrhages, in encephalitis and other cerebral processes. In the latter, the involvement of the ocular musculature is a valuable point of differentiation; it is absent in tetanus.

Prognosis.—In general, the prognosis is very unfavorable; from seventy to eighty per cent. of cases die. In individual instances, the prognosis will depend upon the length of the incubation period and the severity of the infection. Long periods of incubation, relatively infrequent spasms, absence of respiratory impairment and cyanosis and of excessive temperature permit a more favorable although guarded outlook.

Treatment.—The treatment consists chiefly in measures for the relief of the convulsions. Absolute quiet and removal from all possibility of external shock is to be advised. Light moist or dry packs reduce the irritating influence of air currents and of temperature changes.

Chloral hydrate 0.5 gm. (7 grs.), at a dose, up to 3.0 gms. (45 grs.) per day has been used, in most cases in combination with the bromides, preferably calcium bromide, 1-2.0 gms. (15-30 grs.) per day in aqueous solution, or veronal, 0.075 gm. (1 gr.) per dose. These are best given by rectum. The antispasmic action of an injection of a salt of magnesium (20 c.c. of an 8 per cent. solution of magnesium sulphate), may be tried.

Great difficulty is encountered in feeding on account of the trismus. Expressed breast-milk or artificial food is to be given from a spoon, and, if necessary, through the nose. In severe cases, it is preferable to avoid repetitional excitement and to feed only three times a day through a catheter. The required quantity of water may be given in small enemata or by enteroclysis (see page 111).

Serum therapy cannot, as yet, show absolute results. One-half of a tube of antitetanic serum, containing 250 units, should be injected subcutaneously around the umbilicus and the other half into the spinal canal by lumbar puncture.

Since the above was written the value of antitetanic serum has been definitely proved. The physician is justified in giving it even before the bacteriologic diagnosis is established. Much larger quantities up to 1000 to 2000 units, may be given and repeated.

Umbilical Hemorrhage

Umbilical hemorrhage may come from the arteries, either before or after the separation of the cord, or it may come from the parenchyma (idiopathic hemorrhage). Hemorrhage from the vessels indicates a failure of their physiologic closure, while an idiopathic hemorrhage shows a local or general decrease of the physiologic coagulability of the blood or an abnormal permeability of the vessel walls.

Hemorrhage from the vessels while the cord is still attached should never be laid to unsuccessful ligation of the cord, since the physiologic sequelæ of birth, in the way of constriction of the vessels and reduction of blood-pressure through the expansion of the lungs suffice, under normal conditions, to stop the bleeding even without ligation. Hence, it is always well to search for more remote causes of umbilical hemorrhage (pulmonary atelectasis, asphyxia, heart lesions, etc.).

In the rare cases of hemorrhage which occur after separation of the cord, the formation of thrombus and the closure of the vessels by the organization of the clot are delayed by causes as yet unknown; possibly by a reduction of the coagulability of the blood incident to infection.

Parenchymatous bleeding is the result either of general sepsis, which reduces the blood coagulability or, more rarely, of local infection which prevents the formation of solid thrombi.

In the treatment of hemorrhage from the vessels of the umbilical cord it is necessary to tie off the cord with special care and for this purpose elastic bands are to be recommended. The respiration should be persistently and carefully watched. Hemorrhage after the separation of the cord is stopped by painting with epinephrin solution (1:1000), by touching with the thermocautery or by taking sutures around the navel through the abdominal walls. In parenchymatous hemorrhage all these measures are useless. In this, as in all other hemorrhage uncontrolled by such measures, the attempt must be made to overcome the bleeding by local applications and by subcutaneous injection of 10 to 20 c.c. (2-4 drams) of sterile gelatin solution. The injection of human serum, obtained as fresh as possible—a method recently used with success, may be considered.

Experience has shown that hemorrhage from the umbilicus is very frequently a manifestation of idiopathic hemorrhagic disease of the new-born. Coagulation and bleeding time should be tested in every case and when they are found to be prolonged, subcutaneous injections of whole blood should be given in the amount of 30 c.c. once or twice daily, until the hemorrhage is controlled, and the bleeding and coagulation time becomes normal.

Sepsis

While septic diseases of every grade, from the mildest to the most severe, were formerly common in institutions for children and in lying-in hospitals where they are still observed, yet, they have now, thanks to the advance of hygiene, become much less frequent.

In their etiology many forms of micro-organisms are concerned. The ordinary pyogenic bacteria, the staphylococci and the streptococci and the pneumococci, are particularly observed, but other varieties, as the bacillus coli, Friedlaender's bacillus, the bacillus of hemorrhagic septicemia, the bacillus pyocyaneus and certain strains of proteus, are occasionally among their causative factors.

The fetus may become infected, even *in utero*, by the passage of germs from the diseased placenta. A second possibility of infection before birth

occurs between the rupture of the membranes and the end of delivery. Such occasions of disease transmission are, of course, rare as compared with those which develop postpartum. After birth, the umbilicus is the chief port of entry for pathogenic organisms; but the general surface of the skin is the subject of so frequent and so numerous wounds—erosions, rhagades, intertrigo, etc., during the early days of life, that many other opportunities of infection are offered. Usually, only local disturbances, such as furuncles, abscesses and phlegmons, occur; to which, later, general septic or pyemic developments may be added. The mucous membrane may also offer a route of invasion for micro-organisms. From injuries to the buccal surfaces, usually caused by the meddlesome care of the mouth, formerly in so common vogue, stomatitis, and Bednar's aphtha may be traced and general septic disease may take origin. More frequently however, the primary septic focus is to be found in the pharynx, in the nasal mucous membrane, the conjunctiva, the tonsils, the ear, or the intestine. The possibility of general infection from the lung and from the urinary tract must also be considered.

The transmission of the infection results from numerous forms of contact (fomites, hands, utensils, lochia, etc.). The theory, occasionally advanced, that infection comes by way of the mother's or cow's milk, containing germs, is not tenable. Undoubtedly, disturbances of nutrition increase the liability to infection to a great degree by reducing the immunizing forces of the body and therefore, infective disease is more common in artificially-fed children.

The structural basis of sepsis varies widely with the numerous clinical pictures which present themselves. In the more acute cases, the autopsy findings are often very indefinite and are confined to parenchymatous degenerative lesions. In cases of longer duration, more marked visceral changes may be seen. Hemorrhages from the serosæ are especially frequent. Evidences of pneumonia, gastro-enteritis, and numerous metastatic, serous, sero-hemorrhagic or purulent foci of inflammation (embolic abscesses, septic infarcts, osteomyelitis, empyema, synovitis, etc.), are associative.

With its greatly varied etiology and with the large number of entry ports of septic infection, the disease-picture is also extremely variable, as regards alike the severity of the general toxemia and the localized disease in individual organs. However, the majority of cases are similar in so far as the symptoms of general intoxication are concerned and these are usually more marked than is common in older patients, Fever, restlessness, alternating with apathy, a tendency to collapse, and disturbances of consciousness are usually present. Disorders of the digestive tract in the form of diarrhœa, with rapid losses of weight, are almost always observed and these often appear in so pronounced a form that the sepsis runs its course mistakenly diagnosed as a gastro-intestinal disease. The similarity of the picture of severe primary dyspepsia with toxic conditions, as it develops frequently in the course of a pure disturbance of nutrition, will be readily understood. In fact, the septic process usually develops in such a way that the parenteral disease becomes the cause of severe alimentary disturbance.

Sometimes, the sepsis develops within a very brief period and the diagnosis is possible only by bacteriologic examination. In other cases, the process covers a longer time and various other symptoms may be observed.

Symptoms.—Fever is usual; high and irregular rises of temperature especially at the beginning of the disease are often followed, earlier or later, by a marked fall and collapse. At times, every rise in temperature from the very beginning is succeeded by collapse. Chills, so common in older patients, are never seen.

In the urine, albumin, casts and epithelial cells are always found as an expression of the toxic effect upon the kidneys; at times a hemorrhagic nephritis or cysto-pyelitis may develop. Enlargement of the spleen and liver is common, but is of diagnostic value only when it appears during the course of the disease. Numerous changes in the skin are indicative of toxemia. Septic erythemata, which resemble either scarlet fever, measles or erythema multiformi, occur. Hemorrhages, in the form of small petechiæ or large ecchymoses, are characteristic and of diagnostic significance. The color of the skin is at times grayish, as in anilin poisoning. Often the skin and mucous membranes become yellow because of a complicating icterus.

It is customary to distinguish a hemorrhagic, a gastro-intestinal and a pneumonic form of sepsis according to the prevalence of certain groups of symptoms.

A true pyemia characterized by metastatic abscesses is not very common in the new-born, nor at any period of childhood. Probably the low degree of resistance at this age does not favor the slow course of the disease necessary to abscess formation. Nevertheless, it does occur now and then and runs a course showing special tendency to metastases in the skin and to embolic abscesses in the internal organs, to pyemia of the joints or to osteomyelitis. Endocarditis and pericarditis are comparatively rare.

Buhl's and Winckel's disease are to be regarded as forms of sepsis having a peculiar course. Their etiology has not been explained. The Buhl type produces a fatty degeneration of the heart, liver and kidney resembling that of phosphorus poisoning, together with hemorrhages in various organs and in the skin. The children affected by these very rare diseases are born asphyctic and severe manifestations of disease soon appear, such as diarrhœa, loss of weight, hemorrhage and sopor. Most cases die and death occurs early with extreme cyanosis and icterus.

The chief symptoms of Winckel's disease are hemoglobinemia and hemoglobinuria. The disease runs its course with cyanosis, icterus, dyspnœa and gastro-intestinal manifestations and usually results in death in a very short time.

The diagnosis is based upon the recognition of a port of entry, upon the existence of metastatic foci, which may, however, be hard to demonstrate, and upon the appearance of petechiæ, icterus, etc. The chief difficulty lies in its differentiation from alimentary intoxication, a distinction which is all the more important because parenteral infections in the young infant tend

to cause secondary disturbances of nutrition. In alimentary cases, the effect of dietetic treatment upon the fever, the toxic symptoms and the diarrhœa make the primary cause clear. The bacteriologic examination of the blood cannot be undertaken outside of the hospital, because it is not always possible to obtain the required amount of blood, which should be, at least, 2 c.c. (½ dram).

The prognosis is always grave. The more abrupt the onset and the more rapid the course of the septic process, the less prospect is there of keeping the patient alive. In the pyemia of gradual development and slow course the hope of successfully combating the disease is better justified. Occasionally, cases in which hope is apparently lost may recover if nutrition is well sustained.

Prophylaxis.—The experience of hospitals and infant homes has taught us that much may be accomplished in combating sepsis by proper prophylactic measures. It has been shown, first of all, as already noted, that the sources of infection may be reduced and the number of entry ports for infective organisms may be decreased by proper care of the skin and the umbilicus, by frequent changing and by the avoidance of meddlesome cleansing of the mouth. In the home, also, it is necessary to guard the new-born infant with the greatest possible cleanliness and asepsis.

Special care must be taken when the mother has a puerperal infection. In this event, it is better that the mother and the child should be cared for by different persons. If this is impossible, the child's toilet should always be attended to before the mother's, in order to protect it from contamination. This is advisable even when the puerperal mother is well, because even the normal lochia may contain pathogenic organisms. On the other hand, the danger that threatens the child from the milk of a puerperally diseased mother is much overrated. It has never been demonstrated that septic infection is introduced in this way. In cases where there is any anxiety on this score the expressed milk may be fed. Not even the milk of a woman suffering with mastitis seems to be dangerous to the infant, in so far, at least, as the defensive measures of the intact mucous membrane of the mouth are concerned.

Treatment.—In the treatment of sepsis, suitable feeding is the first requirement; since it has been shown beyond a doubt that the formation of immune bodies is favored in the greatest degree by proper diet, while improper feeding definitely lessens immunity. It is often possible to maintain the bodily strength of the child at so high a stage with mother's milk that the infection may be overcome. In fact, those methods of feeding which determine the general well-being of the infant in health are similarly beneficial in sepsis, so that the mother's milk is the main reliance. Particularly in those cases in which intestinal disturbance become prominent much may be expected from proper feeding. They should be treated by the identical methods that obtain in similar conditions arising from alimentary causes. (Compare Disturbances of Nutrition.) Local pus formation must be treated according to surgical requirements. Strong antiseptics, especially phenol and iodoform, are to be avoided.

For the rest, it may be necessary to stimulate the heart by the use of camphorated oil (7 minims, subcutaneously, several times a day[2]); or with citrated caffein or caffein sodio-salicylate 4 c.c. (1 dram) of a 0.5 to 1 per cent. solution, four or five times a day, or with a solution of epinephrin (1:1000), 7-15 minims. In high fever, tepid baths are useful. If there is a tendency to collapse, hot baths, carefully applied may be employed. Neither collargol, by enema or in form of an ointment, nor antistrepticoccic serum have given any definite results.

MELENA NEONATORUM

The passage of large quantities of pure blood from the gastro-intestinal tract has customarily been called melena. The blood is voided either by vomiting or by stool. A form of pseudo-melena (*melena spuria*), occurs, in which the vomited blood is of different origin. It may arise from an epistaxis or be derived from a wound in the nipple of the nurse. In true melena the gastro-intestinal tract of the patient is the direct source of the hemorrhage.

Under this proper designation, however, a symptomatic melena and a true melena may be distinguished. Symptomatic melena is a result or manifestation of some other disease. It sometimes occurs in diseases of the liver (lues), or in other processes causing severe congestion. The most common causes, however, are septic and septo-hemorrhagic infections, in which the melena occurs only as one form of other and multiple hemorrhages. In many cases, such infection is introduced by specific hemorrhage producing organisms.

The origin of true melena (*melena vera*) is still wholly obscure. Nothing more is known than that the blood usually comes from small ulcers in the stomach or duodenum and, more rarely, in other parts of the tract. But it may be possible that even these are absent and that in autopsy only hemorrhage from the mucous membranes or a simple hyperemia is found. Nothing definite can be said about the mode of origin of these ulcers. The once accepted theory that they are caused by embolisms from thrombi of the umbilical veins, does not seem plausible. At present, thrombosis of small intestinal vessels with consequent hemorrhage and resulting tissue digestion is considered more probable. This thrombosis may be caused by a vasomotor-ischemia. The significance of concurrent disturbances of nutrition may be understood from the frequent occurrence of duodenal ulcers in severe atrophic conditions (decomposition). The theory is also advanced that conditions of central nervous irritation may cause hemorrhages from the intestinal mucosa and that the hemorrhagic areas are then converted into ulcers by action of the digestive fluids.

The hemorrhages usually begin by the second to the fourth day, although occasionally they appear earlier or later. Unimportant, at first, they soon reach the stage in which tarry stools are passed, or clotted masses of blood

[2] Recently, objections have been raised against the use of camphor in severe general infections.

vomited. The weakness and anemia resulting depend upon the severity and duration of the hemorrhage. Some children bleed to death rapidly; in others, the condition extends even to the second week. Of the untreated cases, more than one-half die.

The diagnosis must determine whether it is a false or symptomatic melena, or a true melena. This is all important to the prognosis. The symptomatic form and especially the hemorrhages dependent upon sepsis have a very unfavorable prognosis. A prolonged bleeding and coagulation time are practically always present in true melena.

Treatment.—By way of treatment subcutaneous injections of gelatin may be given and repeated if necessary. The gelatin may be obtained free from tetanus and completely sterile in ampules, 10-25 c.c. (2-6 drams). Many cases have been saved by this method. The subcutaneous injection of 10 to 20 c.c. (2½-5 drams) of serum, preferably human, has been recommended. The horse serum may be substituted if the human cannot be obtained. In urgent cases, whole blood may be used instead of serum. Recent experiences show that 30 c.c. of blood taken from the mother by means of the ordinary Luer syringe and injected subcutaneously is very efficient. The syringe may be lined with sterile petrolatum or the blood may be citrated (sodium citrate 2 per cent.) but these precautions are not essential. The injection may be repeated daily, or more often if necessary. All other measures (ice, liquor ferri sesqui-chlorate, epinephrin, etc.), are uncertain. Upon occasion, the usual treatment for collapse and, in certain cases, the transfusion of salt solution, must be considered.

ERYSIPELAS

Erysipelas of the new-born does not differ etiologically from the disease at any later age, but is distinguished from it by its increased tendency to spread and its resulting malignancy. It usually arises from either the umbilicus or the genital organs and begins at the end of the first or second week.

It begins with redness and edematous swelling of the affected part, which increases in diameter and spreads rapidly. High fever develops and in many cases alternates with collapse temperatures. The general health of the child is severely affected. At times, skin necroses, phlegmonous, gangrenous and general septic processes develop from the erysipelas as a base.

In the diagnosis, the differentiation from phlegmon must be especially considered. The prognosis is very unfavorable in the new-born; certainly much more unfavorable than it is in somewhat older infants, to say nothing of children of more advanced years.

The treatment is that generally used in this disease; (ichthyol ointment, alcohol, mercuric bichloride, 1:1000, aluminum acetate, etc., externally). Applications of saturated solution of magnesium sulphate are of great value. Special attention must be given to the maintenance of the general quality of resistance by the feeding of mother's milk and of the strength of the heart, in particular, by means of stimulants.

OPHTHALMIA NEONATORUM

Severe purulent catarrh of the conjunctiva is frequent in the new-born; partly because of a special predisposition upon the part of the mucous membranes of early life, and partly because the passage of the child through the infected birth canal affords especially favorable opportunities for conjunctival infection. In fact, the majority of children are infected with the disease during labor if the mother is suffering from any form of purulent vaginitis. The number who acquire the disease later, by some other form of contact, is small.

Etiology.—The gonococcus is usually considered the most common agent in the production of blennorrhea. The so-called inclusion or chlamydozoa blennorrhea is almost equally frequent, however, with the gonococci form. Clinically it has a great resemblance to the latter, but in general pursues a milder course. By staining smears according to the Giemsa method, the inclusions of epithelium as described by Halberstaedter and Prowazek, may be demonstrated in large numbers, while gonococci are absent. Pneumococcic conjunctivitis, the catarrhal infection produced by the Koch-Weeks bacillus and other forms are known.

Symptoms of the gonorrheal form, which is the most severe, appear by the second or third day in the form of a reddening and edema of the lids, while a thin sero-hemorrhagic secretion appears between them. The eyes can be opened with difficulty, the lids are tense and the conjunctiva chemotic.

In severe cases, the lids may become so tense that danger of gangrene arises, and the conjunctiva shows a membranous exudate. After several days, the swelling goes down and large quantities of pus are secreted while the connective tissue appears slightly granular.

The duration of uncomplicated cases is usually from six to eight weeks, when recovery sets in. The danger of keratitis with perforation always threatens. It is a generally accepted fact that about one-third of all blindness is due to blennorrhea. Gonorrheal arthritis, synovitis, dermal abscesses and exanthemata are among the complications of the local infection.

The course of blennorrhea due to micro-organisms other than the gonococcus is usually shorter and its result more favorable; but damage to the cornea is not always avoided.

The value of Crede's prophylactic method of instilling one drop of a 1 per cent. silver nitrate solution, or some less irritating silver salt, in each eye is recognized. The catarrhal conjunctivitis which the silver may cause may be disregarded in view of the great benefits of the method.

Treatment.—The treatment, during the stage of swelling, consists of measures to reduce the inflammation. Applications of ice are frequently recommended, but the extreme cold may be injurious at this stage. No damage will be done by dry or moist heat. In addition, irrigation with warm antiseptic solutions (boric acid solution, mercuric bichloride, 1:5000), may be useful. After pus formation is fully established, the physician himself should touch the averted lids daily with a 1 to 2 per cent. solution of silver nitrate, after which the eyes should be carefully washed out

with physiologic salt solution. Several drops of a two per cent. solution of protargol or argyrol (freshly prepared without heating) may be instilled into the eye in place of the silver nitrate. In all methods of treatment, injury to the cornea must be very carefully guarded. If only one eye is affected, the sound eye should be washed daily with a half of one per cent. solution of silver nitrate and may be covered with a protection bandage. Involvement of the cornea should be treated according to ophthalmologic methods. The articular metastases will heal, without opening the joint, with applications of heat or of Bier's hyperemia, or, at most, by puncture. In obstinate cases, vaccine treatment may be tried.

SWELLING OF THE MAMMARY GLAND AND MASTITIS

In quite a number of new-born infants of both sexes, swelling of the mammary gland begins two or three days after birth. It increases until the middle of the second week and then gradually disappears. During this period, the so-called "witch's-milk," which very closely resembles colostrum, may be expressed from the gland, so that we have to deal with an actual secretory function similar to that of the maternal breast. The probable cause of this phenomenon is to be found in the lactogen, a substance which, according to the latest researches, is produced by the ovaries and the uterus and which in gravid puerperal women stimulates the production of milk. This agent passes by way of the placenta into the circulation of the child and acts specifically upon the infant breast. Upon the invitation of this physiologic activity, infection may occur and mastitis may develop, especially if, in line with an old and objectional popular practice, the fluid is expressed. The turgescence further increases, fever, redness and abscess formation or phlegmon ensue; from which source, occasionally, a general infection may arise. The prognosis of mastitis, however, is generally good.

The treatment consists primarily in measures to reduce the inflammation, followed later, if necessary, by incision. Radial incision should be carefully done to avoid severing the ducts.

ICTERUS NEONATORUM

Jaundice occurs in more than eighty per cent. of all new-born infants, from the second to the fifth day. It appears first in the face and then descends over the body. The sclera are affected much later. Other manifestations of disease are absent. The feces are colored with bile. The urine is light in color and is negative to the usual tests for bile pigment. Microscopically, however, brownish-yellow masses of bilirubin, either free or in epithelial or hyaline casts ("masses jaunes"), are found (Fig. 31). The jaundice lasts from a few days to three weeks; in exceptional cases only, and especially in premature infants, is it present for a longer time. The whole phenomenon is quite unimportant and is considered a physiologic one.

No anatomic lesion is found in infants with jaundice who die from other causes. Bilirubin crystals may be demonstrated in the tissues.

Of the cause of icterus neonatorum, nothing definite is yet known. The older theories which attempted to show that it was of hematogenous origin and caused directly by the resorption of the products of a catabolism of the hemoglobin derived from extravasated or broken-down red blood-cells, independently of any action of the liver, have been disproved by the demonstration of bile acids in the urine and in the tissues, the presence of which can be due alone to a resorption of bile. Quincke's theory to the effect that the bile massed in the meconium passes directly into the blood through the patent ductus venosus arantii has, also, many arguments against it. The theory of a passing anomaly of liver function, on account of which the bile not only passes into the bile capillaries, but is, in part, also secreted into the

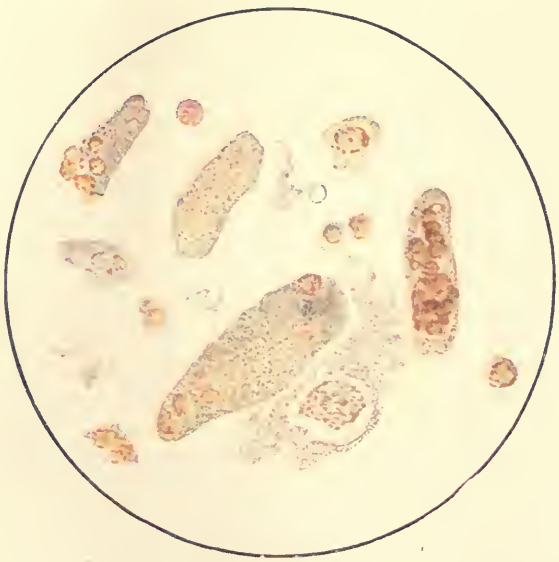


Fig 31.—Urinary sediment in icterus neonatorum (Berlin Children's Asylum).

blood, is a more probable one. Obstruction, causing the flow of the bile in the wrong direction, has been attributed to increased pressure in the bile capillary system, secondary to polycholia or to an excessive viscosity of the bile. But this theory, also, has to be abandoned, when Hirsch and Ylppo were able to show that during the last months of fetal life an increase in the production of bile pigment and its presence in the blood occurs normally, a condition which persists for several days after birth. Accordingly, icterus neonatorum may be considered an actually physiologic phenomenon. Icterus of the skin always occurs when the bile pigment content of the blood passes a certain definite limit. The explanation of this increase of the bilirubin during the first few days of life still remains a problem.

Other forms of icterus also occur in the new-born, which are all the more important because they are more serious. In all these forms bile pigments

may be found dissolved in the urine and examination of the urine is, therefore, very important in the diagnosis. The most common of these forms of jaundice is *septic icterus*. Infectious forms are seen in hospitals and are, at times, epidemic. They produce slight fever and gastro-intestinal symptoms, and in some cases, are fatal. They probably depend upon an enterogenous invasion of bacilli into the bile passages ("maladie bronzee"). Congenital atresias of the large bile ducts are evidenced, during the first days of life, by the jaundice that they cause.

Habitual icterus gravis of the new-born is a rare and puzzling condition. It affects several or all the children of a family. It runs its course with severe general manifestations and, later, develops cholemic symptoms. Usually, it has a fatal termination. At autopsy, internal hemorrhages, small serous exudates in the body cavities, swelling of the spleen and liver, together with marked icteric discoloration of the brain nuclei, are found.

EDEMA AND SCLEREDEMA OF THE NEW-BORN

Mild forms of edema occur so much more commonly in the feeble new-born, and especially in prematurely born infants, than they do in older children, that the term edema neonatorum has been bestowed upon them. Such edema is not associated with nephritis, but resembles in its course the idiopathic edema which is not infrequently seen in cachectic children, during the first and second years of life, and is dependent upon the retention and non-excretion of water which is, in turn, related to disturbances of salt metabolism. It is apt to be most severe when the diet contains much salt and, especially, sodium salts. While this idiosyncrasy comes, in the older child, in the path of disturbances of nutrition and as a consequence of infection, in the new-born and prematurely born it represents a congenital functional insufficiency. It is possible to build up the infant by proper care and feeding, the edema disappears spontaneously.

Scleredema.—The hard swelling of scleredema which cannot be easily indented and, in extreme degree, may cause a stiffness of the limbs, is in direct contrast to the soft doughy swelling of edema. The skin, in this form of disease, has a death-like pallor or is mottled and characteristically cold. The general body temperature is far below normal. At autopsy, the subcutaneous tissue and the skin are saturated with a yellowish fluid, very rich in protein, and possessing the characteristics of serum, rather than of the lymph of simple edema. The skin changes usually begin in the legs and in severe cases spread over the entire body, leaving only the scrotum, the knuckles and the eyelids unaffected. The disease is always accompanied by evidences of extreme weakness, such as apathy, somnolence, thready pulse, superficial and irregular breathing, and anorexia. The prognosis, usually unfavorable, is dependent upon the severity of these general symptoms; although it is occasionally possible to save even apparently hopeless cases by an improvement of their general nutritive condition.

Schlerema.—The disease must be differentiated from fatty sclerema, in which the swelling cannot be indented and depends upon a hardening

of the subcutaneous fat. As a rule, fever is present in this disease. At autopsy, no fluid appears upon section.

The cause of scleredema has not been, as yet, fully explained. The most commonly discussed hypotheses are those on the one hand, which look upon the condition as a septic infection, complicated by vascular changes; and, on the other hand, as an idiopathic edema to which is added a hardening of the subcutaneous fat, ordinarily occurring after death, but which in this case is due to a lowered metabolism and to the extreme cooling of the body. In view of the peculiar consistency of the exudate, a condition related to a coagulative edema, resulting from injury to the vessels because of the extremely low temperature, must also be thought of.

The treatment consists of the application of external heat by means of the incubator, or other similar apparatus (see page 122). Heat may be applied more rapidly by a hot bath. All further therapy must be addressed to the restoration of the reduced strength. Feeding with mother's milk is indispensable.

ALBUMINURIA

Albuminuria (*proteinuria*), is observed so frequently in the new-born that it has been called "physiologic." The quantity of protein is always small. The greatest amount is present from the first to the third day. Traces are often present until the second week and occasionally later. The urinary sediment contains epithelia, leucocytes, and abundant urates.

In spite of much discussion, the cause of the albuminuria, is not definitely known. It is generally looked upon as incident to the sudden changes in the infant metabolism and circulation in the transition from intra- to extra-uterine life. Probably the physiologic congestion arising in the vascular system, during birth, causes proteinuria, as it does in the analogous condition of orthostatic albuminuria.

URIC ACID INFARCTS

The peculiar change of the kidney in which yellowish-red stripes are seen arising from the papillæ, passing into the pyramids and disappearing in the cortex, is called uric acid infarct. Microscopically, urates are found imbedded in the kidney substance. Doubtless there is an increased secretion of uric acid in the new-born. This is most probably due to the decomposition of large numbers of nuclein-bearing cells (leucocytes). Why the uric acid should be deposited in the form of infarcts is still an unsolved problem.

Gradually, during the first two weeks of life, the infarct is dissolved without injury to the infant and gives the urine its characteristic sediment, a reddish-yellow, finely granular mass which stains the diaper red and is seen under the microscope to be made up of casts covered with urates, fine urate deposits and epithelium.

VAGINAL HEMORRHAGE

In rare cases, hemorrhage from the vagina, of slight degree, occurs in new-born infants during the first few days of life. The blood comes from

the uterus, as may be readily seen with an ear speculum. No other symptom or disturbance of the general health appears.

Only recently has a knowledge of the cause of this phenomenon been gained. It is probably incident to a physiologic congestion, resulting in subepithelial hemorrhages of the uterus like the process of menstruation; and like it, caused by a specific internal secretion which circulates in the maternal blood and occasionally passes into the blood of the child in sufficient quantity to become active.

In its differential diagnosis, malignant new growths and septic diseases must be taken into consideration. Precocious menstruation does not need to be considered, because it hardly ever occurs in the first few days of life.

II.

PATHOLOGICAL CHANGES OF THE BLOOD AND BLOOD-FORMING ORGANS

CONSTITUTIONAL ANOMALIES AND DISEASES OF METABOLISM

BY

M. von PFAUNDLER,
Munich.

REVISED BY

M. D. OTT, M. D.,
Associate in Pediatrics, University of Minnesota.

INTRODUCTION

Physiology and Pathology of the Blood

Human blood-cells, as shown in the table formulated by Schridde (Figs. 32 and 33) belong to two different groups or families. Those especially interested are referred to the excellent article by the above named author in *Aschoff's Text-book of Pathological Anatomy (5th Edition)*.

1. The Myelotic Parenchyma.—In embryos of less than 10-12 cm. in length only the primary erythroblasts are found in the blood-spaces. Only at a later date do the other derivatives of the cells of the vessel walls appear and then, extravascularly, only in the liver but after the second month of fetal life also in the spleen and bone-marrow, the latter gradually monopolizing the formation of these cells. From the blood forming parenchyma, the white cells enter the blood stream by their own activity, while the red corpuscles are forced in by the rupture of the centre when the cells have matured. The platelets are derived from the giant cells.

2. The Lymphatic or "Non-granular" Parenchyma.—The cells of the walls of the lymph-vessels, together with the germinal centres of the lymph nodes form the lymphatic parenchyma which appears later than the "granular parenchyma." The genealogy of the large mononuclear leucocytes is still in dispute.

At birth the greater portion of the myelotic parenchyma is confined to the grayish-red, functionally-active bone-marrow and the lymphatic parenchyma is found in the lymph nodes, the spleen, and other lymphoid tissues of the body. In the long bones this functionally active marrow later changes into inactive, yellow, fatty and gelatinous marrow. Under pathologic conditions a return to the phase of widely diffused blood-forming parenchyma is seen, accompanied by the appearance of atypical forms of cells. Retrograde changes of an embryonic character, especially in the direction of a reversion to a gelatinous bone-marrow, is also observed. Both of these tendencies carry with them stimuli of varying nature and intensity

Even the ordinarily adequate, regulative stimulus, incident to the normal breaking down of the blood elements, may become pathologic in degree if this degeneration is marked. Furthermore, the development of endogenous and ectogenous poisons, the nature of which is not fully understood, must be

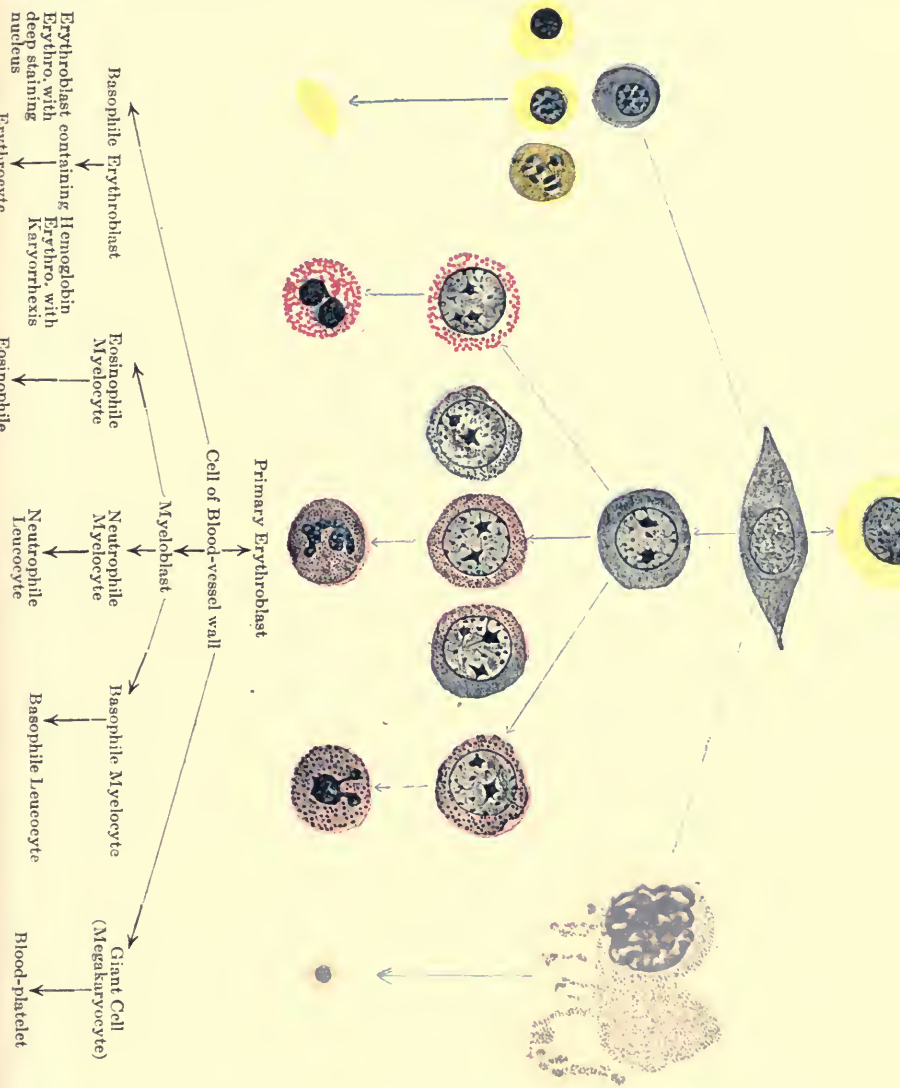


Fig. 32.—Myeloid tissue (From Schridde. The Blood-forming Organs in *Aschoff's Text-book of Pathologic Anatomy*).

considered productive of non-physiologic stimuli. The return to the embryonic type of blood formation, that is, an activation of the myelotic blood-forming foci outside of the bone-marrow, occurs more readily in children than in adults.

The Blood in Childhood.—The most important peculiarities of the child's blood, as compared with that of the adult, are given in the following table:

Cells per Cm. of Blood	Reds (Million)	Whites (Thousands)	Percentage of various forms of whites				Hemoglobin (Sahli)
			Polymorphonuclear		Large Mononuclear	Lymphocytes	
			Neutro	Eosino			
New-born	5–7	20–32	70	2	8	19	110–130
Infant	4.1–5.2	8–13	27–36	2–7	9–15	50–55	60–80
Adult	4.5–5.0	6–8	71	3	4	22	100

The following facts may be added by way of histologic description:

Erythroblasts, nucleated red cells, are frequently found during the first few days after birth and occasionally even later during the first half-year

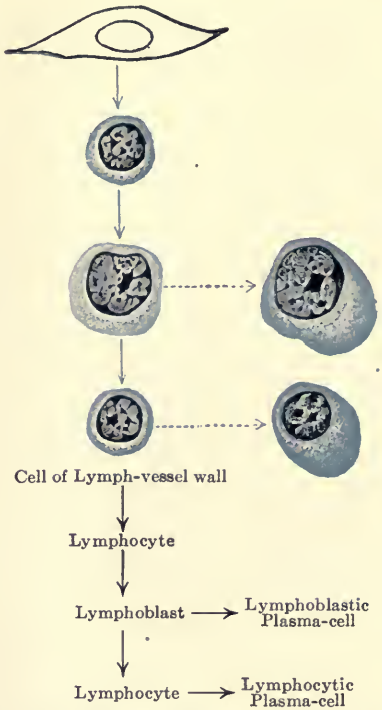


Fig. 33.—Lymphatic tissue (representing stain with Azur II-Eosin). (From Schridde in *Aschoff's Text-book of Pathologic Anatomy.*)

in healthy infants. An excess of the non-granular over the granular types of leucocytes persists until the fifth year. At this age the proportions existing in the adult are rapidly approached. Large forms are remarkably frequent among the non-granular cells in childhood. During the first few weeks an occasional myelocyte is found in the normal blood. Immature cells are frequently found in the blood of normal infants during the first weeks of life; as, in the event of any disturbance of hematopoiesis, they occur more commonly in the blood stream of children than in that of later years.

Hemodynamics.—The pathologic phenomena attendant upon diseases of the blood are much better understood than formerly as a result of recent discoveries and, particularly, of those of Plesch.

The functional ability of each organ, as of the body in general, is first of all dependent upon its oxygen supply, which represents the most important function of the blood and the circulation. According to Plesch, the oxidation process in the tissues is the main and the only indication of the vigor of the circulation. If the oxygen requirement is increased as a result of enlarged functional activity, as, for instance, of the musculature in bodily exercise, this increased demand may be satisfied by an increase of the volume of blood per minute.[1] This result may be attained by increase either of the force or the rate of the heart beat, or by increased velocity of the blood flow. The demand may otherwise be met by more complete oxygenation of the blood, by the increased oxygen-carrying power of the blood, by improved pulmonary aeration, or by the more complete interchange of gases between the blood and the tissues. These factors are very closely interdependent and the laws which govern these many-sided functions frequently find expression in the symptomatology of the diseases of the blood and circulatory apparatus. If an injury alters one of these factors, compensatory changes in the other factors usually appear. If, for example, the oxygen capacity of the blood is reduced as a result of a loss of hemoglobin, the volume of blood per minute will increase. When the blood volume is increased by added force of the heart-beat, resulting in complete systolic emptying of the heart and greater heart capacity, palpitation, anemic dilatation and hypertrophy will arise. If the increase is brought about by added frequency of heart action, an anemic tachycardia will develop; while increased velocity will result in hemic murmur ("bruit de diable"). The oxygen demand of the anemic patient is proportional to the increased amount of respiratory and circulatory work required to compensate the low hemoglobin. E. Mueller's studies have confirmed in many cases the application of the hemodynamic laws operative in the adult to the child.

A. GROUP OF ANEMIAS

General Symptomatology

The conditions grouped under this head are very often secondary or associated manifestations of other disturbances. They are characterized, clinically, by a reduction in the total quantity of blood, by an absolute or relative decrease of the erythrocytes or of hemoglobin, with habitual pallor of the skin and mucous-membranes, and by compensatory and hemodynamic adjustments resulting from them. The reduction of the hemoglobin index which indicates an oligochromemia, that is, a diminution of the total amount of hemoglobin in the blood, serves at present as

[1] By volume per minute is meant the amount of blood which enters one chamber of the heart in one minute, or which passes through the total area of the greater or lesser circulation.

a criterion which may be readily applied. It is a misleading one, however, when the volume of the blood is changed as in hydremia or in over-concentration. The total quantity of blood and the total quantity of hemoglobin can be determined in the living in only exceptional cases.

Even though the common term anemia suggests a reduction of the total volume of blood, or at least a diminution of the essential constituents of the entire blood, nevertheless it must be borne in mind, in dealing with the individual case, that it is not merely a question of the quantity of the blood, but rather one of its functional capacity. The term "thin-blooded" more nearly covers the facts of such functional poverty. It is clear that even though it be diminished in quantity, the blood may be functionally efficient as an agent of circulation and interchange, because it can move more rapidly and thus increase its specific activity. In such cases we speak of a compensated anemia. Attention has been called, under the head of hemodynamics, to the various mechanisms of these very frequent adjustments. Frequently compensation is established by the fact that the anemic blood, in itself, has the power to stimulate the formation of red blood-corpuscles. This stimulus naturally operates in those regions in which the myelotic parenchyma persists after birth, *viz.*, in the functionally active bone-marrow. But, under certain circumstances, it may become active in those organs which do not participate in the erythropoietic function in postnatal life, as, for instance, in the lymph nodes, the spleen, and the liver, in which the myelotic parenchyma may be newly formed. Not only in location, but also in method, such a compensatory erythropoiesis may or may not be analogous to the normal developmental process. A postembryonic type of compensatory erythropoiesis may usually, in fact, be distinguished from the embryonic form. In the latter type, the immature cells, as megalocytes and megaloblasts, which physiologically are found only in the blood of the embryo, at once appear in the blood stream. In the postembryonic phase, however, this occurs only in extreme cases and as a final effort in the blood-forming function of the bone-marrow. Since an actual transition to the embryonic type is probably not determined by qualitative, but rather by quantitative conditions, and is further influenced by age, in childhood at least, it will hardly serve in itself to differentiate the several forms of anemia. The erythropoietic stimuli, activated by anemia, will not exercise any noticeable compensatory influence if the requirements for successful erythropoietic activity are wanting from the beginning.

Pathologic changes in the erythropoietic tissues in anemia cannot always be determined by the methods in use today.

Every systematic classification of the anemias of childhood meets with objections in the present status of our knowledge of the subject. The didactic and practical purpose of this work, however, makes such a classification necessary. Such presentation of the nature of these diseases as our present knowledge will permit will give the thoughtful physician basis for a logical therapy.

I. ANEMIA DUE TO PRIMARY INTERFERENCE WITH ERYTHROPOIESIS.

This form of anemia is characterized by an insufficiency in the blood-forming organs incident either to hypoplasia of the matrix, external conditions unfavorable to its normal activity, or to a lack of raw materials. Its clinical characteristics are chiefly of a negative sort. The signs and symptoms of increased destruction of the erythrocytes in the blood are not evident, nor are there indications of either an excessive, an atypical, or an incomplete erythropoiesis. Actual changes in the total quantity of the blood, oligemia, hydremia, oligocytosis, oligochromemia and, occasionally, polychromasia, and the more marked variations in the size of the red cells, are alone met with.

1. Congenital or Early Acquired Anergic and Aplastic Anemias.—In this group, which has been little studied, insufficiency of erythropoiesis is the result of anomalies in the formation or function of the bone-marrow.

Hypoplastic marrow is not uncommon in anemic infants, nor is a fatty marrow in the long bones of children even in the first two years. The latter finding is considered to be the basis of the anemia seen in congenital myxedema and symptomatically related conditions.

Anemias appearing early in cases of infantilism, with hypoplasia of the genitalia and the circulatory system, in which the blood findings so much resemble those of chlorosis that they have been erroneously regarded as habitual or chronic chloroses, probably belong in this group. Benjamin has recently reported a peculiar type of anergic anemia, with hypoplastic habitus and mental weakness, in which no acquired injuries could be demonstrated and in which both therapeutic results and anatomic findings in the bone-marrow were negative. In this case, probably, the anemia may be considered the result of a congenital functional weakness of the hematopoietic mechanism. Similar anomalies of development, complicating such cases, are found in other organs.

2. Alimentary Anemia (in the restricted sense).—Experimental starvation and states of inanition, resulting, either from stenoses along the alimentary tract or from internal causes, do not produce an anemia but rather an atrophy of the blood which parallels that of the remainder of the body. In young rapidly growing animals, on the other hand, a diet poor in iron impairs erythropoiesis through a deficiency in the material for the synthesis of hemoglobin. Von Hoesslin and others have observed that children who have been fed exclusively upon milk[2] or other foods deficient in iron become anemic after the initial store in the liver has been exhausted. This is especially true in the case of premature infants, twins, and children born of anemic mothers. This so-called chlorotic anemia of the new-born described by the French authors, better termed *oligosideremia*, offers a good prognosis and responds quickly to the administration of iron. That not all premature infants develop this anemia can be explained by the fact that the initial store of iron is quite a variable quantity even in full term infants.

[2] Whole cow's milk contains 0.3-0.7 milligram Fe_2O_2 and human milk 1.5-2 milligrams per litre.

As in other forms of anemia, the constitutional factor must be considered in the pathogenesis of alimentary anemia without, however, invalidating the meaning of the term. Children of this type have a higher iron requirement than normal children, as Schwartz and Rosenthal have recently demonstrated that they have a negative iron balance. There are, of course, other factors to be considered, but these are discussed elsewhere. Even in older children who, from some psychic or other causes, subsist solely on milk may show a reduction of the color index without other appreciable blood changes. The observations of M. B. Schmidt that in pregnant animals an iron poor diet retards the growth of the offspring is noteworthy in this respect.

The form of alimentary anemia described by Czerny and his followers belongs in another category. According to their conception, a high milk diet produces in certain constitutionally predisposed children actual changes in the blood and blood-forming organs through the action of the milk fat, particularly the fatty acids. Whether these substances affect the blood through a withdrawal of alkalies or through some hemolytic action (Kleinschmidt), these anemias should be classified with those of toxic origin. This view is supported by the symptomatology of the severe cases as well as to their failure to respond to ordinary iron medication.

3. The So-called Anemia of Poverty; Tenement Anemia; School Anemia; Incubator Anemia.—It is certain that children reared in city slums, that is, in overcrowded, poorly ventilated, ill-lighted, cold and damp dwellings, who are taken into the open but rarely, and who with poor food find only an ineffective stimulus to physical exercise, not only appear pale and weak, but often suffer from actual anemia. From the further observation that all the objective symptoms of such an anemia disappear after a few weeks or, at the most, a few months of living and exercise in the open, either in the country, the mountains, or at the sea-side, we may conclude that some one of these many injurious elements, or their combination, has had an unfavorable influence upon the formation of red blood-corpuscles. These children, almost without exception, become rickitic during the first-half of the third year and are infected with tuberculosis by the tenth year. These forms of anemia do not, however, appear to be dependent upon these very common diseases or upon any disease of other organs or systems.

To this group should be added the anemia of debilitated infants who are treated for too long a time in the incubator, a form which reacts very favorably to fresh air treatment.

The attempt has been made several times, but without definite results up to the present time, to gain a clearer knowledge, by means of natural or artificial experiments on human beings, animals and plants, of the particular form of injury which produces anemia and of its method of action. It is true that the absence of light causes a loss of chlorophyll, which is the analogue of hemoglobin. In a purely experimental way, however, relative darkness, as experienced in the polar night, in mining operations, or in animals kept in covered cages, does not cause any reduction of hemoglobin in adult human beings or animals, or at least no relative oligochromemia

or oligocytosis. Just the reverse might rather be expected from a reduction of the oxygen tension and the poor decarbonization of the blood. It is possible that the red blood-cell formation of the growing organism responds more acutely to such injuries and especially to the absence of actinic and related stimuli (Schoenenberger). The increased circulation in the musculature and in the skin during exercise in the open, and especially in moving air, with direct or indirect daylight, and the resultant breaking down of the erythrocytes, probably begets erythropoietic stimuli, which are never absent during the developmental period, especially in constitutionally weak children, without resulting injury.

In these anemics a rather mild degree of anisocytosis, and oligochromemia, moderate oligocytosis and, occasionally, polychromatophilia, combined with the general symptoms of anemia or chlorosis, such as headache, sleeplessness, anorexia, general weakness, palpitation of the heart, listlessness, etc., are observed. A special tendency to obstipation, indicanuria, intermittent albuminuria, and vomiting is also to be noted. Increased area of heart dulness and accidental murmurs are almost the rule in school anemias. There is no fever and the prognosis is favorable.

4. Chlorosis, or Green Sickness, and Pseudochlorotic Conditions.—These disturbances almost always begin during or immediately after puberty. They are not really children's diseases and, therefore, they will not be fully considered here. Nevertheless, chlorosis maybe properly included in the symptomatic grouping of anemias, since while many cases have a normal hemaglobin content and exhibit certain signs and symptoms common in chlorosis, that are not due to anemia (Morawitz), yet the absence of all evidences of an increased breaking down of the red blood-cells shows that there is an insufficiency of erythropoiesis and that the chief factor in the causation of the anemia is usually a limitation upon the production of new hemoglobin.

Nature and Etiology.—The characteristic blood findings of chlorotic anemia are confined to a reduction of the coloring matter of the blood, that is, to an oligochromemia, without a corresponding oligocytosis. It has been shown, also, that the total quantity of blood, as indicated by the total number of erythrocytes, where the red cell count is normal, is not reduced but usually increased. This increase may amount to almost double the normal quantity, so that the blood represents ten per cent. of the body-weight instead of about five per cent. It is permissible, therefore, to speak of a chlorotic plethora. The production of hemoglobin does not seem able to keep up with this increase of blood. The cause of the plethora is in doubt. Whether it represents a pathologic survival of the blood proportions, ranging from six to eight per cent. of the body-weight, which exist before puberty; whether it is dependent upon a tendency to high water retention in the chlorotic, or whether it may be regarded as an attempt at compensation of the reduced oxygen capacity of the blood, is not determined. Another viewpoint from which the nature of chlorosis may be considered is suggested by the fact that the disease occurs almost exclusively in females

and at puberty, and that supposedly it has a subsequent effect upon the generative organs.

Symptoms.—In a majority of cases the only definite blood changes are oligochromemia, anisocytosis, polychromatophilia, slight oligocytosis, general pallor, reduction of the color index of the red cells, diminution of the specific gravity of the blood, increased coagulability, and a decrease of the blood-platelets. The subjective symptoms are sleepiness, lassitude, migraine-like headache, dizziness, shortness of breath, palpitation of the heart, fainting spells, side-stitch, epigastric pain, chills, blinking, tinnitus, dyspepsia, anorexia and parorexia, dysmenorrhea, and leucorrhœa. The objective symptoms consist chiefly in a greenish or alabaster pallor, in slight edema and the formation of venous thromboses, both of which are known to occur in the mesentery; in increased area of heart dulness, an exaggerated cardiac impulse, accidental heart murmurs, venous bruit, and softness and frequency of the pulse; in tachypnœa, gastroptosis, moderate hyperchlorhydria, and occasionally enlargement or descent of the spleen. No characteristic changes of metabolism are known.

Diagnosis.—The blood findings are pathognomonic. Certain negative findings and the reduction of the color index are important in differential diagnosis. Pseudo-anemia, is often mistaken for chlorosis. Secondary anemia of toxic origin or following hemorrhage may also cause confusion.

II. ANEMIAS DUE TO LOSS OF BLOOD

In this class of cases, in addition to the blood findings of the first group incident to a relative insufficiency of erythropoiesis, the evidences of stimulation, in the presence of normoblasts, of poikilocytosis and leucocytosis, are found.

Post-hemorrhagic anemia, especially in traumatic cases, is a condition the etiology of which is considerably more clear than in other groups of anemias and in its purely experimental pathology is very instructive. Immediately after an external or internal hemorrhage has occurred, an actual diminution of the total quantity of blood, that is, a true oligemia is always found. In individual cases this is rapidly compensated, but in cases where hemorrhage is frequent or habitual it persists, and the proportion of blood per kilo of body-weight may be reduced from 60 c.c. to about 20 c.c. The first attempt at repair after a hemorrhage is an increase of the volume of the remaining blood by withdrawal of plasma from the tissues. This serves to refill the vascular system and to restore in a degree the blood-pressure. It results in hydremia, oligochromemia, oligocytosis, with normal color index, and to a certain extent in a swelling of the red blood-corpuscles. Later, a restitution of the blood-cells occurs. The loss of blood gives an intense stimulus to the bone-marrow which, if it is not already exhausted, results in putting into circulation stored up mature cells, erythrocytes, and neutrophilic leucocytes alike; and also in the liberation of immature forms, among them cells poor in hemoglobin, nucleated polychromatic cells, basophilic granular erythrocytes, mononuclear leucocytes and myelocytes. In a word, a blood crisis obtains. Secondly, it responds in an active hyperplasia

of the marrow tissue and in a rapid and excessive development of erythrocytes and granular leucocytes. The formation of hemoglobin is a matter of longer delay. A relative leucocytosis appears, although the loss of leucocytes is less than that of the erythrocytes, because the former are more uncommon in the rapid axial stream. A decrease of the color index characterizes this stage of post-hemorrhagic anemia. The regeneration, as a rule, follows the type of a postembryonic erythropoiesis. One exception to this, suggested by the writers' experience and by experimental hemorrhagic anemia in animals, is found in severe umbilical hemorrhage of the new-born. The coagulability of the blood increases during and after the hemorrhage. Lipemia and fatty infiltration of the tissues may occur.

Causatively, aside from the trauma, the so-called hemorrhagic diatheses, Barlow's disease, melena, umbilical hemorrhage, epistaxis, rectal polyposis, certain intestinal parasites, and the hemorrhagic nephritides must be considered.

Symptomatically, large and rapid loss of blood causes faintness, pallor, general weakness and sometimes convulsions. Repeated small hemorrhages may produce cachectic conditions. Hemorrhages are more dangerous in children than in adults. The rapid loss of from one-fourth to one-third of the total quantity of blood, that is to the extent of two per cent. of the body-weight is dangerous to life.

III. HEMOCYTOLYTIC AND MYELOPATHIC ANEMIAS

In this, the largest group of anemias, we have to deal with acquired conditions, which affect, first, the mature blood in the circulatory system, and, second, the blood-forming organs. A strict classification of cases, dependent upon the point of attack of the agent of injury, is neither practically possible nor theoretically justified. For when a hemic poison enters the circulation it not only prejudices the mature circulating cells, but it is likely to extend its influence to the blood-forming parenchyma. Thus a combination of myelogenous and hematogenous injuries is formed. Chemical poisons, such as lead, which have a recognized specific toxicity to the erythrocytes, have been shown, also, to damage the bone-marrow. Further, signs of blood degeneration, by way of the too early, as well as the increased break-down of the circulating blood-cells, are particularly noticeable whenever weak cells appear in the circulation as the result of a myelogenous injury. Finally, it may be supposed that a hematoplastic stimulus, such as a marked degeneration of the mature cells would give to the blood-forming organs, after it has exceeded certain limits, must become pathologic and harmful. It is a hopeless task, indeed, to search the pathologic blood for criteria which will make a definite differentiation between myelogenous and hematogenous anemia possible. Basophilic granulation of the erythrocytes, poikilocytosis, crenation, hemosiderosis, or the deposit in the tissues of increased blood-pigment from broken down cells, and the evidences of reversion to the embryonic type of erythropoiesis, are all important characteristics of the forms of anemia under consideration. They do

not, however, invite conclusions as to the localization of the injury, whether in the mature blood or in the immature cells.

Theoretically, a large part of the anemias of this class may be considered, in the broadest sense, as *toxogenous anemias*; that is, they are caused by more or less well recognized ectogenous or endogenous blood poisons. In the light of their action two classes of these may be distinguished.

(*a*) Hemocytolytic poisons are those which cause the intravascular dissolution or disintegration of a large number of red blood-cells, with certain definite results. The coloring matter passes into the plasma, a hemoglobinemia. A part of it is changed in the liver into bilirubin and urobilin; another part is stored in the liver, spleen, and bone-marrow as hemosiderin; and some is excreted by the kidneys, giving a hemoglobinuria or methemoglobinuria. The erythrocytic débris is taken care of in the spleen, giving a spodogenous enlargement of the organ, and in the blood by the phagocytes. This cleaning up process does not always take place without disturbance, by way of destruction of the leucocytes, intravascular clotting, so-called hematogenous icterus, anuria, etc.

In children the ectogenous substances which are to be considered in this relation are certain sera, the primary toxic action of which is to be distinguished from anaphylaxis; snake venom, the poison of bee stings, and various vegetable poisons, such as aspidium, the several members of the species of solanum, and mushrooms. Among the endogenous hemolytic poisons are the poison arising from burns, the biliary constituents which enter the blood, causing cholemia, and particularly the microbic toxins of sepsis, Winckel's disease, malaria, typhoid and scarlet fever.

Paroxysmal hemoglobinuria is a peculiar disease caused by endogenous hemolytic substances in the nature of autolytic amboceptors. These amboceptors are peculiar in that they attach themselves to the erythrocytes, both *in vivo* and *in vitro*, only at low temperatures, when the natural complement of the blood, which is liable in these subjects to unusual variations, completes the chain and permits the hemolysis. This explains the occurrence of repetitional attacks of the disease. These attacks are brought on by exposure to cold. They begin their course with chills, cyanosis, pain in the back, hemoglobinuria, oligocytosis and relative leucocytosis; and they occasionally go on to embolism with gangrene. They usually last from one to two hours and are followed by jaundice, enlargement of the spleen, and urobilinogenuria. The repeated appearance of these abnormal amboceptors may be closely related to a lessening of the resistance of the red blood-cells. In almost all of the children so affected there is a question of the existence of some congenital or parasyphilitic poison.

Hemocytolysis, with the formation of methemoglobin, occurs with many medicinal agents, among which are potassium chlorate, the phenol derivatives, as acetphenitidin, lactophenin, phenocoll, and phenolphthalein; the anilin derivatives, antifebrin and acetanilid, and pyrogallol. Chemical changes in the hemoglobin, involving its oxygen content, are caused by carbon monoxide, hydrogen sulphide, and hydrocyanic acid.

(*b*) Other hemic poisons may cause early destruction of the erythrocytes without involving their solution in the blood to any great extent. These injurious agents doubtless act, in part, through the liver, spleen, and bone-marrow upon immature forms of cells. Hemosiderosis, the phagocytosis of red cells, and the excretion of urobilin and urobilinogen are observed in these cases. Of the ectogenous poisons, lead, mercury and arsenic belong in this class. Among the endogenous poisons of similar action are those excreted from the body-substance of such animal parasites as tenia, bothriocephalus, ascarides, anchylostomata, and trichocephalus, as well as lipoid substances arising from degenerated body tissues. To these may be added the hypothetical toxins of acute intestinal disease and the nephroses, and, finally, the bacterial poisons and the reaction products they excite in latent as well as manifest tuberculosis and syphilis.

It was thought, because normally erythrocytes are destroyed in the spleen, that hypertrophy of this organ led to an increased destruction of these cells. It is probable, however, that the hemolytic activity of the spleen pulp is confined to those erythrocytes which originally were or have become defective. According to Frank, the reticular endothelial cells of the spleen and lymph nodes elaborate a hormone which is inhibitory to the bone-marrow, and which in case of hypertrophy of the spleen is capable of producing anemia.

The pathologic effects of the action of such poisons upon the blood and the bone-marrow are shown, in part, in retrograde changes—as the atrophy of the marrow in tuberculosis and chronic nephritis, in part by chronic inflammatory changes and by progressive nutritive disorders, by way of hypertrophies and new tissue formation in the active bone-marrow. These new growths may revert in type to the most primitive marrow germ cells, which are really in the nature of capillary endothelium. This metaplastic formation may be no longer confined to the bone, but may appear in widely diverse parts of the body. It is most common in the liver, spleen, and lymph nodes. The stimulation of these reserves may repair the blood injury. This is certainly true in the most common forms.

1. The ordinary mild type of chronic toxogenous anemia includes the so-called secondary or complicating anemias accompanying tuberculosis, lues, and the chronic nephroses.

The clinical picture consists essentially of the symptoms recited and of those of anemia in general, described under chlorosis and post-hemorrhagic anemia.

The prognosis and course depend upon the extent of the injury done by the toxic agent and upon its recognition, avoidance, and removal.

The pathologic effects of the action of such poisons upon the blood and the bone-marrow are shown, in part, in retrograde changes, precisely as the appearance of diffuse metaplastic foci of blood-forming parenchyma, already described, resembles the embryonic hematopoiesis.

2. *Biermer* or *pernicious type* of anemia is recognized clinically by the appearance of large numbers of megalocytes and megaloblasts. At times, these giant erythrocytes are so numerous that the reduced hemoglobin

content of the ordinary red cells is over-compensated and the color index runs higher than one.

The exhaustion of the myeloid parenchyma may be recognized by leucopenia, that is by polynuclear leucopenia, relative lymphocytosis, extreme oligochromemia and oligocytosis, poikilocytosis, anisocytosis and a reduction of the protein content of the blood-serum which is of a deep yellow color. The marked loss of coagulability which underlies a hemorrhagic diathesis, indicated by hemorrhages of the mucous membranes, skin, and retina, is a fact of great importance.

Another condition very rare during childhood and because of that probably often overlooked is the:

3. *A regenerative* (or aplastic) *type* of severe anemia. This type of anemia may occur in children of school age, rarely before and pursues a progressive and rapidly fatal course extending only over a period of a few months. There is a very definite hemorrhagic diathesis but usually little impairment of the nutrition. While the clinical picture resembles that of pernicious anemia, the blood picture is entirely different. There is a complete and constant absence of all signs of blood regeneration such as erythroblastosis, megalocytosis, anisocytosis and polychoromatophilia in spite of a profound reduction in the hemoglobin and the number of erythrocytes and platelets. Furthermore, there are no findings on examination of the liver, spleen and lymph nodes, and the urine is normal.

From the rarity of the pernicious type of anemia in childhood and from certain peculiarities of this disease, such as enlargement of the spleen in younger individuals, it may be inferred that the blood and the bone-marrow of small children react differently from those of the adult to these poisons. This fact has been determined by the experiments of Reckzeh and others, and by observation of members of the same family with reference to the effects of essentially similar blood injuries in individuals of different ages.

4. The *v. Jaksch-Hayem form* of disease, which is closely related to the leucanemic type, seems to be a more frequent form of reaction in the youthful organism.

V. Jaksch and Hayem contemporaneously described, clinically, this fairly definite form of disease, occuring quite frequently in children of one-half to two years, under the name of pseudoleucemic infantile anemia. It resembles Biermer's anemia in that it also presents evidence of intense stimulation of the blood-forming organs, associated with the signs of a breaking down of the blood-cells and hemosiderosis. As in Biermer's anemia, too, the stimulation leads to the extension of the erythroplastic parenchyma of the bone-marrow to new formations of intra- and extravascular hemopoietic foci in the liver, spleen, lymph nodes, and kidneys. Qualitative reversion of erythropoiesis to the embryonic type also occurs. It differs from the pernicious form, however, in the fact that the myeloid foci in the liver, and more particularly in the spleen, develop to so great an extent that the size of these organs is largely increased and an intensive leucopoietic activity, marked by added numbers of white cells in circulation, takes place.

The symptoms may be anticipated from the foregoing description. The examination of the blood usually shows oligocytosis, with from one to three million red cells, and oligochromemia. The reduction of the hemoglobin may be proportionately in excess of the diminution of the number of the red cells —a normocytotic regenerative type with reduced color index—or it may be relatively less. The latter condition obtains when the regeneration of megalocytes is a prominent feature; that is, when these and the megaloblasts appear in the circulation, the color index often ranging to one hundred or more. Poikilocytosis, polychromasia, basophilic granulation, and indications of the breaking down of cell nucleii are characteristic. An associated

Fig. 34.—Sixteen-month-old boy and fourteen-month-old girl with rickets and Jaksch's anemia. Both children recovered after several weeks of treatment.

leucocytosis of varying degree and type may, or may not, be present. The number of leucocytes is supposedly indicative of the severity of the disease and is usually about 20,000. At times and particularly in inflammatory conditions the granular forms predominate, but usually the non-granular are the more numerous. The eosinophilic cells and sometimes the myelocytes are increased. In some cases the large number of mononuclear cells, running up to twenty per cent, is a very noticeable feature.

The rest of the clinical picture of the disease is that of rickets accompanying the high grade anemia. A dull yellow pallor, a general listlessness, and a wilted appearance are very apparent. The persistence of a certain amount of cutaneous adipose tissue gives a pasty look. Numerous small hemorrhages into the skin and mucous membranes occur. The enlarged spleen, hard and with a sharp edge, but not tender, always extends below

the costal margin and may frequently reach to the level of the umbilicus or even lower, greatly distending the abdominal wall. There is marked swelling of the liver, which is soft and also has a sharp, thin edge. There is slight enlargement of all the lymph nodes. Accidental heart murmurs are rare. The pulse is full and soft.

Occurrence, Course and Prognosis.—Jaksch-Hayem's anemia begins between the seventh and the sixteenth months of life. It is almost entirely confined to artificially-fed children and especially to those of the poorer districts of large cities. Gradually, and at times rapidly, it reaches a severe degree. It persists for months, but rarely until the third year or later, after which spontaneous recovery takes place in a certain per cent. of cases. No other form of anemia which, judging by the general symptoms and the blood picture, is equally severe offers so good chances of favorable termination. Rickets is responsible for most of its fatal complications, in the form of intestinal disturbance, broncho-pneumonia, and muscular degenerations. Hydrops and hemorrhages occur but rarely.

Etiology.—From the clinical viewpoint, tuberculosis, syphilis, typhus, other infectious diseases, intestinal parasites, chronic disturbances of nutrition, and poisoning are occasionally given as etiologic factors; but rickets evidently stands in much closer relationship to Jaksch-Hayem's anemia than any of these. Since rickitic skeletal changes may be demonstrated at autopsy in almost every child of this age who succumbs (see page 196), the combination of this disease with pseudoleucemic anemia is not necessarily astonishing. So associated, rickets commonly attains at least a medium intensity and usually a severe degree. It remains clinically manifest and florid until the second or third year.

Recently Aschenheim and Benjamin, as well as Marfan, have attempted to clear up the relationship between the two diseases.

While these views are very worthy of consideration, the writer can not commit himself to them without reserve so long as the knowledge of the etiology of rickets itself is no further advanced. For the present Naegeli's view must suffice for the systematic classification of Jaksch's anemia. According to this view, the disease represents a variety of secondary anemia arising from peculiar biologic conditions in the early stages of infantile development. The primary causes are found in repeated infections and faulty nutrition during the first years of life. On the one hand, it appears that rickets may arise as a sequel of these injuries and, on the other hand, Jaksch's anemia may result from them in that critical and important period of development, at the close of the first year, when there is a tendency to germinal disease.

5. *The "Alimentary Anemia" of Czerny.*—As previously indicated, Czerny believes that in certain constitutionally predisposed individuals an abnormal metabolism results in the elaboration of acid products from milk (also from starches) which are toxic to the blood-forming parenchyma.

To distinguish it from the ordinary form of alimentary anemia previously described and which results from a deficiency in blood-building material, the term trophotoxic anemia would probably be preferable. The

clinical picture of this form of anemia as given by Kleinschmidt is so variable as to make a description of the condition difficult. Some of the children were thin, others fat, some had a yellowish coloration of the skin, others were very white, some had small, others large hemorrhages into the skin. Edema was occasionally encountered. Enlargement of the liver and spleen was often noted, though there was only slight enlargement of the lymph nodes. A reduction in the hemoglobin, usually 30-45 per cent. and a less marked oligocytosis were the only constant blood findings. In contrast to these mild pseudochlorotic cases were the more severe ones with oligocytosis, poikilocytosis, polychromatophilia and erythroblastosis. There was usually a lymphocytic leucocytosis of 15-18,000. Evidences of a marked activity of the bone-marrow, occasionally hemosiderosis, and a fatty degeneration of the heart were the pathological findings.

The sole criterion for the nosological identity and the alimentary origin of this protean condition is the favorable reaction to dietary measures. Whatever benefit is obtained from this must, however, be an indirect one, as the same diet is employed in those constitutional disorders most often associated with alimentary anemia. (Rickets, exudative diathesis, lymphatism and "milchnahrschaden".) Czerny's alimentary anemia especially in its more severe form may be included in v. Jaksch-Hayem's anemia which is better defined. To discard the latter and substitute the former, as some desire, would not be advisable at least so long as the origin of alimentary anemia is hypothetical.

6. *A group of rare diseases* in which the erythropoietic system is chiefly affected, caused also by toxogenous injuries, operating even in the embryo, is observed in childhood. Symptomatically, at least, the diseases of this group are closely related, as is shown by the following common changes which include the general manifestations of anemia, oligocytosis, or more rarely polycytosis, oligochromemia, embryonic erythropoiesis, with the occurrence of megaloblasts. They present, also, an increased destruction of red blood-cells, with its consequences in hemosiderosis, pigmentation of the skin, enlargement of the liver, jaundice, the excretion of bilirubin, urobilin and urobilinogen; early spodogenous enlargement of the spleen, and variations in the leucocyte count, as in Jaksch' anemia. Leucopenia, relative lymphocytosis, and finally a hemorrhagic diathesis may be found.

One of the types included in this group, described as chronic familial hemolytic icterus, suggests the characteristics of paroxysmal hemoglobinuria on account of its familial and early appearance, the demonstrable reduction of the resistance of the red blood-cells, which are broken down particularly after exposure to cold, its paroxysmal character, and its favorable course; and, finally, its relationship to lues and other toxic infections. Autolytic hemolyses, however, have not been found in this condition.

The cases belonging to the Banti-Senator symptom-complex appear at a later period of life, and usually after the fifth year. They follow a chronic course and terminate fatally in spite of treatment. Ascites, icterus, enlargement, followed by contraction, of the liver, commonly appear late. In the final stages of the disease fibrosis, an enormous spleen, and cirrhosis of the

liver are found. The author has never seen an actual case of Banti's Disease going on to recovery, with a negative nitrogen balance, after the operative removal of the spleen as the primary seat of the disease. Nor has he been able to establish, in the cases which have come under his observation, the alleged fact that this symptom-complex always belongs to late syphilis. A very significant sign of disease in these cases, and often the first to be noted, is a profuse, repeated, and even fatal hemorrhage from the gastro-intestinal tract. When in doubt this condition should be treated specifically.

In Gaucher's splenomegaly the early enlargement of the spleen to enormous size is the most important feature. Its course is similar to that of Banti's Disease and it is looked upon as a peculiar disease of the lympho-erythropoietic system.

Attention should be called to the curative effect of splenectomy in the last two types.

The Treatment of Anemia

In those forms of toxic anemia in which the poison is known and its source recognized, a successful therapy aims at the removal of the cause. Examples are found in parasitic or luetic anemias.

The therapy is quite clear in those cases in which there is a deficiency of certain hygienic factors. Exercise in the open air, stimulation of the skin, the influence of sunlight, etc., are indicated. Treatment is self-evident when there is an actual poverty of iron in the food. This, as already suggested, is a very rare condition and the iron supply required should be prescribed from the garden and not from the pharmacy. The natural combinations of iron in food-stuffs are absorbed more completely and in more acceptable form than is the iron of the customary preparations (Krasnogorski). Most of the green vegetables, cereals, legumes, potatoes and fruits have sufficient iron content; they are taken without objection and are comparatively cheap. It is not necessary to be guided by tables as to their iron content, since an excess of iron is readily obtainable. If the child refuses vegetables it is either a question of serious defect in training or of overfeeding with eggs and milk. In that event the quantity of these items may be reduced, or they may be entirely eliminated from the menu in order to force the child to accept vegetables. Both milk and eggs may be readily dispensed with during the second year and may be omitted, if necessary, at the end of the first year. They would appear to have an unfavorable influence upon the results of a vegetable diet. When carefully prepared, mashed vegetables, especially spinach and carrots cooked in their own juices, are usually borne very well at this age. The appearance of green or red stools, containing undigested particles of these vegetables, is no contraindication.

The diet prescribed by Czerny in alimentary anemia is as follows:—

First Meal.—100 c.c. milk diluted with an equal amount of cereal water, a little softened zwieback or bread with a little butter or preferably marmalade.

Second Meal.—Fresh fruit with biscuits, zwiebach or a little bread and butter.

Third Meal.—Broth thickened with cereals, rice, oat or potato gruel. In addition purée of vegetables, meat, also liver (minced, 1-2 teaspoonfuls).

Fourth Meal.—The same as No. 3, or in the case of older children, bread and butter with meat or sausage and fruit in addition.

It may be possible in the anergic-aplastic and trophotoxic forms of anemia to stimulate the hemopoietic function, and especially a new formation of hemoglobin in unknown foci, by means of artificial stimulating substances. Such a stimulating property has been ascribed to iron. It cannot be tested by animal experiments because these forms of anemia cannot be produced artificially. Experiences gained at the bedside, showing that medicinal iron may be depended upon to a certain extent in diseases coming under the group of chlorosis, and probably as well in chronic post-hemorrhagic anemias,[3] which are in some respects similar, would seem to indicate that the action of iron is related to these especial conditions.

For chlorosis, iron is prescribed in the form of pillulæ ferri carbonatis (Blaud's pills) 1 to 3 after each meal. In Czerny's anemia Feer, contrary to the views of the former, recommends ferrum reductum (0.05-0.1 gram, 2 to 3 times daily) in addition to a limitation of the amount of milk. Of numerous other preparations including the ferruginous mineral waters none are as useful as Blaud's pills. Proprietary synthetic preparations of organic iron and the wines and liquors are, to put it mildly, not indispensable. Because of their low iron content very large doses would be required to obtain the same effect. When assimulation is sought, the natural foods suffice, and the so-called stimulating effects are obtained from the simple and inexpensive preparations of inorganic iron.

Another method of treatment is to take advantage of the adequate natural erythropoietic stimulation. These natural stimuli are loss of blood and increased destruction of red blood-cells. The former is secured by blood-letting; the latter usually by the use of arsenic in Fowler's solution (*liquor potassii arsenitis*). Arsenic is also supposed to cause an increased output of erythrocytes into the circulation from the reservoirs of the bone-marrow. In pernicious anemia in the adult, arsenical therapy has been justified in a purely empirical way. In v. Jaksch's anemia, occurring in weak children during the first year or so, this form of treatment is hardly ever indicated. However, Fowler's solution, diluted, in divided doses of from 2 to 12 drops daily, may be tried. It is very important to treat the rickets if there is any present.

Attempts at organotherapy in anemia rest upon the theory of a hormone stimulation of the blood-forming parenchyma. Huebner feeds small amounts of fresh bone-marrow with bread and butter. It should be quite unnecessary to say that from active red marrow alone, may results be expected, and not from the ordinary fat marrow. Freshly expressed meat juice given in spoonful quantities is to be recommended. The transfusion of

[3] A single loss of blood in itself acts as a sufficient stimulus to erythropoiesis. The circulatory system may be more rapidly filled by the addition of water in various ways.

blood, which has been mentioned again lately, has an organotherapeutic effect beyond that of merely compensating blood loss. In post-hemorrhagic anemia, 3 to 5 intramuscular injections of 15-20 c.c. of fresh human blood at intervals of several days may be given, as well as injections of normal saline. Autotransfusion by the use of tight bandages may also be employed.

The treatment of chlorosis and secondary anemias by "physical," meaning by this hydrotherapeutic measures, is the vogue in private hospitals and sanatoriums. In the case of chlorosis, depletive sweat baths (a hot bath followed by a hot pack every five days) are possibly of value. Even here, one recommends cold sponges, cold rubs and even cold baths and douches, the other hot applications and hot water and hot air baths. Such differences of opinion do not awaken special confidence. Probably, the stimulation of the skin is the important feature, and if this is true, the method of producing it is of minor importance. In the enthusiasm over artificial sunlight, the beneficial effects of natural sunlight are apt to be overlooked. (Sun-baths on warm, quiet days, one-half to one hour twice daily, at first only the limbs, later the trunk followed by cool, wet rubs are to be recommended.) The value of high altitude lies in the therapeutic application of oxygen-poor air (David).

For older children the most effective measure is a visit to the country, with suitable outdoor or fresh air camp life at an altitude of 1500 to 4500 feet, the elevation to be governed by the child's condition and by the altitude at which he has been accustomed to live. A sojourn in such a place gives opportunity of recreation and of intelligent and instructive physical exercise in the open. Climates which experience shows to have a bad effect should be avoided. Tedious walks on the sanatorium promenade and the constant contact with convalescents should be avoided. When a southern sea-shore resort is chosen, the baths should be carefully watched. In some instances, it will be necessary to keep the child out of school for a long time, or at least to eliminate the less essential studies. Fresh air cures of a less intense nature than those carried out in the mountains and on the seacoast can be pursued at home, and with certain precautions in the large cities of the north and south. Sleeping in the open with ample protection should be tried.

Changes in the spleen noted by Banti in the syndrome bearing his name and by Eppinger in the Biermer type of pernicious anemia raises the question of the value of splenectomy in these and related conditions. Even though these changes may not be causal (as some believe), nevertheless, the value of this procedure is borne out by clinical experience. Whether exposure to the Roentgen rays can be effectively substituted for this operation is still questionable.

APPENDIX

PSEUDO-ANEMIAS

In many children, superficially adjudged to be anemic, or thin-blooded, either by the laity or by physicians, other conditions present themselves which warrant description in this connection, even though they are properly

classified elsewhere. If pallid infants or older children be examined indiscriminately their hemoglobin index and their red cell count will be found, in the major number, within physiologic limits. These children are either not in any degree anemic, or are really oligemic, that is, the vascular system contains a diminished quantity of a blood which has a normal pigment content and a normal cell content. Until recently it has been impossible to determine which of these two conditions is really present, since the opportunity of autopsy is rare and since the technical difficulties which surround the *in vivo* determination of the quantity of blood are great. However, the gap has been bridged by Erich Mueller. By the methods of Zuntz and Plesch it has been found possible to determine the total volume of blood, the total hemoglobin content, and the oxygen capacity of the blood of pale children over six years of age. Children with chronic nephritis or tuberculosis, and those living under especially unfavorable hygienic conditions, were excluded from these studies. No differences were found in these pallid children as compared with healthy-looking specimens. In such cases, therefore, one has to deal, not with an oligemia, but with a pseudo-anemia. The existence of such conditions has been suspected for some time (Sahli, Strauss and others). A reduced transparency of the epidermis or a lessened fulness of the skin capillaries have been considered as causes of the condition. The ischemia may be due to a decreased development of the capillary net-work of the skin, or to a dilatation of the blood-vessels of other organs, or to a contraction of the skin vessels—a *cuticular angiospasm*. The last two of these factors are especially associated in the occurrence of the marked color changes to which such children are liable.

Plumbism and chronic nephroses, usually responsible for the pseudo-anemia of adults, can be considered in only a small percentage of cases in childhood. An association of phenomena suggests a consideration of cause in still another direction. A group of sickly children, the subjects of hereditary taint and of errors in physical development, with a marked tendency to the lymphatic diathesis, includes many of these infantile pseudo-anemias. Psychic traumata of various types, for example, states of fear and depression associated with school duties or unpleasant home conditions play a great rôle. It is obvious that a correction of these conditions rather than iron, arsenic and the other forms of treatment so often employed, are to be recommended. A careful analysis by the family physician of these conditions as well as the intelligent coöperation of the parents are necessary, all of which of course requires greater effort than the mere feeding of pills.

B. GROUP OF LEUCEMIAS AND PSEUDOLEUCEMIAS

Pathologic research is sufficiently advanced to offer a reliable basis for the scientific classification of this group. The confusion into which the purely hematologic study of the group has been thrown by the variety of clinical findings, particularly as to relations between true and pseudo-leucemias, has been composed by pathologic researches alone. In a degree, the clinical picture worked out at the bedside still fails of likeness to the features which the autopsy presents and fuller conformity must be left to the clinician.

The several forms of disease included in this group are due to marked and more or less widely disseminated changes in the lymphoid or, the myeloid system and, taken in their entirety, may be likened to a progressive disturbance of nutrition. The following scheme will serve the purpose of eliminating a didactic presentation and will suggest the great impetus which an effective clinical terminology gives toward the association of heterogeneous conditions and the differentiation of homogeneous types.

Structural Basis.	Affections of the Lymphoid System. (Lymphadenoses.)	Affections of the Myeloid System. (Myeloses.)	Clinical Terminology.		
General hyperplasia with expansive, but not aggressive growth.	1. Lymphocytomatosis.	3. Myelocytomatoses.	With increase of cells in the blood stream.	Lymphatic and myeloid leucemia and chloroma.	
			Without increase of cells in the blood stream.	True pseudoleucemia (Pinkus-Cohnheim).	
Hyperplasia with aggressive tumor-like growth, and atypical proliferation.	2. Lymphosarcomatoses (Kundrat.)	4. Myelosarcomatoses.		Lympho- and myelosarcomata.	Pseudoleucemia.
Pseudo-hyperplasia; metaplastic proliferation, especially in the connective tissue cells of the stroma.	5. Granulomatoses.			Sternberg's disease.	

Distinctly localized leucemic and pseudoleucemic affections, that is the local lympho- and myelosarcoma of a particular organ, which have the distinct characteristics of a malignant tumor, and solitary granuloma are not included in this classification of more or less generalized diseases.

1. LYMPHOCYTOMATOSES

This group of leucemias presents, from the outset, generalized systemic disease conditions of all the lymphoid tissues. The lesions consist in the proliferation of preformed lymphatic tissue and in the adventitious growth of similar new tissue in the several organs.

Since under these conditions the elements of the non-granular system of blood-cells appear in the blood in increased number, these disorders have been designated as:

Leucemic Lymphadenosis or Lymphatic Leucemia

In lymphatic leucemia the parenchyma of the lymph nodes, the spleen, the tonsils, the lymph follicles of the tongue, and of small, disseminated masses of lymphoid tissue in other organs, as the thymus, bone-marrow and kidneys, is increased to a varying and often irregular extent by a process of atypical proliferation. Surrounding the blood-vessels widespread,

semicircular, and newly formed adventitious collections of lymphocytes are found. Usually the proliferation results in an enlargement of the affected organ which may reach a marked degree, with consequent obliteration of the normal architectural lines. Abnormal forms of cells, probably of an embryonal type, consisting of large mononuclears, atypical lymphocytes and "Rieder's" cells, appear. The infiltration of the surrounding parts often suggests malignancy; but the elements of neighboring tissues do not actually contain new cells, although they may be choked by their expansive growth.

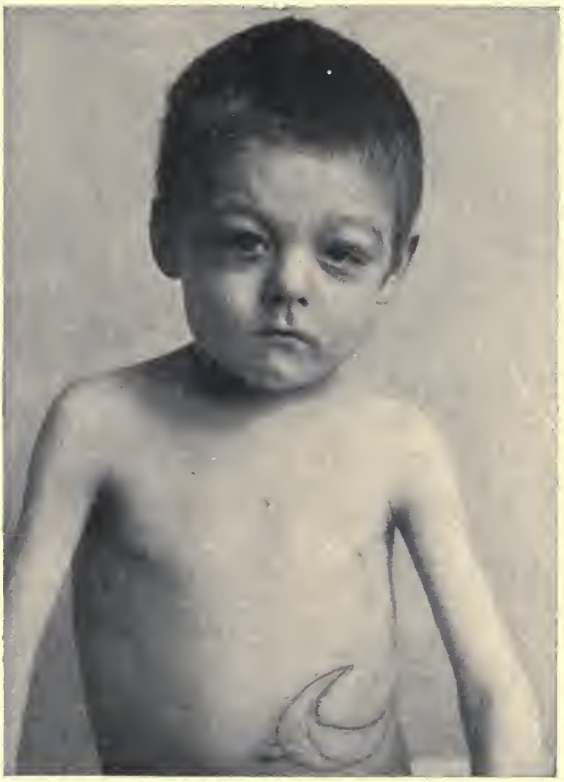


FIG. 35.—Eight-year-old boy with acute lymphatic leucemia, showing swelling of lachrymal glands.

Onset, Course and Symptoms.—The course of lymphatic leucemia, to which childhood shows a special predisposition is almost always acute, that is, it has a duration of several weeks, averaging about two months. It is always fatal. The new-born and young infants are similarly affected. The clinical picture resembles that of a sudden infection, with the prodromes of anemia. Its general symptoms are those of high fever, languor, headache, dizziness, pain in the extremities referred to the bones, numbness, vomiting, diarrhœa, and later, edema. Indications of a hemorrhagic diathesis appear in the form of petechiæ of the skin and mucous membranes, and of foul,

ichorous ulcers resulting in the breaking down of lymphomata. Hyperplasia of the tonsils and a variable degree of swelling of the lymph nodes at the angle of the jaw, in the neck, and, more rarely, in the groin occur. The thymus, liver, and spleen are enlarged and hard. Albuminuria and casts are common, together with an increase of the endogenous purin bases and the phosphates, due probably to the breaking down of cell nuclei. In advanced stages the clinical picture is further complicated by inflammation of the serous membranes. Leucemic infiltrations in the skin, mesentery, salivary and lachrymal glands, etc., are of less frequent occurrence.

The diagnosis is established by the typical blood findings. These consist in the light color of the blood, its slow coagulation, the moderate to high grade increase, both absolute and relative, of the non-granular leucocytes, among which are found undeveloped and atypical forms, and, in particular large lymphocytes and weakly staining polymorphonuclear cells (Rieder's), in excess. Large numbers of lymphocytes of normal size are found less commonly. An absolute and relative reduction in the number of cells which have their origin in the myeloid parenchyma is indicative of changes in the bone-marrow and may resemble the findings in severe toxic anemia, in the way of oligocytosis, poikilocytosis, anisocytosis, basophilic granulation, normoblasts, megaloblasts and myelocytes. If the lymph anemia is retarded because of the not infrequent occurrence of septic infection the condition may be mistaken for so-called glandular fever, for hypertoxic diphtheria with psuedomembranous deposits in the mucous membranes, or for typhoid fever. A leucemic infiltration which is said to cast a characteristic paravertebral shadow, lateral to the descending aorta, has been noted in Roentgenograms by Goett.

For reasons yet unknown, the numbers of lymphocytic elements in the circulation in such lymphadenoses may be very small. They may appear only in the later stages or they may be absent throughout the entire course.

The so-called true pseudoleucemias (Cohnheim-Pinkus), are sub- or aleucemic lymphadenoses. They must be differentiated from Werlhof's disease, from fulminative purpura, and sepsis. The more chronic and afebrile affection is quite rare, however, in childhood.

Furthermore, the physiologic lymphocytosis of early childhood may be increased to such an extent by infectious conditions (typhoid, pertussis, scarlet fever, syphilis), or by the common inflammatory diathesis, as to resemble a lymphemia or lymphadenosis.

2. LYMPHOSARCOMATOSES

Lymphosarcomatoses occur, particularly, in young individuals. They resemble malignant tumors in that their origin is not diffuse, but rather a gradually spreading affection of the lymphatic system of aggressive quality, with the formation of metastases. They represent an apparently slow transitional stage from the lymphocytomatoses (the lymphatic leucemia and pseudoleucemia of Cohnheim-Pinkus), to the true lymphosarcomata.

The most common form is the lymphosarcomatosis arising from the mediastinum. It follows a chronic course and produces a cachexia. The

mediastinal lymph nodes, frequently including the thymus, enlarge to form a densely compacted tumor mass. This mass, on account of the limited space in the thorax, soon produces the signs and symptoms of compression, in the way of dulness, shadows, pressure upon blood-vessels, nerves, and bronchi; thromboses, paralyses, stenoses, compression of the lung apices and the pericardium. Later, other groups of lymph nodes lying in the immediate neighborhood, and at even more distant points, become involved. The spleen and the liver usually remain unaffected, both clinically and anatomically. Toward the close of the disease the evidence of a hemorrhagic diathesis may appear.

The blood findings may be fairly normal for a long while. In contrast to the findings in lymphocytomatoses, the non-granular cells are never increased, but rather very frequently decreased. The consequence is a relative granular leucocytosis which may become absolute in consequence of the formation of metastases in the bone-marrow. In such cases the development of anemia is to be expected

Lymphosarcomatosis of the abdominal lymph nodes produces similar manifestations.

Fig. 36.—Six-year-old boy with so-called pseudoleucemia (Cohnheim-Pinkus). Upon admission the blood contains 5000 leucocytes, 4.2 million reds, no marked oligochromemia, 74 per cent. polynuclears, 18.5 per cent. large mononuclears and 2.5 per cent. eosinophiles. The swelling of the lymph nodes had persisted for nine months and had caused venous congestion. Intrathoracic lymph tumors compressed the trachea. Von Pirquet negative. The tumors began to disappear when the arsenic treatment was instituted and continued to diminish in size spontaneously.

3. THE MYELO-CYTOMATOSES

The myelo-cytomatoses are systemic diseases of the myeloid tissues of the body. They are characterized by proliferation and increased functional activity of the preformed myelotic tissue in the bone-marrow and by diffuse adventitial new formations of such tissue in the various organs. Besides the bone-marrow, the spleen, the lymph nodes, and the liver are especially affected by these intumescent new growths. In these organs the lymphoid parenchyma is choked and replaced by myeloid tissue. The mucous and serous membranes are not affected as they are in the lymphocytomatoses.

Because of the increased number of cells of the granular system which appear in the circulation in the myelo-cytomatoses the terms *leucemic myelo-cytomatosis* or *myeloid leucemia* are employed.

Cases of this rare form of disease have been definitely diagnosed after

the sixth year.[4] Almost all of them have a chronic course extending over several months or years and have proved fatal.

Symptoms.—The first definite symptoms are usually those of mechanical origin, resulting from the size of the tumor and the pressure of the hard, rough spleen, which gradually grows to an enormous size. Even then, the general well-being and the appearance of health may continue for a time. Later, as a result of hemorrhage, symptoms of cachexia and signs of anemia, such as pallor, lassitude, dyspnœa, palpitation, anorexia, and loss of weight appear. Coincidently, spontaneous, and pressure pains in the bones develop and alterations of hearing and a hemorrhagic retinitis are observed. The urine shows protein, casts, and increased output of uric acid. Moderate swelling of the superficial and deep lymph nodes often occurs, but only after the long continuance of the disease. Irregular rises in temperature are frequent.

Fig. 37.—Myeloid leucemia, boy of twelve years; Intermission with disappearance of all symptoms under treatment with arsenic and X-Ray.

If the blood is highly leucemic, its increased viscidity and diminution of color, together with the formation of a grayish-white sediment upon standing, and its slow coagulation, may be noted, but less distinctly than in lymphemia. The anemia is usually of a simple normoblastic type; less commonly it may be megaloblastic. The degree of the anemia varies greatly. The number of white cells is usually between 100,000 and 400,000. All forms of cells of the granular system take part in the absolute increase, so that the blood looks as though bone-marrow had passed into the circulation. Myelocytes of the most variable types of granulation are relatively numerous, ranging to about fifty per cent. Occasionally, the eosinophilic and basophilic granular leucocytes are also relatively increased. The neutrophilic granular polymorphonuclear cells are usually present in but slight excess. The appearance of myeloblasts and abnormal cells toward the end of the disease is a noteworthy fact.

Diagnosis.—Difficulties in diagnosis are encountered when the blood picture and the splenic enlargment have been affected by complicating conditions or by therapy. A high leucocytosis in anemic patients, arising from such causes as sepsis and granulomatosis, is to be considered. In these cases, however, the blood picture is less varied and the increase of the

[4] In younger children, in whom a diagnosis of myeloid leucemia has been accepted, the process was probably always that of Jaksch's anemia. Reports in recent literature seem to indicate that this form is more common than was formerly believed.

eosinophiles and the mast cells is also wanting. For practical purposes the disease may be differentiated from Jaksch's anemia (which, both clinically and pathologically, differs only in quantitative degree from myeloid leucemia), by the minimal age limit in the occurrence of the former to five years. Occasionally, at least, myelo-cytomatoses may take a sub- or aleucemic course, and hence the use of the term myeloid pseudoleucemia.

4. MYELOSARCOMATOSES

The myelosarcomatoses, or multiple aggressive myelomata, developing both within and without the bone-marrow, are of a type of disease which does not occur in childhood

Chloromata

The chloro- lympho- and chloro- myelosarcomata, which may be regarded as deviations from the lympho- or myelo-cytomatoses, or as forms of the latter pursuing a peculiar course, are relatively common in early life. They present, at times, the characteristics of leucemia, but occur more often without them. Most of the cases of chloroma observed have been in young males ranging from two to thirty years. One-half of these have been in children.

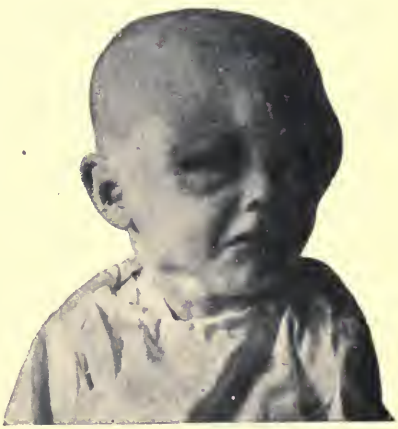


Fig. 38.—Three and one-half-year-old boy with chloromatous tumors on the flat cranial bones, vertebra, and orbits, with hyperplasia of the lymph nodes and anemia.

The chloromata are distinguished pathologically from the corresponding leucemias and pseudoleucemias by extensive, generalized, subperiosteal cell proliferation. For some, as yet unknown reason, these proliferations are often of a yellow or grayish-green color. They are most commonly found in the flat bones of the cranium, especially in the orbital region, the temporal bones, and the zygoma, and in the bones of the thorax. Similar proliferation occurs, less frequently, in the skin and serous membranes.

Clinically they are distinguished by the formation of symmetrical tumors, growing within the cranial cap, the spinal canal, the mouth, and the mastoid cells; and causing such pressure symptoms as protrusion of the optic bulb, paralyses of cranial nerves, etc. These characteristic clinical signs develop early. Their aggressive character gives the chloromatous diseases a position between the cytomata and the sarcomata. In other respects their clinical and pathologic findings correspond closely to those of the common forms of lymphocytomatoses or myelo-cytomatoses. Their course is always acute and very severe. The duration of the disease is, at least, a matter of a few months.

5. GRANULOMATOSES

Granulomatoses are formed by chronic inflammatory processes which gradually develop hard, compact, granulation tumors of the lymph nodes, with a tendency to necrosis, induration, and scar formation, without lymphocytic proliferation or aggressive growth. Essentially they consist of endothelial and epithelial cells, of giant, spindle, and round types, mixed with fibroplastin and fibrin. The granulomata may be generalized from the beginning, or persistently localized. The lymphatic tissue of the spleen and the liver is usually affected during the course of the disease, while that of other organs, as the bone-marrow is but rarely involved.

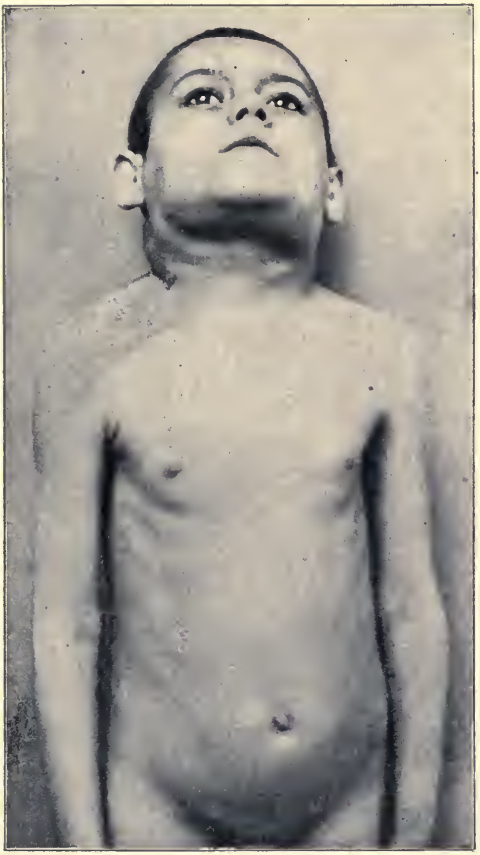


FIG. 39.—Seven and one-half-year-old boy with granulomatosis, most marked in the cervical nodes. Confirmed by histological examination.

Symptoms.—At first, soft, grape-like enlargments of the lymph nodes appear in the neck. These may be unilateral or successively bilateral. Signs of pressure in the mediastinum are soon apparent. In the diffuse form, enlargement of the spleen and liver follows the gradual hardening of the enlarged lymph nodes. Periods of severe intermittent fever, occurring even in tuberculous individuals, are characteristic. The suspicion of granulomatosis is definitely sustained by a persistent diazo-reaction of the urine. The diagnosis is fully established in the living by the histologic examination of an excised gland. The diffuse type of the disease runs a chronic course, unaffected by treatment, producing anemia and cachexia, and always ends fatally.

The localized forms, and especially those occurring in luetic patients, are amenable to treatment. In contradistinction to scrofulous lymphadenitis, the granulomata do not tend to form epidermal scars or perforations, and have no relation to regional diseases of the skin and mucous membranes.

Pathogenesis of Leucemia and Pseudoleucemia

Myelo-cytomatosis occurs physiologically in the embryo in practically the same manner as does myeloid leucemia. It develops after birth by a metaplasia due to the action of infectious, toxic, or actinic stimuli. This is equally true of embryonic lymphocytomatosis, which differs only quantitatively from lymphoid leucemia. Upon these facts (Naegeli), our knowledge of the pathogenesis of the leucemic diseases is based. Their etiology is entirely obscure. Some forms of pathologic leucopoiesis, as initially noted in the evolution of myelocytes in osteosclerosis, may be considered purely compensatory. In others, as in the formation of myelocytes in post-hemorrhagic, toxogenous or infectious anemia, the increased requirement or demand may be a factor. In still others, a direct, specific stimulative action of endogenous or ectogenous substances, serving as specific excitants, may play a part.

The granulomata are, in some cases, directly dependent upon syphilitic poison and, in others, upon a tuberculous infection (Sternberg). The granular form due to the tuberculous virus (Much-Römer), is especially common. In another group of these cases—the malignant granulomatoses—however, neither a syphilitic nor a tuberculous infection, nor any other known cause, can be demonstrated.

Therapy

There is no specific treatment for the leucemias of childhood. It is possible, especially in myelo-cytomatosis, to cause a direct destruction of the white cells in the circulation and to combat their excessive formation and appearance in the circulation by limiting the cytogenesis. In the early stages of the disease it is, strangely enough, not uncommon to note, not merely the remarkable disappearances of blood symptoms, but improvement in other manifestations of the disease and in the general well-being. Relapses always occur. These temporary results, which serve to prolong life, are in all probability similar, in all essentials, to the beneficial action of intercurrent infectious diseases. Benefit is quite certainly and directly secured by the use of arsenic in the form of the liquor potassii arsenitis (Fowler's solution), in doses up to twelve drops a day, continued for months. More recently benzol has given good results. Similar improvement has been obtained by the use of the Roentgen rays, applied in from two to six treatments, over the spleen and the region of groups of affected lymph nodes. A hard tube should be used, placed at least 40 cm.(16 inches), from the body, and the exposures should last from five to ten minutes. Symptoms of arsenical poisoning and toxogenic anemia may, of course, appear, while with the Roentgen method there is always danger of dermatitis. In both forms of treatment violent reactions may be harmful, resulting in fever and fatality. The most careful dosage and an extreme watchfulness will protect the patient absolutely against these accidents. A criterion of judgment of therapeutic action is to be found in a careful observation of the numbers of red cells and of the hemoglobin index of the blood, and in a determination

of the output of endogenous uric acid. It must be remembered, however, that in these cases the full effects of the Roentgen ray usually develop after several days.

In cases of aleucemic lymphadenosis and myelosis, and especially in cases of lymphosarcomata, every experienced observer has occasionally seen the most surprising results under these methods of treatment. Whether complete recovery ever takes place is still doubtful. The treatment of diffuse granulomata is hopeless. Local granulomata may be excised or treated with iodine, the internal and external application of which are often successful. This obtains, however, only when the luetic character of the tumor does not indicate other forms of treatment.

HEMORRHAGIC DIATHESIS OR TENDENCY TO HEMORRHAGE

In text-books it is not customary to group diseases according to their symptoms, but the diseases to be discussed under this title are so grouped because of the greater convenience for study and for use in actual practice. The diseases are not necessarily related, either in their nature or general manifestations. The grouping followed is based upon the more outstanding symptoms with which hemorrhage may be combined.

Group I. Purpura Hemorrhagica.—The diseases in this group are limited rather narrowly to those in which the tendency to hemorrhage is secondary to such blood diseases as leucemia and the anergic nonplastic anemias. The most frequently seen is the symptom group first described by Werlhof and known as Werlhof's disease. The primary conditions may be true blood diseases with characteristic findings, or may be of more indefinite and unexplained nature. In the latter cases the tendency to hemorrhage appears as an "idiopathic morbus Werlhoffi," that is, it is the outstanding feature of the entire symptom-complex. In all the diseases of this group the course of the tendency to hemorrhage—unless death results from loss of blood—is dependent entirely upon the primary blood disease, so that it may be acute and rapidly fatal, or a relatively chronic, benign affair with complete recovery or with intermissions. The idiopathic form may also run a course of continual aggravation or of gradual recovery.

The hemorrhages may be from the skin and subcutaneous or from mucous membranes or into muscles, but very rarely synovial. They occur as petechiæ, or larger spots following slight trauma, are asymmetric and are seen even on the head and face. The hemorrhages from the mucous membranes of the nose, mouth, vagina and at times of the bowel are characteristic. They may be very profuse and even fatal. There are no local symptoms and the general symptoms are closely interwoven with the symptoms of the primary affection and the post-hemorrhagic anemia. The bleeding time is prolonged and the contraction of the clot is incomplete. During the height of the hemorrhagic tendency the number of blood-platelets in the circulating blood is reduced to 30,000 or even lower. From a pathogenetic standpoint great stress is laid upon this "thrombopenia" in view of the action of the platelets in the mechanism of coagulation. According to these findings, the cases of hemorrhagic purpura of the Werlhof type

must be considered due chiefly to hematic causes. The thrombopenia may occur with the various intercurrent diseases and in various ways analogous to the origin of anemias. Its pathogenesis is probably analogous to that of certain primary anemias. In some cases it may be due to a crowding out of the platelet-forming parenchyma in the bone-marrow by leucemic lymphoid proliferation. In others, a marked restriction of the development of bone-marrow. Or again increased thrombocytolosis in the spleen or its associated tissue due to hyperplasia. Further, a congenital deficiency of the platelet-forming marrow in a condition, no doubt, related to familial hemophilia for which Glauzmann has suggested the name "hereditary thrombasthenia" may be a factor. The action of blood poison of all sorts may be causative. Since infectious diseases may also cause a thrombopenia either directly or indirectly by the action of toxins, this type of hemorrhagic diathesis is also seen as a concomitant, or complication of these and more especially after scarlet fever and diphtheria.

In the treatment of these conditions transfusions, injections of autoserum, subcutaneous injections of horse serum and salt solution, and various commercial coagulation producing products are recommended. Internally, liquor potassii arsenitis (Fowler's solution) in increasing doses up to twenty drops per day, calcium lactate or preferably chloride in large doses (6-10 grams per day) and gelatin either subcutaneously in ten per cent. solution or in large amounts by mouth are useful. The use of Roentgen treatments to the spleen is indicated when that organ is enlarged.

Group II. Hemorrhage due to Multifocal Infectious Disease.—This group is characterized, clinically, by the close relationship to obvious infectious diseases such as meningococcus meningitis, measles, influenza, sepsis, chicken-pox, tuberculosis and congenital lues. In these conditions we may have multiple, circumscribed, more or less autonomous disease foci in the peripheral vessels, resulting in emboli, thromboses, hemorrhagic exudations, and erosions of the vessel walls which in turn cause bleeding. So that the hemorrhagic tendency in these conditions is largely due to organic vascular lesions. Its occurrence is in association with and dependent upon the basic infectious disease.

Group III. Hemorrhagic Arthritis (Schoenlein-Henoch).—This group consists of diseases with a rather definite symptom picture. It occurs most frequently in older children, and is not fatal. They are characterized by recrudescences lasting several days or weeks and the entire course lasting for weeks or months. In definite cases a symptom triad predominates. This consists of cutaneous purpura, intestinal colic, and articular pains with or without swelling of the joints. All these symptoms are supposed to be due to hemorrhage or the results of hemorrhage. The subcutaneous hemorrhages are petechial or may become as large as small coins, superficial, and are more or less symmetrical on trunk and limbs. Actual bleeding is uncommon. Frequently skin eruptions, edema and proteinuria is encountered. The skin eruption may be urticarial, erythematous or multiform in type. There may be no fever and the general manifestations are usually mild. The etiology is entirely unknown, but the theory of an

infectious or anaphylactic nature is generally accepted but far from proved. It is supposed that a functional vascular injury, that is a toxic capillary paralysis with dilatation and diapedesis, is concerned in the causation. Usually no treatment other than rest in bed is necessary. The remedies advised under purpura hemorrhagica may be employed.

Group IV. Scurvy.—In this group there is a distinct relation between the hemorrhagic tendency and food deficiency as shown by its prompt reaction and relief by diatetic treatment. True scurvy has been very uncommon in civilized countries, but during the World War was again quite frequent in the countries where lack of proper food occurred. Infantile scurvy, Barlow's disease, in children under two years is not rare at any time. According to recent investigations the two conditions are closely related. Their manifestations differ because the immature osseous system of the infant reacts differently to the injury than does that of older patients. It is still doubtful whether this injury is primarily one of the bone-marrow itself or whether it is due to vascular changes affecting the marrow.

INFANTILE SCURVY (*Barlow's Disease*)

This condition is of great interest to the pediatrist and requires full discussion here. The scurvy of adults is left for text-book of internal medicine.

Etiology.—Naegeli and other authors suggest as a cause of the symptoms a reduced proliferation of the interstitial connective tissue of the marrow, with transition of marrow cells into fibrous connective tissue at certain points of most rapid growth as in the diaphyses, costochondral margins and cranial bones. This is supposed to have two results.

First.—The activity of the osteoblasts and the resulting bone growth is reduced at the affected points. With continuing resorption of the already formed bone, the wall becomes thinned resulting in localized weakness or myelogenous infantile osteotabes. Muscular action and slight trauma cause infractions and fractures of the diaphyses of long bones and the ribs. The growth in length is delayed. (Figs. 40 and 41.)

The bone affection described above differs entirely from true rickets. The addition of rickets, however, may add to the fragility of the diseased ends of the diaphyses. In contradistinction to rickets, the mineral salts, especially calcium, are found to be retained in infantile scurvy and are liberated when diatetic treatment is instituted. During the florid stage, the mineral content of the bones and muscles is low.

Second.—The hematopoietic function of the bone-marrow suffers. The result of this is progressive anemia and a tendency to hemorrhage. The latter is especially seen, in the event of fracture, in the development of subperiosteal and medullary hemorrhages. The tendency to bleeding shows itself, also, at other points.

Symptoms.—The osteotabes causes severe pains in the affected bones; deformities and crepitation; distension, with dough-like swelling of the soft parts from the underlying blood deposits; and tenseness of the skin. Severe pain is localized principally at the ends of the diaphyses and is excited by movement or touch. As a result there may be a degree of pseudoparalysis,

but this is not a constant sign (Fig. 43). Deformities appear in the affected bones and especially in those of the fore-leg and the thigh, at the knee-joint, and more rarely in the bones of the arm and forearm, at the elbow or shoulder-joint, and at the costochondral articulations. The sinking in of

Fig. 40.—Humerus of a child of one year with infantile scurvy. The soft parts have been dissected away in order to show the impacted fracture of the neck more clearly.

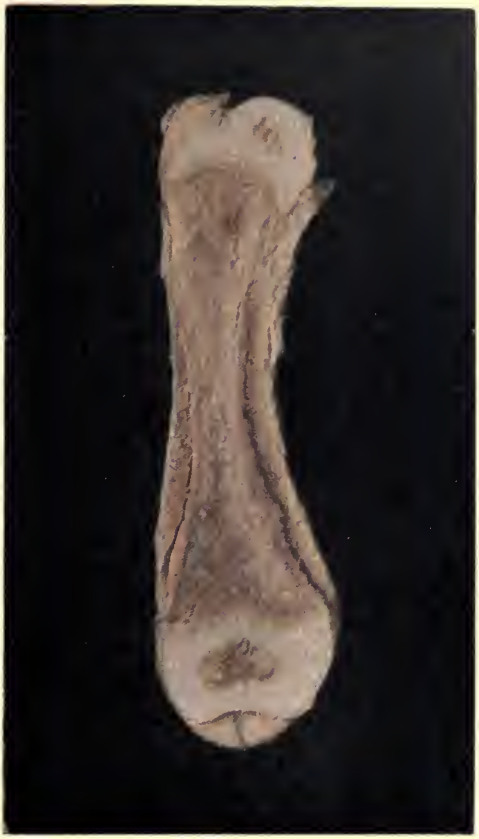


Fig. 41.—Femur of child, infantile scurvy. Hemorrhage into the marrow and subperiosteal tissue with loosening of the periosteum.

the sternum with the costal cartilages is a good example of the deformities which may arise.

In the mucous membranes the hemorrhagic diathesis is manifested commonly by hemorrhagic swelling and softening of the gums. These occur only when the teeth are present or are erupting. It is further indicated by conjunctival, nasal, intestinal and urinary bleeding; the two latter sources being shown by bloody stools and hematuria. Extensive extravasations of blood, of both subperiosteal and supraperiosteal origin appear in the jaw and in the long bones affected. Hemorrhage of the periosteum of the orbital plate is recognized by exophthalmos and by infiltration of the eye-

lids; while bleeding from the parietal periosteum is recognized by the formation of cephalhematomata. In the skin, pallor, petechiæ, suffusions and edema are accepted signs of anemia. In a small number of cases blood changes, in the form of oligochromemia, oligocythemia, poikilocytosis and relative lymphocytosis, may be determined by examination. Tachypnœa, tachycardia, cardiac murmurs and dilatation of the heart may also develop. A typical fever frequently appears and is probably due to the resorption of extravasated blood. Food is often entirely refused.

Scurvy usually presents itself to the observer in the following manner. After very slight prodromes, such as disturbances of sleep and disposition, marked thirst, and loss of color, and sometimes even without these, one notices, usually during the bath or while handling the child, that it gives evidences of pain upon being touched and especially in the legs. Soon after, swelling of the knee, on one or both sides, and of the ankles becomes apparent. The type of the deformity is different, however, from that produced by hemorrhage into the joint cavity. It is clearly a disease of the long bones themselves. The legs are held motionless upon the bed, in external rotation, with abducted thighs and slight flexion of the knees. A few days later, a spongy thickening of the gums around the teeth, or beside the teeth which are erupting, is to be noticed. At the same time, small hemorrhages, resembling flea-bites or larger suffusions of blood are seen, here and there, upon the skin. The mother reports a peculiar red discoloration of the soiled diaper and a sediment, consisting of erythrocytes, appearing in the fresh urine. The latter may even present the characteristic findings of acute hemorrhagic nephritis. The general condition of the patient becomes more serious, the face increasingly pallid, the restlessness more marked, while the immobility of the limbs—consequent upon the suffering heightens the impression of extreme debility.

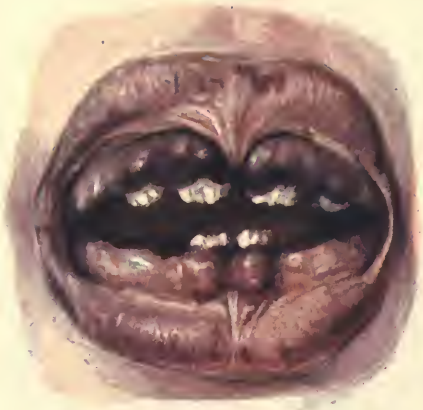


Fig. 42.—Changes in the gums in a case of infantile scurvy.

Diagnosis.—The affection of the gums, the pseudoparalytic symptoms, the painful swelling of the limbs, together with the hematuria and hematomata are especially significant in the diagnosis of scurvy. The combination of the hemorrhagic diathesis with multiple lesions of the bones, associated with certain external conditions, is almost pathognomonic. However, hematuria is occasionally the only symptom. The Roentgenogram may be very characteristic. It shows a dark shadow strip at the border of the diaphyses and may also show the shadow of blood extravasates under the periosteum along the shaft. (Fig. 44.)

Course and Prognosis.—The onset is usually gradual and is marked by a certain disturbance of nutrition, its most delicate symptom. Its typical qualities appear successively. Untreated, the disease is chronic and is slowly progressive for months. Recovery may take place spontaneously in mild cases. The severer forms are usually supposed, however, to be fatal, terminating in enteritis or pneumonia. The prognosis depends upon the skill of the attending physician.

Occurrence and Etiology.—The great majority of cases begin from the ninth to the fifteenth month. Cases that develop at a late period, say, during the second, third or even the fourth year, or those beginning earlier in the third to the fifth month, are much rarer. The disease is more frequent in

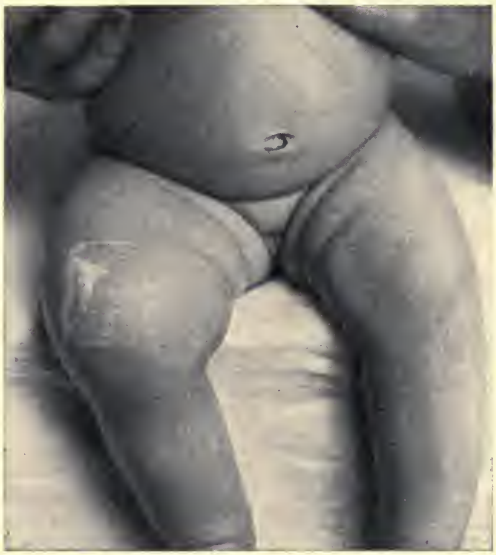


FIG. 43.—Position of the thighs in infantile scurvy.

Germany and in the north of Europe than it is in the south. In some southern countries it is almost unknown. This infrequency is actual and is not due to any want of recognition. It seems to spread from the northern latitudes (see Rickets). Male children are affected almost exclusively and especially those who are fed entirely, or to a large extent on commercially prepared and sterilized milk or other artificial food. For this very reason, scurvy is uncommon among the poorer classes. It goes without saying that not all children so fed are affected, but only those with a definite predisposition. None of the hypotheses, of course, such as injury to the milk by heating, by bacteria or bacterial toxins, by icing and storage, or by silicic acid derived from glass containers, are tenable. In fact, the attempt to classify scurvy among disturbances of nutrition results in stretching out conceptions of the disease beyond all reason.

Therapy.—Since Barlow first recognized scurvy there has been no disease of childhood that has proved so gratifyingly amenable to simple treatment. Nor is there a disease of this period upon the course of which pediatric ignorance has had more serious results. It has been mistaken for rickets, osteomyelitis, fungus, syphilis and sepsis.

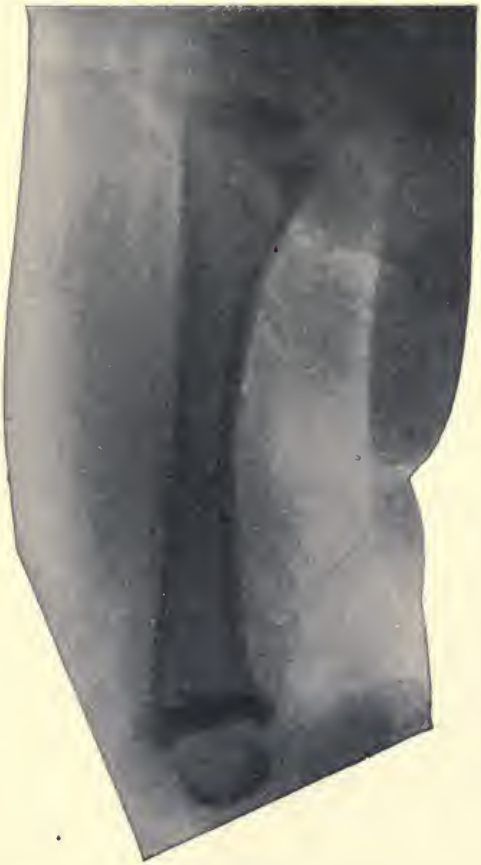


Fig. 44.—Roentgen picture of the right thigh of a girl of one year with scurvy. Subperiosteal hemorrhages of the femur.

In its treatment, natural foods, such as raw cow's milk, or human milk; several dessertspoonfuls of fresh orange, lemon, or grape juices, each day; ripe, finely divided fruits, such as apples in season; mashed vegetables, or raw meat juices should be given. Intestinal catarrh is no contraindication for these foods. The results of their use are truly miraculous. They are subjectively recognized in a very few days and all objective symptoms disappear in a few weeks. The repair of severe bone lesions requires, however, several months. Fractures do not require supporting splints, since the remaining periosteum seems to suffice to keep the bones in place. Baths, antiphlogistic applications, and all unnecessary handling of the child should be avoided.

Certain mechanical disturbances of circulation may lead to hemorrhage as in case of congestion due to cardiac decompensation or diseases with convulsions or coughing. Besides this severe cachectic processes, many organic nervous disorders, even psychic influences may give an increased tendency to bleed. The hemorrhagic diseases of the new-born will be discussed in another section.

RICKETS

Pathologic-Anatomy.—The most important pathologic changes in rickets are those of the skeleton. In the acute stage we find, macroscopically a more or less general softening of the bones, which become deformed by swelling, decurvation and formation of callus. The periosteum and bone-marrow are hyperemic. Over some bones especially those of the cra-

nium, the periosteum may become so greatly thickened as to resemble a spongy exudate.

The most important microscopic findings, also are seen in the bony framework and are as follows:

1. The bone and cartilage formed during the progress of the disease are insufficiently calcified or entirely uncalcified, so that in acute rickets, non-calcified cartilage and bone tissue, the so-called osteoid tissue, of much greater thickness or width than normal, is found throughout the skeleton. This especially is seen in those parts where, normally, the growth is particularly active, as in the periosteum and in the subchondral zones. The new formation of osteoid tissue occurs at an abnormal rate in these parts. At the diaphyses, the points where tendon insertion and muscular attachment constantly subject the bones to extreme flexion or extension, the mechanical insufficiency due to the extreme softness of the osteoid tissue leads to an attempt at compensation by excessive new formation of bone, or by decreased resorption of it. This active proliferation and mass formation is probably a secondary manifestation and naturally occurs as a result of increased vascularity. This is known as Kassowitz' inflammation, rickitic osteitis, or periosteitis. When this condition is most marked it is described as a hyperplasic osteophytic form of rickets.

2. Bones, which at the onset of the disease were completely formed, become poorer in calcium salts and therefore softer (osteomalacia). This decalcification must not be considered analogous to that produced in the treatment of bone with acid, to make it easier to cut, as was once taught. It is not a chemical decalcification. It occurs, rather, as an actual biologic process, by which the physiologic resorption of the older portions of the bone tissue continues, while the absorbed portions are replaced by newly-formed tissue, poor in calcium or containing no calcium at all, so that the removal of the lime salts in the mature bone is actually a result of the non-calcification of the osteoid tissue, described in previous paragraphs. It is entirely comparable to the decalcification which occurs in osteomalacia. It is, accordingly, most pronounced in those parts in which the bone is normally thin, as in the craniotabetic areas on the posterior aspect of the skull. The loss of the salts in rickets may convert the long bones into rubber-like, elastic strands that are easily severed—the osteomalacic form of rickets.

According to von Recklinghausen this softening of mature bones takes place at a rapid rate in rickets. It goes on in circumscribed areas and is produced by the tryptic autolytic action of the osteoclasts and newly formed vessel shoots.

3. The endochondral ossification is disturbed in an equally characteristic manner. The preliminary calcification of the cartilage is wanting, and under markedly increased vascularity the zone of cartilage proliferation becomes irregular in form and greatly extended. This extension arises from the delayed resorption, which in turn is due to the disturbance of calcification. The impairment of the endochrondral ossification, characteristic of rickets, is closely related to the longitudinal growth of the bone

during the course of the disease. In consequence, it is absent in the osteomalacia of adults. In a word, the disturbances of ossification are due entirely to the non-calcification of both cartilage and bone.

4. Occasionally, fibrous foci have been found in the bone-marrow of rickitic children. Recently, however, Marfan, in opposition to older observers, has held that the changes in the bone-marrow in rickets are constant and pathogenetically of great importance (see below). According to this observer, the first or clinically latent stage of rickets is characterized by an irritative, atypical and aberrant proliferation of the cells of the parenchyma of the bone-marrow and of the cartilage. The second and clinically manifest phase is characterized by the formation of a fibrous marrow (see Scurvy). Ziegler, also, considers the proliferation of the cells of the bone-marrow, and especially of those which he calls endosteal cells, an essential change.

In the stage of recovery, the temporary calcification of the cartilage and the changes of the osteoid tissue to hard, solid bone, *i. e.*, eburnation takes place.

In severe rickets, rather characteristic changes of the soft parts are also found and especially in the striated as well as in the unstriated muscle tissue. These consist in the development of slender immature fibres, with increased nucleation and in the modification of the longitudinal striations. They are interpreted as specific disturbances of nutrition of a retrograde character.

The liver and spleen are often enlarged, due to hyperplasia of pulp and follicle. The lymphoid tissue throughout the body is markedly swollen. The brain is enlarged as though swollen.

All these lesions bring about the characteristic habitus and picture of rickets; the large head, the short rounded chest, the pendulous abdomen, and the short plump extremities.

Chemical Findings.—In rickets the osseus tissue is extremely poor in mineral components and especially in calcium and phosphorous. The soft parts are less affected. The magnesium salts are reduced absolutely but increased relatively. The ash of the dry tissue of the ribs and vertebra may be reduced from sixty per cent. to as low as twenty per cent. The swelling of the cartilage is probably caused by the loss of lime salts, since the Ca-ions inhibit the process of water absorption. This plays, however, but a minor part in the increase of mass. It should be noted that the insufficient calcification of the bony skeleton in rickets corresponds to a reduced calcium retention throughout the body. The loss of salts in the rickitic infant may occasionally cause a negative calcium balance, which involves an increased calcium requirement during recovery—a fact which can be proved by metabolism experiments. Up to the present time, no further findings of particular pathogenetic importance have been demonstrated.

The calcium content of the blood in rickitic infants is subject to wider variations than in non-rickitic infants, both above and below the normal (Aschenheim). Since this figure consists of three quotients, that is, the true blood calcium, the calcium of the food metabolism and the waste

calcium, the interpretation of this variation is not too clear. Negative balances of alkalies, chlorides, sulphur, and magnesia are not characteristic of the mineral metabolism in rickets, which explains the difference from infant atrophy and clears up their relation to tetany.

Pathogenesis.—The anatomic study has shown that the basic cause of the characteristic findings in the skeleton is due to the failure of proper mineralization of cartilage and bone. With the recognition of this fact, a further study of the pathogenicity of the disease was given a definite basis for promising inquiry, but beyond it positive knowledge does not extend.

Why do the newly developed parts of the skeleton take up little or no inorganic salts from the beginning of the disease? Three plausible possibilities present themselves:

1. The intake of the mineral constituents of the body may be quantitatively insufficient, or the form in which they appear in the blood may be unsuitable. This might be due either to a scarcity of calcium in the foodstuffs, or to a relative inadequacy of the calcium digestion and absorption. Proceeding upon this theory, researches, into which obvious factors of error entered, were presented to sustain the surprising view that the mother's milk could not long fulfill the demand for lime salts put upon it by the growing organism of the child. This hypothesis, repeatedly advanced, but unsupported by pediatric authority, may be dismissed. Confirmed experiments offer no proof whatever of a primary disturbance in the calcium or in any other specific phase of metabolism.

The premises upon which this teaching rests are not tenable. Brubacher, Stöltzner, Cronheim and Mueller, contradicting the findings of other writers, show that the alleged reduction of the calcium and other salts does not manifest itself in the soft parts of the body as it does in the bony tissue.

Further, the consensus of judgment of numerous pediatrists indicates that neither an increase of the calcium intake, nor an improvement of calcium absorption will successfully serve as either a prophylactic or a therapeutic measure in acute rickets. The form of disease induced, in growing animals, by a calcium-free diet differs from rickets.[5] This is both histologically and biochemically true. Under such calcium starvation, an extract of the muscular, cartilaginous and bony tissues dissociates and takes up the calcium ion from a neutral solution of the calcium salts with avidity. This is not observed to so marked a degree with rickitic material.

2. An intrinsic disease of the growing skeleton itself may interfere with its proper mineralization. This is not to say, of course, that rickets is an affection limited to the skeletal parts; but rather that an abnormality occasionally hindering the mineralization of the skeleton might be considered analogous to such other disturbances, in other systemic organs, as myo-dystrophia, anemia, etc.

Such prominent pathologists as Pommer and von Recklinghausen do not concede, as Kassowitz supposes, that actual inflammatory processes play any part in preventing calcification. Schmorl opposes the statement

[5] Stöltzner, Schmorl.

of Stöltzner who holds that the fundamental fact in rickets is an inhibition of development; that the normal metaplasia, microchemically demonstrable, which matures the osteoid tissue as a calcium absorber, is absent in rickets. Similarly, the recent experiments of Adele Hartmann show that pieces of rickitic cartilage placed in the abdominal cavity of the rabbit do not retard calcification any more markedly than do non-rickitic tissues similarly introduced. According to these researches, the tissues of the rickitic patient do not lack the power of calcium absorption. They tend, rather, to show that there is, in the rickitic organism, a functional limitation upon the mineral metabolism.

3. This view is embodied in the third of this group of hypotheses, which postulates a continuous decalcification and a consequent failure to store the lime salts as a result of an abnormal acidity of the body fluids. Aside from the fact that the condition is one, not only or merely of decalcification, but of actual demineralization, this theory violates Stöltzner's finding of a normal reaction of the blood serum in rickets.

As a result of the discovery of primary, irritative, systemic, changes in the bone-marrow, Marfan has recently come to the conclusion that these processes affect the function of the osteoblasts arising from the bone-marrow and thus hinder calcification. The proposal of this theory of the myelogenous nature of the disease, in spite of Zeigler's teaching as to the functional independence of the blood-forming and the bone-forming marrow seems especially noteworthy in view of the relation of rickets to scurvy and to von Jaksch's anemia. Ribbert finds a toxogenous necrosis of the cartilage cells in rickets.

Other hypotheses and, in particular, that which lays the cause of the disease to the absence of an internal secretion of certain glands, as the thymus, thyroid, suprarenal body or liver, lack, at present, reliable positive support. Erdheim claims to have produced rickitic bone changes in rats by the excision of the parathyroids. Animals in which rickets occurs spontaneously are said to have enlarged parathyroids.

Occurrence and Etiology.—It is difficult to form any statistical estimate of the frequency of rickets because the subjective signs of it so closely simulate physiologic conditions. Systematic reports of anatomic findings in the large mass of unselected material coming to autopsy should be accepted, rather than the extraordinarily variable and but crudely oriented results of clinical examination. The following table is computed from the autopsy reports of Schmorl. This indicates that the disease never appears before the middle of the second month and only occasionally after the end of the second year, and that it affects almost every child between these two periods.

It should be noted that this table includes children who have died at an early age; and that they were probably, in large part, of the poorer classes. It is also to be observed that rickets is exceptionally common in the material presented by this author.

It is the experience of the author that premature infants are not affected

with rickets, at the same actual age as other infants, but at periods of relative development.

In certain families the frequency and severity of rickets appear to be influenced by hereditary factors. Not only does the disease appear even under favorable circumstances in the children of rickitic parents, but it has been noted, selectively, in the progeny by different fathers of a mother who was rickitic. The heredity of a structural tendency to rickets is doubtful.

The occurrence of the disease is influenced by the method of feeding. Severe forms are very much more common in overfed and artificially-fed babies than in the breast-fed.

It cannot be said that other diseases, *e. g.*, disturbances of nutrition and infections, determine the appearance of rickets. In fact the diseases which

TABLE II.

Rickitic and Non-Rickitic Children at Various Age Periods.

Age in months	0–1	1–3	3–6	6–9	9–12	12–18	18–24	24–36	36–48
Non-rickitic children	100	39.4	3.0	6.0	2.7	1.7	9.1	12.3	29.4
Early rickets, microscopically recognized	...	48.5	55.8	32.0	9.2	6.8	6.1	....	....
Acute rickets recognized macroscopically and microscopically	...	21.1	20.6	43.2	60.0	54.2	30.3	20.1	5.9
Convalescing cases	...	....	20.6	18.8	26.7	25.4	33.3	21.5	8.8
Recovered cases	...	....	....	....	1.4	11.9	21.2	46.1	55.9

limit growth seem rather to counteract the development of rickitic changes. Atrophic infants, for instance, are never severely rickitic. Nevertheless, the signs of rickets are not commonly seen in naturally-fed or in suitably, if artificially-fed children who are scrupuously cared for and are of normal or supernormal development and have shown no evidences of nutritional disturbance. If fat infants, who are frequently ill and of consequently lowered resistance, seem to be especially predisposed to rickets, it is possible that their diminished bodily activity plays a more or less important part. In general, the children of the well-to-do are affected as often, but certainly not so severely, as the children of the poor.

Furthermore, rickets, as a disease of the masses, is influenced by modes of living. All those influences of domestication and civilization which disturb the natural and primitive habits of life; which tend to close and crowded housing of the poor; which favor indoor and sedentary occupations, and permit but limited and unbalanced physical exercise, favor the occurrence and development of rickets.[6]

The most potent factor resulting from domestication, to which Kassowitz calls particular attention, is the element of respiratory injury. It is held responsible for the high prevalence of rickitic disease in densely populated tenements.

[6] Von Hansemann, Neumann.

Findlery's observations, of a strictly experimental order, show that in forms of disease identical with, or closely resembling human rickets, produced or spontaneously incurred in young animals kept in confinement, the toxic, infections or nutritive injuries present were less directly causative of the disorder than was the enforced change from a natural mode of living to existence in close, dark cages.

Numerous historical facts sustain this teaching. The geographic and racial distribution of rickets is in accord with it. Its gradual increase in frequency since the seventeenth century; its present maximal dissemination through all the countries of the temperate zone; its relative scarcity, without evidence of racial immunity, in the arctic circle, in the tropics, at high altitudes and in all sparsely populated regions, are all significant. Its prevalence or greater severity in the winter and spring months, and in the densely peopled parts of large cities; and, finally, the important influence of fresh air, natural feeding and intelligent management in the treatment of the disease, testify in support of such a rational theory.

A factor recently added to the etiology of rickets and brought into much prominence is the question of the lack of certain accessory food substances (vitamins). While the theories about this factor have formerly been rather vague, American observers in animal experiments and pediatricians in the central empires, during the food shortage, caused by the blockade have added considerable research to this view. A fat soluble vitamin (fat soluble A) is said to prevent rickets. It occurs in various foods in varying degree, as follows: whole milk, cod-liver oil, butter, egg yolk, red meat, and to some extent in fresh vegetables. It is supposed to originate in green vegetables and germ cells of cereals. It is fairly thermostable. It does not originate in the animal body. It is well to await further research upon this large question before dropping further study of the etiology of rickets.

Clinical observation of the disease indicates that two important factors enter into its pathogenesis; the first, a latent predisposition dependent upon a specific heredity, a rickitic diathesis or constitution, as it were, of the nature of which we are profoundly ignorant; and second, the infliction of certain nutritive injuries, arising from numberless causes and occurrent perhaps even in intra-uterine life.

CLINICAL SYMPTOMS AND THEIR ORIGIN

Skeletal Manifestations

First Group: Phenomena of delayed bone development; hypogenesis ossium.
Second Group: Phenomena of bone softening, osteomalacia.
Third Group: Phenomena of excessive new formation of bony tissue; osteoid hyperplasia and development of osteophytes.

In *the first group* of symptoms, the delayed growth may affect any or all parts of the skeleton; it may cause a large number of abnormal conditions and may particularly disturb the symmetrical proportions and measurements of the body. The slow growth of the facial bones gives the cranium the appearance of being unduly large. Disease of the vertebral column and of the long bones affects the growth in height. Rickitic children are shorter

than others, even if no actual distortion of the bones occurs. During the acute stage, and even later, there may be long continued and complete arrest of growth and consequent dwarfing. The latter result is especially common in cases which appear late and persist for a long period. This retarded growth and the accompanying flaccidity of the muscular tissues, with the painful periosteal points at the insertion of the muscles, is apt to delay the acquirement of the static and dynamic functions of the child, as

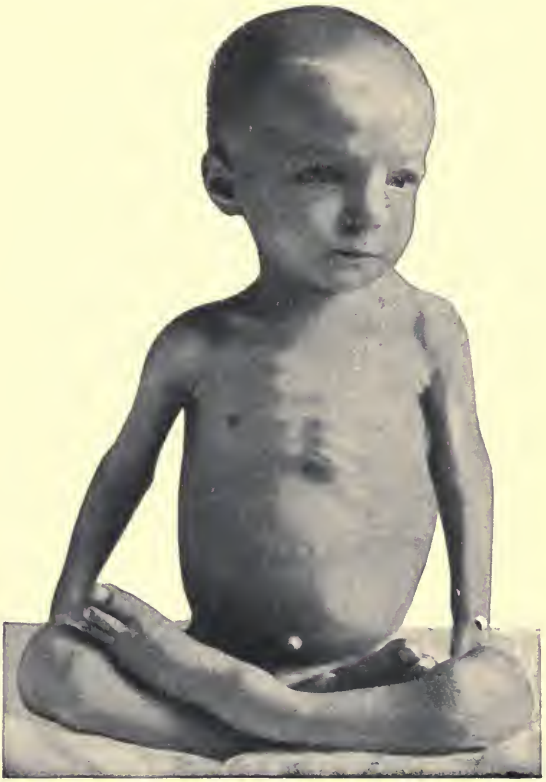


FIG. 45.—Three and one-half-year-old boy with rickets. Characteristic sitting posture with legs doubled under the body and arms supporting the spine. Slight deformity of the head, thorax and limbs

in the raising of the body, in sitting, crawling, standing and walking and in the general mobility of the extremities. When the disease appears late in infancy or in childhood, the ability to walk may be lost. When a rickitic child is lifted, it does not attempt to stand, but keeps its legs drawn up. In sitting, the legs are doubled under the body and the arms are used to support the spine. The impaired ossification of the flat cranial bones is shown in their abnormal width and in the delayed closure of the sutures and fontanelles. The anterior fontanelle increases in size for a time and occasionally remains open, or membranous, until the third year. The retarded

development is especially noticeable in the teeth. Both the beginning and the end of the first dentition may be delayed for as long as eighteen months, so that the first teeth appear at two years and the last as late as three and a half years. The individual teeth appear at unusually long intervals; erupt asymmetrically and in atypical order. Particularly in the upper jaw, they are frequently small, soft, easily broken and discolored by caries, to which they are peculiarly liable. They are often frightfully misshaped and foreshortened. There is occasionally an excessive formation of enamel. The temporary teeth show striped or circular erosions at neck and root. The permanent teeth, the germs of which are also affected, show these

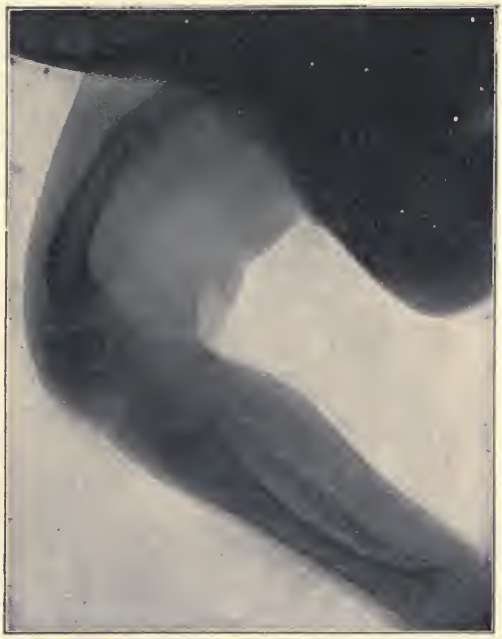


Fig. 46.—Thigh and leg of a three and one-half-year-old child with severe rickets. Infraction and characteristic cupping at the diaphyses.

erosions at the crown. Examination with the Roentgen rays, reveals the small centres of ossification and the abnormal transparency of the bone tissue throughout the entire skeleton, but especially in the wrist and in certain of the long bones. (See Characteristic Roentgenogram, Fig. 46.)

In *the second group*, an important early symptom which may exist, temporarily, alone and may be continuously present, is the so-called craniotabes. Palpation over the occipital bone and over the posterior portions of the parietal bones discovers circumscribed areas of softened bone which can be depressed They are rarely confluent and are about the size of a small coin. The sensation received upon pressing one of these areas may be compared to that which is given in pressing upon parchment, or over the convex surface of an old stiff felt hat.

The continued pressure of the body-weight and the strain of the constant muscle tonus upon the persistently soft bones will inevitably produce characteristic changes in the way of deformities and decurvations in the skeletal framework. Similarly, external violence will cause breaks of continuity much more readily than in the normal structure. Since the bony substance is elastic, it will bend rather than actually break. Even when true fractures do occur the ends usually remain in apposition by virtue of the thickened periosteum. Fractures and infractions are therefore easily mistaken for deformities. True rickitic bone decurvations usually involve a pathologic excess of the physiologic curves. Space will not permit a more detailed description of the varying conditions and possible combinations which these deformities may present. They are of interest chiefly from the orthopedic standpoint. An attempt has been made to present pictures of the most common and most serious deformities. (See Figures 47 to 52.) Special attention is called to the following types: The head shows enlargement of the cranium, with softening of the edges of the bones. The lower jaw is shortened sagittally (trapeziform) and the upper jaw is narrowed (lyraform). The lower alveolar process is directed inward and the upper is directed outward, with such consequences to the teeth as transverse placement of the upper incisors and projection of the lower. The occluding surfaces are but poorly covered by enamel; the palatal arch is high; the zygoma is sharply bent.

FIG. 47.—Two-year-old child showing rickitic deformity of cranium and thorax.

The spinal column is kyphotic, with dextroconvex, bow-like scolioses in the lumbar and thoracic regions, and with compensating curves in other parts. In the production of these deformities the flaccidity of the muscles and the manner of carrying the child have important influence.

The thoracic changes consist in a flattening and inversion of the lateral walls, especially from the third rib down to Harrison's groove at the level of the ensiform process of the sternum. Below this the costal arch is rolled outward; its lower margin is widened, while the upper or cervical rim is narrowed. The lower segments of the sternum and the costal cartilages are pushed outward as a result of the increased negative pressure in inspiration, the direct action of the respiratory musculature on the flexible bony wall, and the distention of the abdomen combining to cause chicken-breast, or a heart-shaped cross section of the thorax.

The pubic angle is spread, the promontory is pushed forward and the

conjugates are shortened, producing a flat pelvis. The neck of the femur is horizontal (coxa vara) to the shaft of the bone, which curves with convexity forward and outward. The tibia is curved forward in its lower third (Saber tibia). Genu valgum or genu varum may be either unilateral, or symmetrically bilateral, or the contrary. Pes valgus occurs.

The upper extremities are usually less deformed than the lower. Multiple infractions of the clavicles, ribs, radius and femur are common.

The Third Group.—The picture of the rickitic deformities of the skeletal framework is finally completed by the enlargments and protuberances upon the flat bones which are incident to the excessive formation of new bone.

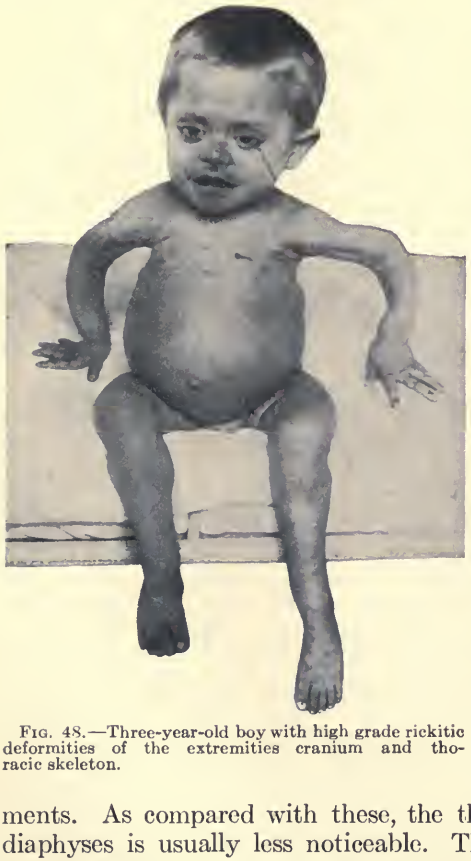


Fig. 48.—Three-year-old boy with high grade rickitic deformities of the extremities cranium and thoracic skeleton.

Rounded deposits of bone, at first fairly circumscribed, are often formed symmetrically upon the frontal and parietal eminences and, more rarely, at the edges of the cranial bones. Between these, the sagittal and coronal sutures are depressed, forming the so-called saddle or cross-bun head. Such protuberances, together with a flattening of the occiput, give the enlarged head a quadrilateral form, the so-called square head, or caput quadratum.

Upon the outer and more often upon the inner surfaces of the costochondral articulations large twin nodules appear. The line of nodules forms a divergent arc, which is called the rickitic rosary.

The epiphyses of the long bones, particularly at the wrist, are the seat of nodular enlargements. As compared with these, the thickening and rounding out of the diaphyses is usually less noticeable. The bones of the wrist, as well as the phalanges, are enlarged at intervals, presenting the appearance of a string of beads. The severe decurvations of the bones and particularly the large calli forming over slowly healing infractions may be mistaken for rickitic osteophytes. While the decurvations, infractions and enlargements of the various parts of the skeleton may present extreme and fantastic malformations in severe or neglected cases, they are ordinarily not excessive and are in part concealed by the fatty panniculus. They may even be entirely absent. The most common and noticeable among them

are the rosary, the disturbances of dentition, the large fontanelles with soft edges, and the nodular enlargements of the epiphyses at the wrist.

The order of appearance of the skeletal changes is synchronous with the normal periods of rapid growth of the several bones (Vierordt). This development occurs earlier in the cranium and the thorax than in the extremities. Therefore, the craniotabes and the rosary are among the first symptoms to appear. The depression of the walls of the thorax and the enlargement of the fontanelle are noticed later. The square head (caput quadratum), the kyphosis, and the changes in the extremities follow only at the end of the first year. The grosser and more permanent deformities of the limbs develop quite late and particularly, of course, when they are functionally employed, as in sitting, creeping and standing. Deformities of the thigh, however, have been found even in infants who were still in the cradle. If rickets occurs later in life craniotabes does not appear.

Most of the clinically recognized changes and disturbances of function attendant upon rickets, appearing in other organs, may be generally interpreted as complications or sequelæ of the disease without compromising the conception that rickets is a general dyscrasia. (Glisson, 1650.)

A direct mechanical relation doubtless exists between the thoracic deformity and certain respiratory disturbances. The flexibility of the bony chest wall limits the excursions of the diaphragm; this, in turn, impairs the ventilation of the lung and lessens, again, the respiratory interchange of gases. The contraction of the diaphragm, instead of promoting the proper expansion of the lungs, draws in the lateral thoracic walls and even the sternum. This results in dyspnœa, tachypnœa, cyanosis, expansion of the nares and diseases of the respiratory tract. The relation between the diminished agility with the increased irritability of the rickitic patient and the pain developed upon active and passive motion and due to the disease of the skeletal framework and musculature, is quite apparent. In the acute stage of the disease, fear, timidity, irritability, and distrust are depicted upon the infant's face. The child is afraid of everyone who approaches the bed and protests either by cries or by peculiar and vigorous motions of the hand, against being touched.

Another group of possible complications includes anemia, marked enlargement of the liver and spleen and general hyperplasia and induration of lymph nodes, tonsils, etc. The anemia is characterized by pallor, oligocytosis, oligochromemia, poikilocytosis, erythrocytosis, lymphocytosis, and increase of the mononuclear cells. The liver and spleen are often displaced downward by the thoracic deformity, a tendency which should not be mistaken for actual enlargement. It is a widely accepted view that these general symptoms are proportional to the severity of the skeletal disease and are relational to the lesions of the bone-marrow, whether primarily so, as Marfan holds, or in a secondary way. (See the pathogenesis of von Jaksch's anemia, etc.)

Rickitic myopathy, to which reference has already been made, is almost always present. It manifests itself in general weakness and lassitude, muscular atony, atrophy and flaccidity, and in an abnormal mobility of the

joints. It is probably primary to a degree. The tempting hypothesis that this myopathy is merely a sequel to severe disease of the skeleton and is conservative, since it relieves the strain upon the bony framework, is supported neither by the structural quality of these muscular changes (Bing, Martins) nor by the facts of their distribution. Nor is such a hypothesis compatible with the involvement of the entire unstriated muscular system, frequently observed and indicated by an abnormal dilatation of the heart and the arteries, by gastro-intestinal atony, obstipation and tympany. Furthermore, other soft parts, such as the skin, the panniculus, etc., are no less flaccid than the muscular tissues.

The impaired mobility and delayed development of the rickitic child readily lead to a delay in its social and mental development, evidenced by a low grade of intelligence, imagination, and a limited vocabulary. Con-

FIG. 49.—Three-year-old girl with rickitic cross-bun head, protrusion of the tuberosities and defects of the temporary teeth.

versely, any previously existing marked degree of feeble-mindedness which tends to confine the child to its bed, may be a cause of increased severity in the course of rickets.

Spasmophilia, often combined with and closely relational to rickets, probably belongs to the group of manifestations coördinate with the skeletal disease.

The onset of rickets is usually announced by a number of premonitory symptoms, the nature of which is not clear. The child becomes listless, restless, peevish. It bores its head deeply into the pillow and wears off all the hair over the occiput by a continual to and fro movement. During sleep a profuse, clammy, acid perspiration, especially about the head, appears, accompanied by numerous sudamina. Transient erythemata and pressure spots are further signs of vasomotor disturbance. The abdomen becomes distended. The urine has an unusually sharp, penetrating odor. The earlier evidences of disease in the skeleton and the musculature are noticed only after days or weeks.

Course, Complications, Termination

The first skeletal symptoms, craniotabes, kyphosis and the rosary may develop rapidly to a serious degree. Otherwise the course of the disease is chronic and often remittent, extending from a period of months up to two years. In every case that survives, spontaneous recovery eventually obtains.

The beginning of recovery is heralded by the disappearance of general symptoms and by the evidences of functional progress, as in attempts at standing, uncertain efforts to walk, etc. The calcification of the skeleton becomes complete in recovered cases. In the course of years, deformities and especially the decurvations of the long bones are often overcome to a surprising degree in the process of growth. Dwarfism, hump-back, scolioses and other malformities, on the other hand, may persist throughout life.

Fig. 50.—Two-year-old child with high grade rickitic deformities especially of the extremities and thorax.

Rickets is never a direct cause of death, but it is very often a contributory factor. The mortality of rickitics is especially great under hospital conditions. Outside of hospitals, the death-rate is greatest among the poor. The most common and serious complications, induced by intercurrent infections, are bronchial catarrh, capillary bronchitis, with or without broncho-pneumonia, severe forms of spasmophilia with eclampsia and laryngospasm, and chronic enteritis of long duration. The gastro-intestinal infections follow milk feeding injuries and are characterized by foul smelling, watery stools and pseudo-ascites, in the course of which the nutrition is reduced to the lowest stage. Other complicating events, as diastasis of the recti, hernia and nystagmus, are of less consequence. The distorted respiratory mechanism, involving the decurvation of the thoracic walls, the continued decubitus and the flaccid and distended abdomen are invitations to pulmonary disorders.

Differential Diagnosis

Even with the appearance of a number of characteristic skeletal and general symptoms which seem to insure a definite and reliable diagnosis of rickets, certain errors are still common. In order to avoid them it should be remembered that congenital forms of disease, of whatsoever type, are never of rickitic nature.

Certain structural imperfections, such as the physiologic decurvation of

the infant leg, are sometimes mistaken for symptoms of rickets. "Fetal rickets" is a misnomer and under this head *osteogenesis imperfecta*, in which the lack of development of the cranial bones simulates craniotabes is often included. The soft bone areas in this condition differ from craniotabes in that they are situated in the temporal bone or near the sagittal suture, and not in the occiput. The fontanelles are sharply circumscribed by hard bone. The deficiency gradually disappears during the period within which rickitic craniotabes makes its appearance.

Congenital myxedema and mongoloid idiocy have so many symptoms strikingly in common with rickets, that many distressing diagnostic errors have been made. In fact, a relationship between these several diseases was, at one time, accepted. The cretinoid face, the low mentality, the dryness of the skin and hair, the gelatinous consistency of the subcutaneous tissue, the macroglossia, the lower trachea destitute of its thyroid covering, are indicative of myxedema. The Mongolian face and the purposeless posturing indicate mongolism. The disturbances of motion in Oppenheim's congenital myatonia, in atonic infantile paralysis, early infantile progressive amyotrophy, Tay-Sach's idiocy, etc., may be confused with rickitic myopathy.

FIG. 51.—Rickitic beaded fingers, two and one-half-year-old girl.

If rickets occurs in combination with scurvy, quite a common event and probably with pathogenic reason, the scurvy may remain concealed. The manifestations of a hemorrhagic diathesis are characteristic of the latter. The presence of painful swellings over the ends of the diaphyses, rather than over the epiphyses, is not always a reliable distinction. Rapid and favorable therapeutic results often substantiate the diagnosis of scurvy in retrospect. To the expert, Roentgenography gives positive evidences.

In cases in which severe endochondral disturbances of ossification, with myopathy, have caused a form of rickitic paralysis due to pain, Parrot's pseudoparalysis may be suggested.

Rickitic kyphosis may be differentiated from the kyphosis of Pott's disease by the fact that in the rickitic the curvature is flatter, is not fixed, and almost always disappears when the patient is laid upon the abdomen.

The pyriform distension of the hydrocephalic cranium may be readily distinguished from the rickitic tête Carré, or square head, caused by the

thickening of the frontal and parietal eminences. The softness of the rickitic cranium, however favors, the development of the hydrocephalic enlargement; while venticular dropsy due to vascular congestion and lymphedema is common in rickets. A true cerebral hypertrophy is also supposedly related to rickets. Periosteal processes on the rickitic cranial bones may be mistaken for chloromata.

With capillary bronchitis it is often impossible to determine whether small lobular pneumonic foci are present. Pulmonic dulness is sometimes simulated by scolioses, by thoracic deformities and by thickening of the scapulæ.

Rickets and osteomalacia, according to recent researches, do not differ in their pathology, but only in the period of their occurrence and probably in the matter of cause. Osteomalacia affects the mature bone; rickets affects the rapidly growing bone. In those rare cases in which the disease occurs between the third year and puberty, or in which it continues, with remissions, throughout childhood, we speak either of delayed rickets (*rickitis tarda*), if the increased formation of osteophytes, the endochondral perversions and the decurvations (coxa vara, genu valgum, pes planus, etc.), are present, or of juvenile osteomalacia if the osteoporosis is the more prominent.

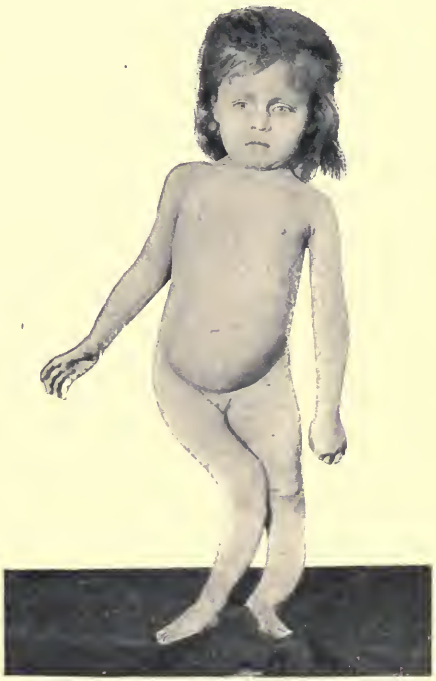


Fig. 52.—Five-year-old girl with deformities of legs following rickets.

Prophylaxis and Therapy.—The measures of prophylaxis are those used in the treatment. By far the most important factors in prevention and care of rickets are moderate stimulation to bodily exercise and stimulation of the body as a whole.

First, we may place the use of active and passive motion, suited to the condition of the musculature and of the skeletal framework. Such exercise cannot be obtained if the child lies continuously upon its back, wrapped in tight diapers infrequently changed. A good nurse may give the growing child sufficient stimulus for mild bodily exercise. She will, at least, give its natural tendency to such exercise free play while bathing and by permitting the child to lie naked on its abdomen. The arrangement of the bed, with a moderately firm flat mattress, proper mode of carrying, sufficient play and creeping all tend to prevent deformity. The child should be not encouraged in too frequent attempts to stand or walk. The Epstein rocking-chair may be used. (See page 103.) In the more severe osteomalacic forms,

which seldom occur save in neglected or irrationally treated cases, extreme caution is, of course, necessary on account of the danger of infractions, even while changing or bathing the child. The use of salt baths and of massage doubtless promotes active and passive motion. It is well to begin these procedures carefully; at first, with a gentle stroking, then by the use of a dry rub; later with warm sponges and finally by tepid baths with salt content increasing up to one per cent., continued for some ten minutes; all these measures being constantly controlled by the so-called "reaction" of the skin (Heubner).

It is also extremely important that the little patients are continuously in the fresh air in sunny, sheltered places. This, of course, is accomplished very satisfactorily by a visit to localities of especially favorable climate. Children can be protected against low temperature by woolen clothing. For children in the second and third years who are not severely anemic, high altitudes and mountain resorts may be considered. In the city, every sunny hour during the day should be properly used in keeping the child in the yard or garden, or on the veranda in an open crib or baby carriage. During the day the child should, if possible sleep in the open and at night in a freely ventilated room. During the summer months, carefully administered sun baths lasting at first for only two to three minutes may be of great value. Artificial light is said to be very efficacious and is not as irregular as the sunlight. Ultra-violet ray has been suggested.

The observation that the treatment of rickets in hospitals usually accomplishes but little, in spite of the common use of phosphorus and cod-liver oil, probably turns upon the fact that these two determining factors of fresh air and exercise have, until recently, received too little consideration.

As to the dietary of rickets, it is at present quite certain that no régime, not even rationally conducted breast feeding, is a positive prophylactic against the disease, even in its milder forms. Further, it is known that every sort of feeding, including that with breast-milk, which encourages excessively rapid growth, favors the appearance of the disease and increases its severity, suggesting a disturbed relation between organic and non-organic growth. It is a generally accepted fact that a limited dietary but slightly denaturized, moderate in quantity and of sufficiently varied quality is the most desirable in this condition. So that in threatened or acute rickets, we should vary from the rational methods of feeding for healthy children, suggested elsewhere in this work, only in so far that we may select carefully prepared additions of soup and bread and of various freshly boiled vegetables at an earlier date than usual. If these additions are well borne, which is not invariably the case, they may be given together with a limited quantity of milk, not exceeding one pint a day. Buttermilk frequently gives better results. The reduction of the supply of milk, together with careful hygienic care, is the best method of overcoming anorexia.

In chickens and animals that are fed phosphorus, the bones become sclerotic. On the strength of these observations, Wagner recommended phosphorus in the treatment of rickets and Kassowitz found it very useful. These experiments, however, were made upon animals and upon

non-rickitic animals at that. The effect produced in them was probably the opposite of osteoporosis. But this condition plays a secondary rôle among the phenomena of rickets and is one of those manifestations which do not suggest the etiologic character of the disease. From this point of view, therefore, the phosphorus therapy cannot be regarded as scientific or as in any way relational to the cause of the disease (Stoeltzner).

This is true of phosphorus in emulsion of almond oil and gum acacia as first used by Kassowitz. Because this emulsion would not keep, it was replaced by the solution of phosphorus in cod-liver oil. This mixture has proved its value empirically but there is much question as to which of the two constituents is the more effective. The customary prescription is as follows:

Olcii Phosphorati	2.0 (40 minims)
Oleii Morrhuæ	200.0 (8 ounces)

The phosphorated oil is a one per cent. solution of phosphorus and the above prescription gives one part phosphorus in 10,000 parts of the oil. Other forms in which phosphorus may be prescribed seem much less active or otherwise less useful. The "non-purified" cod-liver oil is recommended. It should be protected from sunlight and other oxidizing influences. The dosage is four c.c. (one teaspoonful) twice daily and must be kept up for several months.

During the period of recovery, the calcium and phosphorus requirement is naturally increased. In view of this increase, and not because of the older view that the rickets was caused by insufficient lime in the food, the addition of calcium and phosphorus salts have been, recently, highly recommended. Cow's milk is a food with a high content of these salts but in certain cases the fat obstructs the calcium retention. The benefit of these salts is hardly great enough to pay their purchase price, especially if the all important factors of general hygiene, diet, and fresh air are neglected because of their use.

Organotherapy has not proved of sufficient value to necessitate discussion at present.

The recent work on the accessory food substances, the vitamins, shows that cod-liver oil contains a large amount of the anti-rickitic element, bearing out the previously empirical use of this treatment. Numerous authors now recommend the use of the oil without the phosphorus.

The use of emulsions, while it greatly facilitates the administration, is to be avoided because of the small percentage of cod-liver oil contained. Very few contain over fifty per cent. and most commercial preparations are even lower.

Early orthopedic interference, to prevent deformity, is inadvisable because of the necessary immobilization which could only result in further demineralization and softening. In severe angular deformities, however, interference may be imperative even in infants but usually braces, etc., should not be applied until school age is reached. In some cases, the further

demineralization and softening produced by plaster dressings may be taken advantage of to soften the bone. Usually about six weeks will soften the bone sufficiently to permit molding by extension and massage.

DIABETES MELLITUS[7]

The disease which is termed true diabetes mellitus occurs much more rarely in children than in adults. It is extremely rare in the first years of life, but is somewhat more frequent in the second decade. True infantile diabetes is commonly supposed to be of pancreatic origin and closely related in its nature to that which develops in the adult. The observations of pediatrists largely sustain the view that the condition may be considered an hereditary endogenous degeneration. Degenerative factors, such as parental lues and various injuries due to intermarriage and to the presence of certain homogeneous stigmata, frequently appear. Few of these cases come from parents of the middle class.

It is commonly said that the course of diabetes in childhood is uncomplicated, but rapidly fatal. That it is ordinarily under observation for a short period is probably due in part to the fact that the condition, because of its rarity in children, is not suspected and for this reason, is diagnosed relatively late in its course. Its severe stages may have been preceded by disturbances of metabolism extending over a long period, without exhibiting any marked subjective or objective signs. The transition to more rapid progress may be determined by some infectious disease or other injury. Only when the tolerance for carbohydrates quickly decreases or has already been greatly reduced, so as to cause glycosuria with a minimal absorption of sugar, does the disease become noticeable. Loss of weight, weakness, lassitude, occasional pains in the limbs, marked polydipsia and diuresis, often leading to enuresis and irritation of the external genitals, are its indicators. The skin is dry and irritable. Skin affections, as urticaria and pyodermia, occur less frequently than in the adult. Very often, the absence of any definite sign in a more or less pronounced illness calls attention to the possibility of diabetes and suggests an examination of the urine by means of which the diagnosis is established. The acetone odor may also serve as a guide.

In the daily quantity of urine, which may amount to from 3 to 6 litres, we may find several grams of acetone and ammonia (ammonia coefficient, 30-40 per cent.), large quantities of acetoacetic acid and oxybutyric acid and, at times, from 100 to 300 grams (2-8 per cent.), of glucose. In advance cases, protein and casts are usually found.

Actual or pseudo-melituria of a non-diabetic character occur much more frequently in children, and especially in very young children, than in adults. In the majority of these cases it is not a matter of glycosuria, but of the output, rather, of other kinds of sugar or of other reducing substances, the nature of which is not yet fully understood. The most common melituria in the infant appears to be a lactosuria; that is, the excretion of uncoverted sugar of milk. Occasionally a galactosuria occurs. The common occasion

[7] For the pathogenesis of this disease, see the literature of internal medicine. Certain peculiarities of the symptomatology of infantile diabetes are briefly considered in this work.

of these disorders is some disturbance of nutrition. Many children, however, of the lymphatic type, in the course of constitutional diseases, but without manifest disturbance of nutrition, excrete substances which give reactions for the sugar group.[8] Furthermore, the acute infections predispose children to melituria, usually of an alimentary type. We are apt to refer the fact to a lowered limit of assimilation of sugar, failing to take into account the truth that in the present state of our knowledge of the renal excretion of the various forms of sugar, whether monosaccharides or disaccharides, we must recognize more than one fundamental form of disturbance of carbohydrate-metabolism. Neither a continuance of glycosuria for several days or weeks, nor reliable evidences of acidosis, of which the mere increase of the ammonia coefficient is not adequate, justify a diagnosis of diabetes in these cases. Only an habitual and progressive glycosuria, a true excretion of dextrose with a decreasing carbohydrate tolerance, associated with general symptoms, are conclusive.

The termination of diabetes in children, practically always fatal, is preceded by very definite signs of true acidosis, such as vomiting, acetone breath, jactitation, and coma lasting for two or three days. The average duration of the disease is given as three to six years, while its more severe period lasts from one and a half to three years (v. Noorden).

The treatment of diabetes in childhood cannot cure the disease, but may prolong life. It is essentially dietetic. Watching with the greatest care the most important indications, we are able to recede from that absolutely rigid withdrawal of carbohydrates which is instituted to prevent glycemia, and thus to avoid the changes of autophagic or excessive combustion of the body fat (acid poisoning). In the treatment of acidosis, it is customary to attempt neutralization by the use of such alkalies as sodium bicarbonate or citrate in teaspoonful doses, in lemon juice. This treatment is not always successful and is purely symptomatic. In employing it, the danger of the occurrence of edema must be kept in mind. The dislike of the patient for an exclusively meat and fat diet sometimes becomes urgent.

Oatmeal is not only better borne by diabetics than are other starches or sugars, but is occasionally found to increase the carbohydrate tolerance. Van Noorden's dietary of oat-products is based upon this supposition. It consists of a diet of 100 to 200 grams of oatmeal gruel, 200 to 300 grams of butter, and several eggs daily for a period of one or two weeks. I have seen surprising results even in severe cases, but the gains are not permanent. The same benefit is often achieved with mashed potatoes. Inulin and hediosit are harmless but expensive carbohydrates. Edibles free from or poor in carbohydrates are meat and meat broths, ham and bacon, aspic, green vegetables of all sorts, cauliflower, cheese, cream, sour milk, and the various factorial food preparations for diabetics. The statement of the manufacturers concerning the carbohydrate content of these prepared foods, is, however, frequently open to doubt. Recently, protein milk has received some consideration. Relatively large doses of alcohol have been recommended (100 grams per day).

[8] Aschenheim and others.

OBESITY

Obesity is a symptom which, in extreme form, is quite rare; but in milder degree is very often met with among children, especially in later childhood. It may be produced by various causes. The most frequent type is the purely ectogenous or alimentary obesity caused by the use of excessively rich food, especially in the form of carbohydrates and fats. Inquiry into the child's history in this regard, shows that its food requirement has been over-estimated by the parents. The child is often actually overfed even though the fact be strenuously denied. Special attention should be paid to the habit of eating between meals, to the use of milk as a beverage, to a dietary of sweets, artificial foods, etc. Alcoholism, to the frequency of which in childhood Hecker and others have testified, must also be considered. Both the laity and physicians, learning of the low nutritive value of alcohol, fail to include it as a heat-producing material in their caloric estimates; although beer, for instance, represents a caloric value two-thirds that of milk.

In another class of cases, the quantity of food is only relatively in excess; that is, in proportion to the abnormally small amount of muscular work and bodily exercise undertaken by the child. This may be due to laziness caused by heredity, training or example, etc., and is comparable to the fattening of the animals for market; or it may be caused by disease which in its course develops myopathic or skeletal disorders, pain upon muscular contraction, etc. (rickets, paralyses). In this group the signs of basic disease are often hidden beneath the obesity. It may occur, for instance, in certain endogenous forms of nerve disease, as in the hereditary ataxia of Werdnig-Hoffmann. Such cases represent a transition to true endogenous obesity, which alone may be termed a true adiposis.

These adiposes are etiologically divisible into two types; those, on the one hand, which constitute a phenomenon of hypo- or athyroidea; and, those, on the other hand, which are sequela to hypoplasias and to functional errors in the germinal organs (castration, genital infantilism, etc). The absence or functional incapacity of these glands of internal secretion leads to a retardation of the metabolism, a result experimentally proven. Of similar origin are those forms of endogenous obesity which, developing wholly without symptoms, offer no definite points of departure for etiologic study. With respect to the pathogenesis of these conditions clinical opinion has for centuries favored the idea of an hereditary constitutional anomaly, of a reversion of cell function due to a reduction of the total metabolic values of the germinal cell. For a long time, this view could not be sustained by experimental observation. Evidently suitable cases had failed of analysis —those exceptional cases which maintain their excessive weight upon an extraordinarily small amount of food; in which, for instance, an energy quotient of seventeen under bodily exercise and of twelve under rest, being about one-third of the normal standard, persists for weeks and months. Very recently, however, it has been actually determined that among these adiposes are individuals, some of them young and without myxedema or

genital infantilism, who have an habitually reduced metabolism, who actually "use less fuel" than others (v. Bergmann).

Finally, we must consider as a pathogenic possibility a special disturbance of fat metabolism. If it be true that certain lipolytic ferments of the lymphocytes are concerned in the catabolism of the body fat, the relation of obesity to certain systemic diseases of the lymphatic organs, such as hypoplasia and hyperplasia would be explained in accord with clinical observations.

The treatment of obesity in children must take this factor into account. Therapeutic measures addressed to cause are effective in many cases in which the condition is brought on by overfeeding, alcoholism, irregular living, or hypothyreosis. In the matter of diet, caution must be exercised to avoid the loss of nitrogen. For this reason, the use of preparations of the thyroid gland, when the gland is functionating normally, is generally contraindicated. According to Hellesen it is more difficult to prevent loss of nitrogen in the dietetic treatment of obesity in children than it is in adults. The total food supply estimated by its energy value, should never be reduced below fifty calories per kilogram of net body-weight when at rest. The body-weight to be reckoned in such estimates should be the mean of the individual's present weight and his normal weight for his given height. A bulky diet generous enough to satisfy the appetite and containing ample nitrogen, water and cellulose, but poor in fats and carbohydrates, should be selected. It is not necessary to curtail the quantity of water, as the obese person loses more water during exercise than the normal individual.

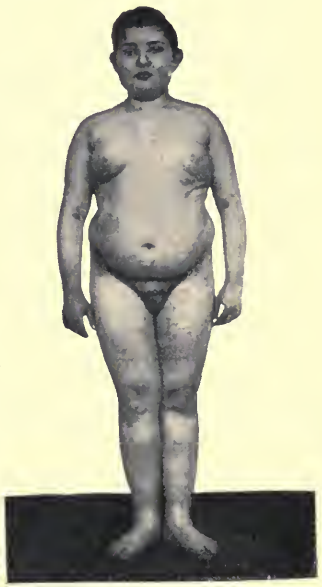


Fig. 53.—Obesity, twelve-year-old boy. (University Children's Hospital, Breslau, Prof. Tobler.)

For older children the following regimen may be suggested. At noon, an abundant allowance of broth; later a meal consisting of meat and vegetables (cabbage, spinach, turnips, etc.) and for the rest of the meals skimmed or preferably separated milk, or tea sweetened with benzosulphinidum (saccharin), bread, a little honey and fruits. Water may be given freely. Active, but regulated physical exercise should be prescribed. In individual cases an exact estimate of the food taken should give its caloric value.

UNDER-NOURISHED OR FRAIL CHILDREN

The question of persisting leanness or even emaciation in children is frequently met with in practice. A discussion of the causation of this condition must, however, take innumerable factors into consideration. In the first place, the persisting leanness may be the result of a long wasting disease

from which the patient has recovered but never got back to his full robust energy. Chief among such conditions is tuberculosis with its variable manifestations and its slow recovery. Then chronic toxic infectious injuries to the nutritive apparatus may be rated. In the second place, the cause of emaciation may be due to the food itself. The starved looking urchin may never have had enough to eat or the food may have been poor in quality and wretchedly prepared. Then again, there may be certain mechanical interferences with the assimilation of the food. Among these we may have pyloric stenosis, cardiospasm, and esophageal obstruction, with persistent vomiting and wreching.

Inanition occurs frequently in infants, not by any fault of the mother, but as a result of poorly functioning breasts, or of insufficient artificial food at times on the advice of the physician or commonly upon following the directions on the box of some patent infant-food.

However, all of the above factors, which really require no further discussion, cover only a small percentage of the large number of children that can be classed as "under-nourished." Indeed it can easily be shown that these children do not react to food as one would expect of a hungry child. Additions to the diet do not result in any marked increase in weight. Nor do they present the characteristic objective signs of inanition.

Even this class of lean or frail children is not an entity. After eliminating those cases in which the leanness exists only in the imagination of the mother whose ideal of childhood is the fat pasty infant, or who interprets the period of growth in height as an abnormal phenomenon, we still have those children in whom there is a true lack of fat (lean in the narrowest sense), those who are poorly muscled, and those that are especially small boned (asthenic).

In our present system of education and training, the development of the musculature by proper exercise is often sadly neglected, especially in children from six to twelve years of age. This, naturally, results in boys and girls with small legs and arms even though the panniculus is fairly abundant. This child, because of insufficient exercise, requires little food and is supposed to be sick. Others are lean because of actual anorexia. In such, the anorexia may be due to a vitiated taste, spoiled by highly flavored candies and desserts, spiced foods and carbonated drinks. Protracted anorexia brings with it other unpleasant symptoms such as dulness, restlessness, disobedience and lack of concentration at lessons. Grouped together these manifestations are often called "school sickness."

Still another group of the so-called "frail" children is found in the high-strung, fretful, restless, neuropath. They may be individuals of distinctly abnormal bodily and mental excitability, born as such or brought up in this way by the constant association with members of the family similarly afflicted. Of weak will-power, they are urged on physically and subjected to outbursts of temper, etc., until they themselves become neuropathic. These stimuli can often be traced into early influences.

A certain stress must also be laid upon heredity. It is not at all uncommon to find that the father or mother presents the same characteristics of

growth as does the child. Or one of the parents may have been of a similar build at the same age. There is no doubt that the failure of panniculus, etc., is as transmissible as obesity. In connection with this, attention must be drawn to the fact that the association of hereditary leanness, with varying degrees of stigmata of degeneration, is not at all uncommon. We may indeed speak of a "degeneration form of frailness."

Remembering the discussion under obesity concerning children who are fat because of a lower metabolism (slow oxidation) it may be assumed that there are individuals who have a habitually more active metabolism and a resulting leanness. This, of course, has not been shown by metabolism test.

Finally, it is definitely known that there are children that do not thrive upon a diet upon which others of the same age and in the same surroundings grow at the accepted normal rate.

Treatment

The frail children of the well-to-do regularly become the sacrifices of the rather "hypertrophic" industry of advertised foods. The discussion of the causation of the condition is really quite enough to lead the physician into proper therapeutic paths. Careful scrutiny of the dietary with corrections, giving simple but sufficient nourishment, should not be neglected.

PECULIAR PREDISPOSITIONS TO DISEASE (DIATHESES); AND CONSTITUTIONAL ANOMALIES

In certain children a remarkably frequent appearance of peculiar disturbances of health is observed and this in the face of the exercise of the greatest care in the avoidance of injuries which are recognized as standing in causative relation to such disorders. In view of this evident fact, we are forced to conclude that in such children some organic peculiarity exists, a special disposition or a favorable soil, as it were, for the agents of disturbance. Instead of the phrase "predisposition to disease," the Greek term diathesis has come into common use. The abnormalities to which the organism may show such predisposition are of varying character. They may belong, for instance, to the group of catarrhal affections of the mucous membranes and the skin, or to forms characterized by swelling of the lymph nodes; or to the type of neuropathic disorders. We may speak, therefore, of a catarrhal diathesis, of a lymphatic diathesis or of a neuropathic diathesis without standing committed to any hypothesis, but expressing simply facts supported by numerous observations. Such a diathesis is *not a disease*, but merely a state of predisposition to certain forms of disease. The principle of this conception has been often disputed, but never logically disproved. The elements which determine the diathesis induce manifestations which often differ but slightly from, or are closely identical with, those which figure in the diseases of individuals not so predisposed. In other cases, again, these manifestations represent not only quantitative, but qualitative reactions of the organism to pathologic irritation which are essentially abnormal or illegimate. They exhibit, indeed, certain stigmata, which call

the attention of the experienced observer to constitutional peculiarities which may underlie the condition.

All diatheses, however, are not definitely latent. There are predispositions to disease which are continually manifest, whether as persistent sequelæ to distinct attacks of disease, or as externally recognized deviations of structure or quality from the normal habitus. The term constitutional anomaly is often applied to this type of cases.

A further diathetic fact is that different predispositions to disease may be combined in one individual. Thus we frequently find the predisposition to catarrhal disease associated with the tendency to involvement of the lymphatic tissues. In these cases, moreover, the swelling of the lymph nodes cannot be regarded as a consequence of the catarrhal condition of the skin and mucous membranes. The two states are rather in large measure coordinate to each other; and the fact justifies us in recognizing a combined catarrhal and lymphatic diathesis. Frequently another member of the diathetic group may be associated, *viz.*, the predisposition to nervous disease giving a combined neurolymphatic diathesis. Desirable as it may be to make advances in this difficult field by distinct classifications and divisions, it must not be forgotten in the desire for a schematic presentation of the subject, that as every observer knows from his own experience, that combined diatheses are frequently met with in practice.

THE INFLAMMATORY OR EXUDATIVE DIATHESIS

For the pediatrist this is the most important of all the abnormalities belonging in this group. The condition was described by Theo. White, among others, in 1782 and has recently been reviewed by Czerny.

Occurrence.—The exudative diathesis is a widely distributed condition and in frequency can be compared with rickets. It is recognized more often than rickets by the physician because its manifestations continue for a longer period. The tendency is familiar. Heredity, especially through the mother, plays an important part; exhibiting itself chiefly in homologous, but, also, in heterologous types.

Initial Appearances.—The condition is not discoverable usually in the new-born. Apparently the maternal organism exerts a sort of protective influence, as it does in certain disturbances of the organs of internal secretion, for example, in aplasia of the thyroid. The smallness and delicacy of the child are no more indications of the diathesis than is a long lock of hair standing on end over the coronal suture. Suspicion may, however; be aroused by delayed reaction from the initial weight-loss following birth, or by an early flattening of the weight-curve in spite of an adequate supply of natural food. This may suggest a combination of the diathesis with disturbances of growth and development. Such a diagnosis must be made, however, with exceeding care.

Characteristic evidences of this predisposition to inflammatory disease and to changes in the composition of the body as a whole usually appear only after the first few weeks.

1. The habitus of the diathesis is variable. Two types are recognized; that of the delicate, weak, exudative patient and that of the large, apparently strong and fat, but muscularly weak child. The latter type is frequently met with in a singular variety, to which A. Paltauf has applied the term: *status thymico-lymphaticus*. This type tends to become pathologically fat and shows a pallor due rather to ischemia than to true anemia, and in part due to the increased water content and the reduced turgor of the subcutaneous tissues. This form is also called the "pasty" habitus.

2. A more or less constant hyperplasia of the thymus and of the lymph nodes is usually associated with the latter habitus. Its clinical signs are as follows: moderately hard, palpable tumors of the lymph nodes in the neck, over the inner aspect of the joints and perhaps, in the abdomen; an enlargement of the spleen, a visible hyperplasia of the faucial and pharyngeal tonsils, with a redundancy of the circumvallate papillæ.

Infantilism is of less frequent appearance and is usually first noticed in older children. It consists in delay of the development of the body in point of size and in the secondary sexual characteristics.

3. Certain manifestations of the diathesis appear upon the surfaces of the body and are apt to have certain sequelæ and complications. The following table gives an outline of these tendencies.

Manifestations of the Exudative Diathesis

Seat	Primary forms	Secondary forms	Sequelæ and complications usually of "nervous" quality
The skin.	Seborrhœa capitis, crusta lactea, intertrigo, prurigo.	Eczema, impetigo, abscesses.	Severe itching, great restlessness, disturbed sleep, readily induced fright.
The mucous membranes.	Transient desquamation and turgescence in various regions	Exudative processes. Palatal angina, pharyngitis.	Hyperpyrexia, cough, vomiting, anorexia.
		Gastro enteritis.	Vomiting, pylorospasm, colic, obstipation, mucomembranous diarrhœa.
		Coryza, laryngitis, bronchitis, bronchiolitis.	Hay fever, pseudo cough, convulsive cough, bronchial asthma.
		Conjunctivitis, phlyctenulæ, blepharitis.	blepharospasm.
		Balanitis, vulvovaginitis, cystitis.	dysuria, ischuria.
The lymphatic organs.	Hyperplasia of the faucial and pharyngeal tonsils. Hyperplasia of the palpable lymph nodes in the neck and at the joints.		

The appearance of milk-crusts, seborrhœic deposits, and intertrigo is usual in the first year. Prurigo [9] may occur later. Anemic catarrhal affections and the hyperplasia of the lymph nodes which occur throughout infancy and childhood may also be present, to a degree, in the nursing infant.

The milk-crust or vesicular eczema of the scalp appears as an exudate in the form of dark gray or brown scales which become firmly attached to the epidermis. The skin beneath them is of bright red color and moist. Ec-

FIG. 54.—Pasty habitus, especially in the face. Beginning eczematous eruption.

zema rubrum with a white bran-like desquamation occurs in sharply circumscribed triangular areas of reddened and thickened epithelium upon the cheeks.

Intertrigo or chafing, which in healthy but neglected children occurs around the anus and the genitalia, develops in others also behind the

[9] Prurigo occurring in lean children, forms diffuse, hard yellow itching nodules (*lichen*); which, in fat children, are preceded by red papules (*urticaria rubra*). These lesions develop chiefly on the nates, the body, the legs and arms, but hardly ever on the face. The term lichen urticatus or strophulus is often confused with the quite different prurigo of Hebra.

ears, in the wrinkles of the neck and in the folds of the joints. In any of these skin conditions, scratching and uncleanliness may cause infection, and the appearance, in consequence, of a severe, obstinate, wet, impetiginous eczema

Geographical tongue is caused by exudations occurring in striæ, over the upper surface of the tongue. Prominence of the papillæ and desquamation of the epithelium give the characteristic white color to these lines.

Primary disorders of the skin and mucous membranes are probably of identical nature. The process consists essentially of a superficial desquamation and an accompanying exudation. The definite localization of these lesions, suggestive of local injury or dystrophy, and their appearance as distinct attacks without any apparent external cause are especially characteristic. The lesions appear singly or in the most variable relation. Eczema rubrum, prurigo and especially the geographical tongue are transitory and change their appearance daily.

Czerny claims that the catarrhal manifestation of the diathesis are analogous to the eczemata and may also be laid to infections for which the soil has been prepared by the irritable condition of the mucous membranes. Numerous observers note catarrhal affections of the mucosa of the digestive tract which are quite different and of early occurrence. They are indicated by diarrhœa, muco- and sanguino-purulent stools with eosinophilic cells. They are not attended by severe general symptoms. Obstipation and a fetid breath usually accompany the throat conditions. In half of the "exudative infants" Lust finds an organized urinary sediment, consisting of epithelial cells and leucocytes, arising from a desquamative process which runs a definite course and increases the disposition to infectious catarrhs. (Colon pyelocystitis.)

These infectious processes in the skin and mucous membranes increase in a degree the hyperplasia of neighboring lymph nodes and of the tonsils; a hyperplasia which in these diatheses may be produced, also, by alimentary injuries, etc. The marked and permanent hyperplasiæ of the faucial and pharyngeal tonsils and their sequelæ, discussed in detail in other parts of this work, arise in this manner.

4. An increased liability to disease and a heightened reaction to causes of irritation, which the exudative diathesis carries with it, may be manifested also in other functional systems. Children so affected are commonly prone to disorders of metabolism; they are disposed to disturbances of nutrition of varying degree, to spasmophilic phenomena and to functional and structural changes in the vascular mechanism, in the way of palpitation, cardiac dyspnœa, and dilatation and hypertrophy of the heart.

In the status thymico-lymphaticus sudden death, primarily due to heart failure, often occurs. It may follow immediately after birth or happen at a later period without any evident cause or upon such slight provocation as overfeeding, anesthesia or the excitement attendant upon a slight operation, especially when this involves the throat, or upon the application of bandages, the use of baths or other hydrotherapeutic procedures.

In all cases in which death occurs without preceding illness, the thymus

is found to be relatively large. It is an organ especially affected in all the retrogressive changes of infancy. It has been suggested that the measurements of the thymus taken in event of thymic death were normal and that they were counted abnormal only because they were compared with the measurement of the organ taken from cachectic bodies. This error may have entered into the estimate occasionally, but, nevertheless, in the condition under discussion of enlargement of the thymus is definitely established by accurate observations. The organ may be said to be enlarged when it weighs more than twenty grams in infancy and more than thirty grams in early childhood.

Where very young children have suffered from tetany and laryngospasm for a time, or when they have gradually developed symptoms resembling intoxication, death comes less unexpectedly.

The ultimate cause of death in cases of status thymico-lymphaticus has not been determined. The presence of a thymic tumor has been especially considered. It appears, however, that the mechanical pressure of the enlarged organ involving the trachea, bronchi, nerves, blood-vessels, or esophagus, does not often play an important part. We are inclined to look upon the result, as one arising from a pathologic secretion from the thymus, a hyper- or hypo-thymusation, which is not necessarily associated with enlargement of the organ. The more recent view is that enlargement of the lymphatic glands or of the thymus is not the actual cause of this sudden death, but rather that it is a coördinative consequence of a severe, even though latent general disturbance.

All of the manifestations of the exudative diathesis continue, usually, but with longer or shorter interruptions, up to the tenth to the fourteenth year. Early childhood is the most severely affected period. Toward puberty a recession of symptoms is usually noticeable. Continuance or relapse after puberty is rare.

Diagnosis.—The experienced observer is often able to make the diagnosis of an exudative diathesis instantly from the habitus of the patient, or from the characteristic tendency to illness arising spontaneously or from a very slight injury.

Possible findings in the body fluids in the way of relative lymphocytosis, eosinophilia, dietetic hyperglycemia, and melituria are of very limited value from a diagnostic standpoint. This is also true of the test devised by Rachmilewitsch, which consists in the formation of papules when a skin lesion is irritated by the application of mustard paste.

The value of findings by percussion or by the Roentgen ray, in demonstrating an enlargement of the thymus, either by dulness or by a shadow deepening to the left, must be carefully weighed. A valuable diagnostic point is found in inspection of the follicles at the back of the tongue.

Ultimate Nature.—The various hypotheses as to the nature of the predisposition to inflammatory disease are more or less vague. The theories of a primary anomaly of metabolism, of chemical malformation, of endogenous food injuries, of disturbances in the regulation of the water content of the tissues are among them, and these have been recently attacked.

Aschenheim and Tomono could not support the theory of Eppinger and Hess that the exudative diathesis is based upon a vagus hypertonia. The careful investigations of H. and L. Hirschfeld show, moreover, that the serum of exudative patients has a marked vasoconstrictive action. Similarly, according to Samelson, the therapeutic results obtained by Krasnogorski with atropin, which apparently gave support to the vagus hypertonia theory, are problematic. Nevertheless, there is something tangible in the proposed relation between the status lymphaticus and this condition

The exudative diathesis has a demonstrable structural basis, in a definite overgrowth of the several parts of the lymphatic system. This hyperplasia affects the lymph nodes of the neck, the axilla, the prevertebral chain, the intestinal submucosa, the mesentery, the spleen, the follicles of the nasopharynx, the base of the tongue and the gums.

Together with this general hyperplasia, there is also a new formation of lymph follicles in the liver, thyroid and bone-marrow, and, finally, an enlargement of the thymus. This enlargement, according to Schridde, consists of a hyperplasia of the medulla and a hypoplasia of the cortex of the organ, with atypical corpuscles of Hassal. As general manifestations we note a characteristic pallor of the skin and an increase of the panniculus adiposus. Other, but inconstant findings, are a hypoplasia of the vascular system, of the chromaffin system (the suprarenal medulla and the solar plexus), of the cerebrospinal and genital systems and, lastly, various malformations which result from a general developmental failure.

According to Bartel and Stein's preliminary reports, the cause of the changes in the lymph nodes is a failure in the development of the lymph channels and of the medullary striæ of the lymph nodes, with a proliferation of the zone of cortical follicles in the first stage of growth and a secondary atrophy of their specific parenchyma. The atrophy of the remaining lymphoid tissue is probably compensatory. The process may be described as a developmental error, the first results of which appear in the connective tissues and directly involve the lymph nodes. These results, accordingly are the expression of a general hypoplastic tendency in the genesis of which the hypoplasia of the vascular system, possibly, plays a primary and fundamental rôle.

This conception agrees with that which the author had reached from a different point of approach. A congenital, or a true hereditary weakness of various organic systems reduces their capacity and their resistance. This, in turn, results in an increased and qualitatively different reaction to natural stimuli which, under ordinary circumstances, would not be pathogenic, but with this diathesis, provoke the manifestations of disease. According to its capacity to respond to demands made upon it, the weakened parenchyma may be strengthened by exercise or injured by excessive use. In the above case, we may have also accompanying the general connective tissue, diatheses, multiple scleroses in the various endocrine organs causing deficiency disturbances (pluriglandular insufficiency of Wiesel).

Exudative infants constitute a pathologic species of unnumbered variety. The combined diatheses break up into component predispositions, each of which depends upon the functional weakness of a particular system and results in the tendency to a definite symptom-complex. From tests of the functional capacity of affected organs, criteria of the component predispositions may be obtained.

The injuries which produce inflammatory conditions upon the basis of this existing increased predisposition to disease are very many. Irrational forms of dietary and particularly those which are popularly termed "strengthening," but are merely causative of the excessive formation of fat, are among the most active of these injuries and that irrespective of the preponderance of their protein, fat or carbohydrate components. According to Czerny, the most injurious consequence of such a fattening process is the increased disposition it affords to secondary infections, which favor the development of severe and often dangerous secondary changes. It is an accepted fact that dietetic injuries produce disease symptoms more readily when they occur in patients who are subject to this complex abnormality which we term the exudative diathesis. Too great stress, however, must not be put upon the effects of overfeeding in the pathogenesis of these disturbances. The writer discerns a tendency to exaggerate their importance when authorities call attention to the striking and constant influence which the manner of feeding has upon individual symptoms and regard this alleged fact as a criterion in the recognition of the picture of a diathesis.

As factors which may convert a latent diathesis into an active process with characteristic signs of its presence, we must reckon with certain intercurrent infectious diseases, as tuberculosis and measles. Even vaccination, the application of the tuberculin test, or the accident of an insect bite may serve as the spark which lights up the potential tendency. With the predisposing hypersensitivity of the body surface, mechanical, thermic, actinic and sensory irritants may act as the agents of injury.

Exudative diathesis and the status lymphaticus are frequently but not inevitably associated. Their relationship is shown in the fact of their frequent reaction to similar influences, as seen in their tendency to improve under the dietetic measures prescribed. Czerny and others consider the status lymphaticus as a component element in the severer forms of exudative diathesis. Unquestionably, the latter stands in close association with certain neuro—and psychopathies. The irritability and instability of the nervous system of children of exudative diathesis, exerting, as it does, a distinct influence upon the severity of the disease complex, (see table, page 215) may be in the nature of a vasomotor unbalance which serves as the basis of all its manifestations (Moro) and is, in certain cases, of inherited or congenital quality. In others the neurosis of the patient is an acquired feature and often results from errors in training, for the indulgence of which the exudative diathesis, with its repeated exhibitions of ill health, has given ample opportunity.

ARTHRITISM IN CHILDHOOD

Quite recently and contrary to preëxisting beliefs, a relationship between the exudative diathesis of children and the uric acid diathesis of adults, is accepted as a probability in view of the demonstration of an infantile disturbance of uric acid excretion. (Kern, Uffenheimer.) The condition which forms the clinical connecting link between them is *arthritism*, a condition which Comby defines as an hereditary and habitual disturbance of nutrition. This phenomenon is a widely distributed one, but is not observed in all classes of society and in all places. It occurs chiefly in the well-to-do families of city life, among the hypercivilized and the highly intellectual of the people. Very few cases are seen in the free dispensaries, while private practice among the wealthy furnishes numerous examples. Homologous and heterologous types of hereditary transmission are distinctly recognized in this expression of disease. According to Comby, the members of an individual family may be subject to diabetes, gout, obesity, renal calculi, migraine, asthma, and the various neuropathies and psychopathies, known as the group of Bouchard's bradytrophy. Even though the beginning of the disease may be traced back to the early history of the child, most of these cases are first recognized at school age.

The habitus of these children is variable. A distinct division into the various types, such as the anemic, erethismic, plethoric and obese, is neither possible nor practical. Very frequently we find cases representing successive transitions, or mixed cases, or even those in whom the external habitus does not differ from the normal.

As we have already said, the manifestations of the exudative diathesis are very numerous, but from the combination of symptomatic elements extremely variable pictures arise. According to reports in the French literature these manifestations may include the entire symptom-complex of the inflammatory diathesis if we consider that this is not confined to the first months or even years of life, excepting as to such incidents as seborrhœa, intertrigo, cradle-cap, etc. The recurring catarrhal disorders and the nervous conditions connected with them (see table page 215) are the most conspicuous. To these may be added a large number of symptoms of greater or less note. Definite rises of temperature, resembling those of malaria, but without apparent cause, with a persistent increase of the rectal temperature to about 38° C. (100.4 F.) especially after exercise; changes of color in the face, transient erythemata, cold hands and feet, chilblains, sweats, fainting, etc.; tachycardia, palpitation, habitual arhythmia, cardiac asthenia and dilatation, diminished blood-pressure, accidental heart murmur and venous bruits; affections of the upper air passages, as spasmodic sneezing, cough and hay fever, all of these are possible features of the complex.

Referring to the intestinal tract, the condition invites anorexia, esophageal spasm, cardiospasm, gastric and intestinal atony, indicated by clapotage and distension, obstinate habitual constipation, intestinal colic, muco-membranous enteritis, appendicitis, nervous cyclic vomiting, with or

without acetonemia, occurring as car-sickness or as a consequence of psychic excitement or responsively to other marked irritation, and sometimes in the early morning when the stomach is empty.[10] The genito-urinary system, also, may be affected and present such symptoms as cloudiness of the freshly voided urine, with non-organized sediments, (uric acid, urates, phosphates and oxalates); intermittent albuminuria of the orthostatic type; polyuria, cystic spasms, dysuria, diurnal and nocturnal enuresis, cystitis and urethritis, balanitis, with erections and masturbation.

As nervous phenomena of the diathesis, may be numbered pavor nocturnus, choreic restlessness, tics, the facialis phenomenon, severe typical and atypical migraine, neuralgias, rheumatoid pains, arthralgias (from which the rather unfortunate term arthritism has been suggested), osteal-gia without objective cause, unusual flaccidity of the muscles and postural errors.

In the hyperplasia of the lymphatic system the most important involvement is that of the faucial and pharyngeal tonsils with its common sequelæ. Next in consequence comes the hyperplasia of the bronchial and mediastinal lymph nodes causing anomalies in the respiratory sounds, areas of pulmonary dulness confirmed by Roentgen shadows, and signs of varying compression especially upon bending back the head. The spleen may be palpable and occasionally the lymph nodes in the neck and in the joint flexures become markedly enlarged.

These and companion symptoms may appear singly or in variegated grouping. They may appear at more or less regular intervals in varying degrees of severity, becoming progressively more and more definite. Individual manifestations have often been looked upon as separate and distinct diseases and their appearance in orderly succession has been regarded as accidental. The more carefully, however, the history of the patient and that of his immediate family is studied, the longer the observation of the patient is continued and the better the observer becomes acquainted with his conditions, the more readily will the relation of the individual pathologic elements to the special predisposition or diathesis be recognized.

Even though rarely fatal, such manifestations of the disease as hyper-

[10] The acetonemic vomiting, a very characteristic symptom of a combined neuropathic and metabolic disturbance, occurs in the following manner. Children, especially boys, previously affected by atony of the stomach and intestines, with habitual obstipation, become ill every few weeks with frequent, violent and uncontrollable vomiting. This may follow some active voluntary exercise or may be entirely spontaneous or independent of any exciting cause. The attacks may last for several days, during which time all food is ejected. The vomitus may be bile-stained and bloody. The accompanying exhaustion may become alarming, but the patient usually recovers very quickly after the vomiting has ceased. During the attacks, there is not only acetonuria, but evident excretion of acetone from the lungs. The presence of acetoacetic acid, and of β-oxybutyric acid in the urine and expired air may further indicate the acidosis. Icterus and enlargement of the liver, with elevations of temperature may be noted. Apparently the condition is a cataclymic eruption ot an otherwise more or less latent disturbance of metabolism. According to Hecker, it is an interference with the catabolism of fat due to the hypoplasia of the lymphatic system. The eruption is seemingly brought on by nervous influences. Suggestive treatment is occasionally effectual. The writer prefers an active catharsis once a month, with the daily use of calcium chloride or of alkalies, with physiologically alkaline food, for a long period.

pyrexia without objective cause, succeeding an equally causeless collapse, acetonemia, paroxysmal tachycardia, pavor nocturnus, colic, enteritis, migraine, fainting, skin eruptions are, to say the least, very distressing and unpleasant complications.

Often, as a result, the child is unable to attend school and the diathesis is harmful from the social standpoint while such injuries as impairment of hearing, aprosexia in adenoid disease, enuresis, masturbation, etc.— have an ethical or moral bearing.

Treatment.—The treatment of the exudative diatheses cannot, perhaps, be directed against the fault in the germ or the resulting perversions of metabolism, but only against those certain external influences which excite its manifestations, such as the frequent intercurrent infections and those complicating structural abnormalities and functional disturbances which aggravate the initial error.

The secondary infections which, as Czerny believed, may convert the relatively innocuous primary manifestations of the diathesis into severe forms of disease, may be combated preventively by reducing both the predisposition to attack and the opportunity of infection. In the accomplishment of this first aim, dietetic management has proved useful in a purely empirical way. Procedures which serve to remove the excess of fluid from the tissues, if promptly applied, will have a favorable influence upon the hyperplasia of the tonsils, etc., and should take, therefore, a very important place in the treatment.

After the first two years, a largely vegetable dietary is to be recommended, supplemented with only a small quantity of meat and but one-fourth to one-half a litre of milk a day. Eggs, cream, butter and sugar are not well borne. During the first two years, the smallest possible quantity of milk compatible with gradual gains in weight and general development should be given. Human milk is to be preferred, but with any form of feeding the number of meals and the length of the individual feeding must be curtailed. If, in spite of these measures, a child is inclined to grow too fat, carbohydrates, broths and vegetables may be substituted for a part of the milk even during the latter half of the first year. At a year and a half the child may be put upon the diet of more advanced childhood.

A poor development of the breast-fed infant with exudative tendencies is often improved by a change to mixed feeding, in which such additions as buttermilk, gruels or malt soup are desirable.

An example of a menu for a child of two years, as advised by Czerny, follows:

Breakfast: Milk, diluted with hot water, and toast without butter; followed at 10 A. M. by raw fruit.

Luncheon: A thick soup, preferably of well-cooked legumes; finely divided meat; and such fresh vegetables as spinach, carrots, kohl-rabi, cauliflower, lettuce or string beans.

At 4 P. M. Diluted milk with toast or cake.

Supper: Finely divided meat, with bread, potato or rice, and a very little butter. Weak tea, or water with fruit juices, may be added.

The most efficient method of lessening the opportunities of infection to a minimum is to keep the child, whether in the country or in the city's suburbs, in a place where the air is free from dust and smoke. The summer vacation spent in climatically favorable places, avoiding hotels and sanatoria, should be extended as long as possible. The little patient should be kept away from disease carriers, such as adults with catarrhal infections or so-called colds. Chilling should be avoided by out-of-door exercise. Attempts at hardening or accustoming the child to infective agencies are useless.

Finally, great emphasis must be put upon the importance of psychic treatment in cases which show signs of present or impending nervous disturbances. This consists in seeking to withdraw the attention of the patient from his physical ills by systematized play or occupation, by the parental avoidance of anxiety and excessive care and by abstaining from medication and sanatorial treatment. The child should be protected from association with neurotic or mentally unbalanced persons. In certain cases it may be necessary to remove the patient from the home and to change his entire mode of living and training.

The main indications in the treatment of *arthritism* are similar, for this disposition frequently, although not always, arises from the inflammatory diathesis and, as a result of harmful psychic influences gradually tends to a more and more neuro-psychopathic type.

In these phases of the condition the largely vegetable dietary recommended may occasionally exert a favorable influence upon the viscero-somatic symptoms, but will have a less definite or indeterminable effect upon the stated neuro-functional disturbances. Tonic remedies, whether foods or medicines, iron, arsenic, quinine—in fact, practically all drugs that have been tried, are useless and even harmful, for their use tends to fasten the child's attention upon its ailments. For the same reason, all highly artificial measures as hydrotherapeutic and other physical treatments in sanatoria or elsewhere should be avoided. It is better to place the child in natural surroundings, adapted to its physical and mental development, which will approach as nearly as may be to "the simple life."

Certain symptoms, of course, require special methods of treatment. These should be brought into accord, however, as nearly as possible with the essential principles of management already laid down.

As the detail given may suggest, the entire treatment proposed approaches at many points, the methods applied in the so-called "nature cures." Nevertheless, the aim of this counsel has been to avoid the injuries so commonly incurred in these systems; as the sleeping in large wards, the entire deprivation of meat, the forced cold water treatment with resulting chills, etc.

In cases of the status lymphaticus, the danger of operative shock and of serious nervous excitement, as that attending the use of anesthetics, or the Roentgen treatment, or even the administration of hot and cold baths, should be emphasized; while the special risks of acute infectious diseases, as in the severe toxic form of scarlet fever, must always be borne in mind and the patient treated with more than ordinary precautions.

Formerly, great difficulty was encountered in the differentiation of the status lymphaticus from scrofula. The two were repeatedly but mistakenly associated and, as a result, progress of pathogenic and clinical research was materially impeded. It is true that careful observers, in their bedside investigations, had attempted, long ago, to make a definite distinction between these conditions; but it was Koch's anaphylactic methods of diagnosis and the safe and handy modifications proposed by v. Pirquet and Moro which first made their positive separation possible. Hence we have come to the acceptance of the view that scrofula is that form of tuberculosis in childhood which occurs in cases of lymphatism (Moro, Escherich).

APPENDIX

PATHOLOGY OF THE GLANDS OF INTERNAL SECRETION

A. PATHOLOGY OF THE THYROID

In the consideration of the pathology of the thyroid body the greatest clinical interest attaches to those disease-pictures which are proven to arise from functional disturbances of the organ. These disturbances may be due, on the one hand, to complete or partial absence of the gland or to qualitative changes in its activity, described as athyreosis, hypothyreosis and dysthyreosis, and, on the other hand, to a pathologic increase of its functional activity, known as hyperthyreosis. For the general pathology of these conditions we must refer the reader to text-books of internal medicine. However, the point of especial interest to the pediatrist is that during intra-uterine life, and probably during early infancy, the lack of the thyroid gland in the child may be met by the increased activity of the maternal gland. The active principles of the thyroid secretion undoubtedly pass through the placenta and, perhaps, through the mammary gland, and thus reach the infant organism. Therefore, fatal disease due to absence of the thyroid gland is never seen. This is significant since the young and growing organism is especially susceptible to injury from disturbances of the functional activity of the thyroid. The group injuries due to hypothyreosis are seen only in childhood. In these conditions perfect therapeutic results can be obtained only when the status is recognized and treated in early life.

Hypothyreosis; Athyreosis

These conditions may be conveniently classified in accordance with their anatomic and etiologic causes.

1. Hypothyroidism may be caused by anomalies of structure, resulting in complete absence of true thyroid tissue. The anatomic conception of this complete thyroid aplasia, the congenital athyreosis, coincides completely with the clinical picture of congenital myxedema.

Cystic formations are found occupying the place of the lateral thyroid germ centres and developing epithelioid tumors at the base of the tongue. The epithelial cells persist in this type. The condition is one of actual primary aplasia of the gland with an absence of its arteries.

Short of total aplasia of the gland we may have hypoplasia and congenital insufficiency, which lead to partial degrees of congenital hypothyroidea.

2. Hypothyreosis may also result from a more or less extensive degeneration of the gland in extra-uterine life. This may take the form of either a primary atrophy or a goitre-like dystrophy; in either of which forms the active glandular tissue may entirely disappear. According to Wieland,

this is a condition affecting the germinally weak gland in what may be called a "hypothyroidic diathesis."

In this event, the parathyroid bodies may be included in the degenerative process. For the sequelæ of this involvement the reader is referred to the Chapter on Spasmophilia (Tetany).

Such thyroid degenerations are observed, (*a*) in certain localities where they appear as an endemic cretinism, arising from injuries common to the region, of the nature of which but little is known; and (*b*) as entirely sporadic cases, presenting varying conditions of the thyroid, but resulting in an acquired form of infantile myxedema. Either of these types is frequently seen in abortive forms.

3. Finally, hypothyreosis may be caused by the partial or complete surgical removal of the gland which results in an operative myxedema, (*cachexia thyreopriva*). This condition is of great interest from an experimental standpoint, but may no longer be considered clinically interesting.

All of these forms of a- or hypothyreosis share the group of symptoms which constitue the phenomena of absence, relative or complete, of the function of the thyroid gland. Their clinical relationship is therefore a very broad one and their differentiation depends primarily upon the demonstration of the etiologic factor in each case. Certain signs, such as the disturbance of dentition, are, of course, clearly manifest when the functional activity of the gland has been wanting from the earliest postnatal period; while others appear only after the condition has existed for years. It is quite probable that in cretinism there are injuries to other and possibly intracranial organs besides those which have led to the disturbance of thyroid function.

General Symptoms and Their Causes

1. Affections of the Skeleton.—These are among the earliest and most constant of symptoms. They arise from a disturbance of both the endochondral and periosteal growth, with atrophy of the bone and the blood-forming marrow.

The cartilage formation at the epiphyses, the extension of the marrow capillaries into the cartilage cells, the resorption and to a degree the apposition of the bony tissue are all retarded. The process of calcification is entirely undisturbed. The result is the development of sclerotic bones of normal shape, but of reduced size. It is apparent that the hypothyroideal affections of bone are, in some respects, the precise opposite of the rickitic forms; so that children with myxedema never give definite signs of rickets. The epiphysial centres develop late or are entirely absent, while their cartilaginous structure persists. Longitudinal growth is delayed and often continues until after puberty. The Roentgen picture usually shows a characteristic dark shadow at the diaphyses representing a transverse lamella of bone extending toward the epiphysis. (Figures 55 and 56.)

Clinically, the disturbance is recognized as a fairly proportionate dwarfism, which becomes more and more pronounced; by changes of form due to anomalies of the soft tissue, to be described later; by a depression of

the bridge of the nose not appearing as an initial dystrophy, but as an inhibition of growth at the tribasilar suture; by the permanently open fontanelle and cranial sutures, and by anomalies of dentition and of teeth formation. These dental anomalies resemble those of rickets. The closure of the flat, low, wide cranium may be delayed even longer than it is in rickets, but the edges of the bones are hard. Fractures heal very slowly in the hypothyroidic.

The several characteristics of the cretinoid skull (after Scholz), are: orthocephalia, platycephalia, plagiocephalia, platyrrhinia, hypsicephalia, and prognathus.

Recently this disturbance of the bony growth has been repeatedly laid to the hypothyroideal injury of the blood-forming marrow (compare rickets, scurvy, etc.). The involvement of the blood-forming marrow may be regarded as the cause of such marked evidences of anemia as the pallid or dull yellow skin, the oligocythemia, oligochromemia and polynucleosis. It may be held responsible, also, for the hemorrhagic diathesis and for the enlargement of the tonsils and of the lymph nodes which such cases exhibit.

2. Changes in the Skin and Mucous Membranes.—As a result of a peculiar redundancy of the elements of the subcutaneous connective tissue, the nature of which has not yet been determined, the skin, at certain points, becomes loosened from the underlying tissues (*cutis laxa*), and has a gelatinous, doughy or slippery feeling—the myxedema of Ord. True edema, however, with complete loss of elasticity does not occur. When the myxedema disappears the skin is left slack and wrinkled. The interference with the nutrition of the epidermal layers causes desquamation, cracking of the finger-nails, dryness and falling of the hair; the lanugo, however, persisting. A suppression of the perspiration also results, in all probability, from the dryness and the reduced electro-conductivity of the skin.

Similar tumefaction and loosening of the mucous membranes of the nose, pharynx, middle-ear, Eustachian tubes, and especially of the tongue (macroglossia), of the larynx, the eyelids and the digestive tract, are held responsible—although not always convincingly—for such functional disturbances as mouth-breathing, snoring or groaning respiration, hoarseness, impairment of hearing, refusal of food and obstipation. The symptoms of these three groupings produce very characteristic anomalies in the external habitus of the patient. The low, flat brow, covered by wrinkled, hairy skin; the flat saddle-shaped nose, the narrowed aperture of the swollen eyelids, and the widely gaping mouth, allowing the enlarged tongue to protrude between the thick and almost trunk-like lips, are its principal features. There is often, also, a double chin; the ears are large and malformed; the normal contour of the body, moulded upon the skeletal lines, is obscured by the irregularly thickened soft tissues. This certain grotesqueness is increased by spongy skin-pads formed over the clavicles, the scapula and the hips and around the nipples. The limbs are cylindrical and pillar-like and the fingers and toes seem shortened and stubby. The hand resembles the paw of a mole. The abdomen is greatly distended and, in younger children, is frequently and further deformed by an umbilical hernia.

3. Psychic Anomalies.—The mental development is arrested at a very low stage or rather is retrogressive to such a stage. In the more severe cases, the intellectual capacity of the patient is less than that of intelligent animals (the vegetative man of Kocher). The mental aberration is always of the characteristically anergic form. The child is uncleanly and drowsy; it lies for days without reacting to sensory impressions, its gaze apathetically fixed, its movements automatically responsive when forced. It roars when hungry or in pain. Cases of lesser severity are unable to fix their attention, to recognize objects or to speak. In mild forms there is observed

FIG. 55.—Hand of a six-year-old girl with congenital athyreosis. Height 72 cm. (28.8 inches.) Thick sclerotic bones. All the epiphysial centres of ossification but one small one in the wrist which appeared after treatment with thyroid extract, are lacking.

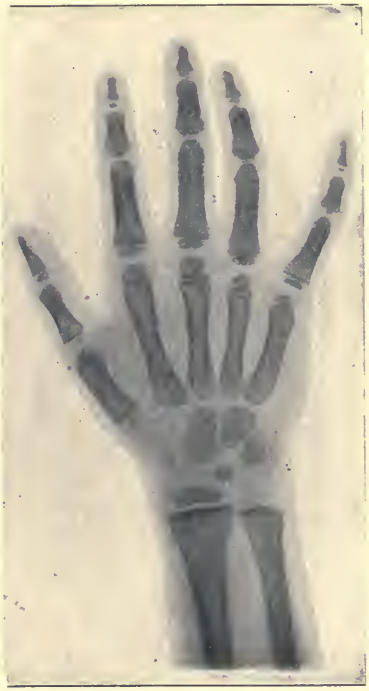


FIG. 56.—Hand of normal six-year-old child for comparison. (Children's Hospital, Zurich, Prof. E. Feer.)

only a certain weakness of the intellect and memory, an inability to concentrate attention and a mental dulness. In such a case the timidity, the cretinoid demeanor, the gait, the grimaces, the inarticulate sounds, and the dull, morose, animal-like physiognomy of the child may lead one upon superficial acquaintance to under-estimate its actual mental ability. The delayed development of child's static functions, as in the matter of sitting, standing or walking, is, in part, due to these psychic disturbances.

There is still much diversity of opinion as to the organic cause of these mental perversions. Severe affections of the auditory apparatus, both of peripheral and labyrinthine origin, and to the extent of deaf-mutism, and of

the tubal and middle ear tissues incident to adenoids are often concerned in its etiology.

4. Changes in Metabolism.—These changes consist in a slowing of the metabolism, particularly in the field of protein, water and salt interchanges. The consumption of oxygen may be reduced to one-half normal. It is supposed that the low average body temperature, ranging 1°-2° C. below normal, and the tendency to obesity are connected with this slow metabolism. As a result of the reduction of the katabolic processes there is a tendency to nitrogen and phosphorus retention. As a further result, the patient

FIG. 57.—Three-year-old child with aplasia of the thyroid and congenital myxedema. (University Children's Hospital, Munich, Prof. von Pfaundler.)

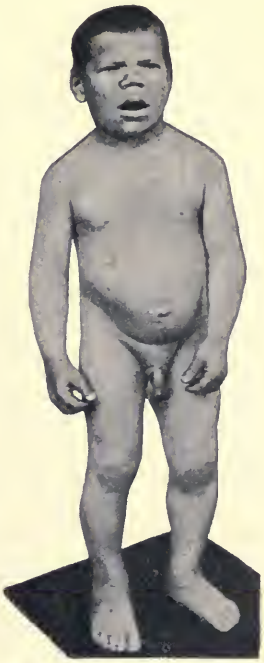


FIG. 58.—Endemic cretin from the Odenwald.

can maintain weight upon quantities of food which would be altogether insufficient for the normal child of the same age. The limit of assimilation for dextrose is raised.

5. Affections of the Muscles.—The musculature is usually flaccid, pale and wanting in power. It is said also, to be microscopically changed. At the same time it may be of large bulk and even pseudohypertrophic. Slow movements, delayed static functions, a dragging gait with equinus and bended knees, a flattening of the head of the femur, double-jointedness, lordosis, abdominal distension, severe obstipation, diastasis of the recti, umbilical or inguinal hernia and, probably, cardiac weakness with small labile pulse, cold mottled skin and a tendency to cardiac asthma, may all be

related to this muscular impairment. In some cases one finds on the other hand, a large and firm body musculature.

6. Disturbances of Sexual Development.—In the hypothyreoses, hypoplasias of the germinal organs and of the external genitalia are found. Partial development of sexual functions, a lack of the secondary sexual characteristics, postponed puberty, genital infantilism and menorrhagia follow.

Special Clinical Considerations of Hypothyreoses

1. Congenital myxedema is more common among females. Neither hereditary factors nor any other recognizable causes enter into consideration in the hypothyreoses. The first indications of the deficiency, apathy, delayed growth, and the changes in the skin and the mucous membranes, are usually noticed during the first week in artificially-fed infants and after the first month in breast-fed babies. The structural changes in the skeletal framework originate at birth. The entire symptom-complex develops rapidly, as a rule, to the point of severity. Most of the cases die during their first years and only rarely do they pass into the second decade. The entire absence of the thyroid gland may hardly ever be demonstrated clinically on account of the changes in the skin and in the muscle topography.

Fig. 59.—Six-year-old boy, sporadic cretinism.

2. (*A*) Endemic cretinism affects man and animals alike in the so-called goitre districts of certain mountainous countries of Europe. These districts lie particularly in the Swiss Alps and the Black Forest but are found in other parts of the world. The disease is somewhat more common among males. Hereditary and familial influences are in part responsible. Presumably they consist merely in a peculiar congenital lack of resistance of the gland to injurious influences. A degree of significance appears to attach to the family residence in a dangerous locality. Children brought from other and healthy districts into such localities are frequently affected at an early age; while children of families affected with cretinism who leave the infected district in early childhood or are born in other parts, often recover or remain healthy. The cretin districts are not confined to mountainous regions but also exist in localities marked by the emergence of certain geological strata of the earth's surface, or lie along the lower reaches of rivers which flow through such formations. There are many indications that the noxious agent is carried in unboiled drinking water. The hypothesis that the mineral substances, taken up by the water from these rocky foundations, may exert in young persons a

damaging or degenerative influence upon the thyroid gland (Author, 1907), is quite compatible with the known facts. The writer considers the attempts that have been made to demonstrate infectious or contagious influences in the causation of the disease as complete failures or very unsatisfactory in results.

FIG. 60.—Twelve-month-old girl with myxedema.

Clinically the thyroid is found to be enlarged in about sixty per cent. of cretins. Structurally, these cases show a variety of goitrous degenerations, cystic, parenchymatous, hemorrhagic and, more rarely, atrophic in type. Portions of the glandular tissue may remain anatomically intact and are, perhaps, functionally active.

The symptoms of endemic cretinism appear comparatively late. Usually, the disease may be recognized definitely only by the fifth or sixth year, or even later: The disease-picture becomes very gradually definite and hardly ever reaches its extreme development during childhood. A constant qualitative difference of the entire symptom-complex from that of other hypothyreoses can hardly be said, however, to exist. Besides the signs of hypothyroidism one finds incidentally those of hypogenitalism and hypopituitarism.

2. (*B*) Acquired infantile myxedema is charged to various acute diseases of the gland and differs from cretinism chiefly in the fact of its sporadic occurrence.

The abortive forms of hypothyreosis are of special interest to the physician. They may depend initially upon congenital causes, such as an actual hypoplasia, an arrested development or a functional inadequacy of the gland. They may be a manifestation of hereditary degeneracy, due to alcoholism, lues, or some cachexia in the parent. They may depend upon injury acquired in some cretinic

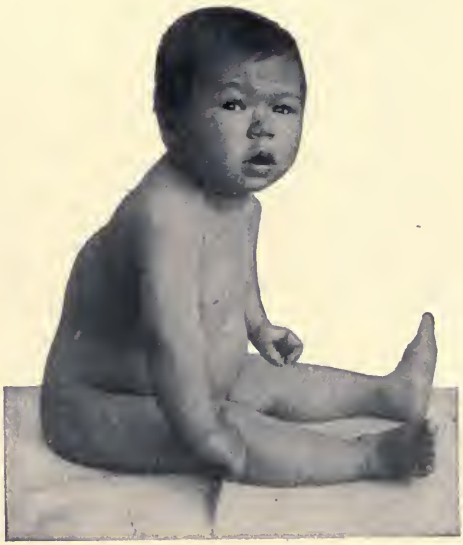


FIG. 61.—Three and one-quarter-year-old girl with slight manifestations of myxedema, height 80.5 cm. (32.2 inches), anterior fontanelle patent, 8 incisors, cannot stand and kyphosis on sitting. (Children's Hospital, Heidelberg, Prof. E. Feer.)

district or at large. Etiologically, in these abortive types, the clinical examination of the thyroid is of little value and the findings are variable.

Usually the psychic symptoms, as, for instance, voice changes, depreciation of mental ability, etc., are not definite. The somatic picture is confined essentially to dwarfism, to disturbances of dentition, to a slightly myxedematous, dry and irritable skin, to the cretinoid, care-worn physiognomy and to the general demeanor. The disease may be characterized, also, by such functional disturbances as chills, anhydrosis, awkwardness of gait, enuresis, obstipation, monotony of speech, and mental inefficiency. The Roentgen picture, showing the delayed ossification and the transverse diaphyseal plates, may support a diagnosis which will receive even greater encouragement from the therapeutic results to be reviewed.

Fig. 62.—Two-year-old boy with mongoloid idiocy. Characteristic laxity of the joints.

Infantilism

Clinically, these cases, may be correctly included in the symptomatic group belonging to the condition of so-called infantilism.[1]

Under the term infantilism are included conditions of widely varying etiology, in which the physical and mental development, relative to the actual age, present an abnormally youthful habitus. Some authors distinguish two varieties of infantilism arising from insufficiency of the thyroid function. Of these, the Brissand-Hertogh type has true hypothyroideal manifestations. The type of Lorrain, characterized by delayed growth in height, with a symmetry of proportions, slender bones, graceful extremities, small narrow head and girlish appearance, deviates markedly from the former and its fit placing in this class is very questionable. In this type, it would seem, rather, that a functional lack in other glands, as the ovaries, the suprarenals, the thymus or the pancreas, or that injuries due to improper feeding, early infections, intoxications or organic diseases, may enter into the causation (Anton).

The abortive infantile hypothyreoses are characteristically benign and tend to spontaneous recovery.

Treatment

The treatment of the hypothyreoses is based upon physiologic substitution. The reliability and applicability of this method, in all forms of the

[1] The histologic findings in the osseous system resemble, of course, a senile marasmus rather than an infantile dyscrasia (Dieterle).

disease in early childhood, is now generally accepted. Doubtless, the simplest and most rational procedure would be the implantation of a thyroid gland of the same species at any suitable place in the body of the patient, provided that such a transplanted organ could maintain its functional activity. This has been attempted in a number of instances but, according to report, it has seemed impossible to keep the transplanted organ duly active. Under the law of biologic specificity, it would not appear possible. Fortunately the fairly thermostable active principles of the animal thyroid are transferable and may even be absorbed through the digestive tract. It is deemed best to feed the thyroid gland of food animals. This is given in fresh raw form, finely divided, and prepared with butter, eggs, and spices. The glands of sheep are most commonly used, but those of cattle or hogs may be employed. Care should be taken to see that the animal from which the gland is taken is healthy and that such other organs as the thymus, salivary glands, lymph nodes, or pancreas are not substituted. One or two pairs of sheep glands are given two or three times a week.

As a similar substitute, the more easily obtained pharmaceutical preparations of the dried, or desiccated or extracted thyroid gland are recommended. Thyroxin, the active principal of the gland may be used. Other methods of administration, as by subcutaneous injection or enemata, have been discarded.

The results of this treatment are usually very marked. It affects all the somatic and psychic manifestations of hypothyreosis at once. Improvement may be noted within a few days and may lead to the complete disappearance of the symptoms. Discontinuance of the remedy quite naturally causes a remission. Especially favorable results are seen in the treatment of congenital and acquired myxedema, but benefit is had even in endemic cretinism among young patients. The average of results of the treatment gathered from large numbers of cases would probably be even better than it is, if occasional complicating diseases, unrelated to the hypothyreosis, could be excluded. In the cretin, the treatment does not affect the disturbances of hearing as markedly as it does other symptoms and the unrelieved deafness may hinder mental development.

The treatment with the thyroid gland must be begun very carefully and, at first, the patient must be scrupulously watched. Injuries resulting from excessive doses may be of hyperthyroideal nature, such as restlessness, palpitation of the heart, cardiac weakness, flashes of heat, perspiration and vomiting, or they maybe of a toxic character, resembling those of *botulism* or meat poisoning. The latter accident occurs only when suspicious pharmaceutical preparations are used.

GOITRE

Congenital and acquired hyperplasias, or rather hypertrophies, and strumous degenerations of the thyroid gland are seen quite frequently in certain families in which the disease prevails; but are otherwise quite rare. Congenital goitre frequently appears to be caused, in large part, by a vascular congestion of the organ; and with suitable treatment, or even

without treatment, often disappears rapidly. In other cases, parenchymatous overgrowth and the formation of nodes and cysts are observed. In the former event, a scleral degeneration with hypothyreosis, and in the latter the very opposite condition may constitute the permanent injury. A congenital struma may spread very gradually until it extends to the lower jaw and may occupy the entire neck. This may result in symptoms of compression of the esophagus, with reflex vomiting; of the cervical vessels, leading to cyanosis and edema; and particularly of the soft trachea, causing dyspnœa, stridor, pulmonary atelectases and broncho-pneumonia. Fibrous goitres surrounding the trachea and extending downward beneath the sternum are especially dangerous, but are fortunately quite uncommon.

At puberty the frequency of goitre is again marked and especially in girls. In these cases, we may also find general disturbances, due to an excessive secretion of the gland. They may go on to the development of Basedow's disease. Glands enlarged by degenerative changes may fail to give symptoms of hyperthyreosis (*q. v.*), even though there is a huge goitre present.

Fig. 63.—Facial expression of a ten-year-old boy with cretinoid degeneration.

Treatment.—The greater number of strumata in children react favorably to iodine treatment in the form of potassium iodide given internally, 0.1-0.5 gms. (2-8 grs.), a day; or to the external application of potassium iodide ointment with the addition of a small amount of iodine. Preparations of the thyroid gland cannot be recommended and are, in fact, contraindicated. The vascular strumata of the new-born should be treated with applications of ice alone.

BASEDOW'S DISEASE

This disease is rare in childhood and is met with, but little more frequently toward puberty. It is a hyperthyreosis of unknown etiology, characterized by a rapid and demonstrable increase in the size of the gland. Its manifestations and treatment in the child are the same as in the adult.

INFANTILE BASEDOWOID DISEASE

Recently this term has been applied to a condition found by Hochsinger in one case. It is said to be characterized by mild ocular symptoms, resembling those of Basedow's disease by tremor, palpitation, and hyperhydrosis. This condition coexisted with true epilepsy and yielded to treatment with the thyroid gland.

B. PATHOLOGY OF THE GERMINAL ORGANS

Hypogenitalism, eunuchoidism, dystrophia, adiposo-genitalis, are terms applied to a condition resulting from perverted interstitial development of

the reproductive glands. It resembles the status which follows castration. Eunuchoid individuals are tall and long-limbed and inclined to adipose deposits over the lower abdomen, thighs and eyelids, or sometimes to general obesity. The development of the secondary sexual characteristics is retarded and poorly achieved. The epiphysial grooves remain open for a long time and the genitals are small. Abdominal or inguinal cryptorchidism is common. Eunuchoid boys (for in females the condition is, to say the least rare), are remarkably quiet, unassuming and dependent. The condition is most frequently recognized at puberty. In the course of years spontaneous recovery may occur. Thyroid treatment is useless.

C. PATHOLOGY OF THE HYPOPHYSIS

Hypopituitarism, or the reduction of the function of the glandular portion of the hypophysis, may obtain in later childhood as a result of pathologic changes in the organ itself, or in the neighboring structures. Such a disturbance is associated with a dystrophy of the genitals (hypophysial form) since it causes a retardation of the development of the interstitial and generative portions of these organs. Aside from the obesity or abnormal distribution of the fat which becomes especially marked about the thighs, breast and hips, the small genitals and the failure of the secondary sexual characteristics with reduced metabolism and increased carbohydrate tolerance; we have here also the growth anomalies of the eunuchoid type. This is characterized by the great height, due to the continued growth of the long bones after the normal period for the attachment of the epiphyses which, in turn, is delayed because the generative organs do not stimulate the growth. In many cases, however, a disturbance of ossification of true pituitary origin counteracts this continued growth. The pituitary action here is to delay the growth of the centres of ossification resulting in a small child-like body. The two forms of genital dystrophy with obesity can further be distinguished by the appearance of focal brain symptoms or by the appearance of the cella turcica in the radiogram.

The treatment with pituitary extract by mouth is apparently of some benefit, surgical interference and Roentgen treatment of doubtful value.

D. DISTURBANCES OF GROWTH

General Physical Anomalies Occurring without Recognized Relation to the Hemic Glands

Chrondrodystrophia (Kaufmann, 1892), achondroplasia (Parrot 1878), or micromelia chrondromalacia, is a disease of the fetal cartilaginous skeleton. It occurs chiefly in females. The proliferation of the cartilage at the junction of the epiphysis and the diaphyses, which represents the method of longitudinal growth of the bones performed in cartilage, is inadequate. On the contrary the calcification and ossification of the shorter cartilaginous shafts, as well as the periosteal ossification, are not affected or proceed even more rapidly than usual. The end result is seen in abnormally short and thick bones and in premature synosteoses. An abnormal strand of connective tissue passing from the periosteum into the epiphysial-diaphyseal

boundary—the characteristic periosteal lamella—causes an irregular one-sided growth and results in the decurvation of the bone. The width of the cartilaginous epiphyses varies. Occasionally it is diminished; again it may be enlarged to a mushroom-like thickening, representing both the hypoplastic and the hyperplastic forms.

The clinical picture presents a micromelia, determined by the shortness of the limbs and accompanied by the true saddle-form nose caused by synosteosis of the tribasilaris. The excess of fatty, but not gelatinous skin hangs upon the short limbs in folds and wrinkles like trousers or sleeves that are too large. These appearances are often mistaken for the myxedematous cutis laxa and for the true cretinoid physiognomy. For the cranium is large, the neck heavy, and the three stub-pointed middle fingers, on account of the union of their basal phalanges and the spreading of their distal phalanges, form the characteristic trident hand. Other malformations and strumata are common in these cases. The children who survive attract attention because of their muscular weakness, double-jointedness, the delay in the acquirement of their static functions and in the closure of the fontanelles, the rotatory spasm and the profuse sweating. The condition is sometimes mistaken for rickets. When the patient learns to walk with a characteristic waddling gate, the preëxisting lumbar kyphosis becomes a lordosis with a markedly protruding abdomen.

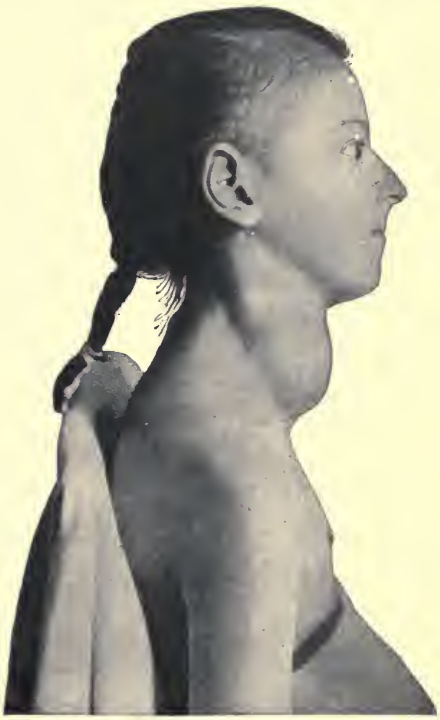


Fig. 64.—Eleven-year-old girl with Basedow's disease. Improvement after ligature of the arteries.

His intellectual development is not retarded and is often surprisingly good. It is said that court jesters and clowns have been recruited from this class.

Treatment with thyroid preparations is generally useless.

OSTEOGENESIS IMPERFECTA (VROLIK)

Osteopsathyrosis

This is a condition of deficient endosteal and periosteal ossification with normal formation of cartilage. It is a disturbance of osteoblastic function. The bones grow long, but are thin and very porotic. On this account, numerous so-called spontaneous fractures occur in all parts of the skeleton.

both in fetal and postnatal life. These fractures, and there may be many, lead to malformations and to a shortening of the extremities (micromelia). The development of numerous calluses may produce a conditon which resembles an abnormal thickening of the bones. In the cranial bones, preformed in membrane, large membranous openings may persist. (See Rickets.) During life, the Roentgen picture shows this condition very plainly (Fig. 66). Therapeutically, phosphorus and cod-liver oil to prevent rickets has been found valuable. Adrenalin has been suggested. No doubt, fresh air, sun-baths and diet must all be looked after. All cases in which the diagnosis was definitely established died during the first year. The so-called late form appearing in older children appears to be a different disease.

Fig. 65.—Month-old child with chondrodystrophy, habitus and form of face, unusual laxity of the skin.

Mongolism-mongoloid Idiocy.—(Langdon Downs 1866.) This is a complex congenital abnormality which suggests a "reversion to the ape-man type." Its structural peculiarities are associated with serious impairment of mental functions. It produces characteristic changes of habitus and frequently implicates to a great extent the functional systems of the affected individual. According to Jodiche, the serum of a mongoloid patient, studied by Abderhalden's serodiagnostic method, showed the degeneration of a number of organs and especially, of the reproductive glands. Little positive knowledge of its etiology has been acquired. Varying indications of senility in the mother during pregnancy seem to play their part. The disease only occurs sporadically, but appears in all strata of society and in all countries. It has seemingly increased in frequency of late years. The sexes are affected equally.

Fig. 66,—Upper extremity of a newborn infant with osteopsathyrosis, multiple fractures, and callous formation.

The habitus, faintly resembling that of the Mongolian, is the most reliable criterion of recognition. It is marked by brachycephaly, a small saddle-shaped nose, slanting eyes, the inner canthus being lower than the outer, with narrow lid apertures, epicanthus (the sickle-shaped, vertical fold of skin over the inner canthus), habitual conjunctivitis and blepharitis, and frequently divergence of the rather prominent eyeballs. The mouth gapes and is constantly salivated. The cheeks and chin show a distinctly circumscribed red color, suggestive of a clown's mask. The external ear is atavistically malformed. The abdomen is distended and a diastasis of the recti muscles is observed. A double-jointedness, resulting from a muscular

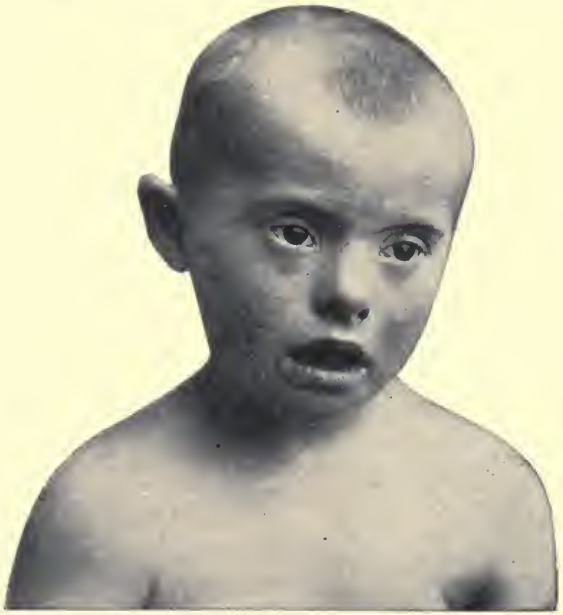


Fig. 67.—Mongoloid. (Children's Hospital, Breslau, Prof. Tobler.)

flaccidity or aplasia is common. Finally the shortened and incurvated small finger, incident to hypoplasia of the distal phalange, is in evidence.

The demeanor of the imbecile or idiotic child is very characteristic. While it is, at first, apathetic, by the end of the second year, or even later, it becomes restless, active and aggressive, with a tendency to imitation, to make grimaces, and to gesticulate in an ape-like fashion. It employs itself energetically with all sorts of mischief, in the way of tipping over objects, pulling things apart, and especially in climbing. It likes to talk, but foolishly; it gives the more or less definite impression of a happy and lively disposition, adaptive to its surroundings.

This mental change, which is hailed with joy by the layman, as evidence of decided progress in the psychic development of the child hardly ever leads to the attainment of useful coördinated acts and purposeful mental capacity. The Mongolian child hardly ever learns to speak correctly, to com-

prehend any great number of words, to recognize its surroundings, to keep itself clean and the like. It is scarcely ever capable of school training but remains upon its mentally low plane or falls back into a mental torpor. Its

FIG. 68.—Twenty-one-month-old child with mongoloid idiocy. Brachycephaly, typical form of face and attitude, strabismus.

special love of music is often noticeable. Milder cases generally acquire abnormal habits.

The skeleton is affected by a group of facultative qualities, such as retarded growth, delayed development of the epiphysial centres of ossification, delayed dentition, tardy closure of the fontanelles. Chicken-breast is of common occurrence. In the reproductive system there is a lack of development of the sexual characteristics and functions. Mongolism in its later course is not infrequently found in association with true hypothyreosis, with its characteristic physiognomy, myxedema, microglossia, faulty breathing and subnormal temperatures. It is also complicated, sometimes, with rickets or with adenoid vegetations and their sequela. The general defect may be coincident, too, with a number of congenital malformities, *e. g.*, heart lesions, cleft palate, polydactylia, etc.

FIG. 69.—Eighteen-month-old girl with indistinct but recognizably mongoloid features. Later developed into a typical mongoloid idiot.

Atypical cerebral convolutions suggest a more or less constant structural substratum of the psychic syndrome of mongolism.

The skeletal symptoms are based upon fatty and interstitial changes in the bone-marrow, with the formation of transverse bands of bone at the epiphyseo-diaphyseal junction. This anatomic basis of the hypothyreosis and of such sexual symptoms as the delay in the development of the reproductive glands is to be found in a hypoplasia of the thyroid gland with proliferation of its interstitial tissue.

The semigenesis of the mongoloid habitus itself is still rather obscure. It may be readily imagined that an unknown constitutional injury affecting

the cerebral cortex may also affect the bone-marrow and the several glands of internal secretion, either directly or indirectly, without producing any characteristic or constant structural change.

The first somatic indication of mongoloid degeneracy, in the physiognomonic appearance, may probably be noticeable at, or soon after birth. The psychic manifestations attract attention during the first year. The laity and physicians who have not learned to recognize Mongolism often miss any clear picture of the disease. This is surprising enough to the man of experience. But very rarely does a general disappearance of all symptoms and a more or less complete recovery from the disease occur in the course of the child's development. The morbidity and the mortality of the Mongoloid type is surely very great.

Therapy.—Hypothyreosis, coincident with Mongolism, can be overcome with thyroid treatment. But, in spite of the fact of its popularity among mothers, this treatment has no distinct affect upon the Mongolism itself. It may be readily understood that any stimulation of the metabolic functions by the active principles of the thyroid gland may have a favorable, but non-specific influence upon the somatic and psychic processes, particularly in the apathetic stage. This effect, however, never suffices to make the patient anything but refractory to pedagogic training. He remains therefore, socially impossible. Furthermore, the thyroid treatment has its elements of danger (heart failure, nephritis, stomatitis, etc).

III.

DISEASES OF THE DIGESTIVE SYSTEM

BY

H. FINKELSTEIN and L. F. MEYER,
Berlin.

REVISED AND EDITED BY

Dr. JOSEPH BRENNEMANN,
Attending Pediatrician, Children's Memorial Hospital, Chicago, Ill.

DISEASES OF THE MOUTH

Stomatitis

Injuries of many kinds, thermal, chemical and bacterial, may cause a primary inflammation of the mucous membrane of the mouth. They may also become secondarily involved when, in the course of general diseases, a weakening of the local and general resistance causes a predisposition to the growth of pathogenic organisms, as, for instance, in all kinds of fever, gastro-intestinal disturbances, etc. Finally, changes of the mucous membrane may be symptoms of many general infections (measles, scarlet fever, etc.).

Catarrhal stomatitis is the most common and important of the primary forms. A diffuse, dark, reddening appears upon the swollen, readily bleeding, mucous membrane of the gums, cheeks and fissured lips, while the tongue, usually covered with a heavy white coat, appears roughened because of the swelling of its papillæ. In children with teeth, we may have salivation which irritates the skin surrounding the mouth; fetor exists, and, in severe grades, restlessness. The feeding is interfered with because of pain. Slight rises of temperature and, occasionally, slight regional adenitis occur. In new-born and very young children catarrhal stomatitis presents several peculiarities. It is usually accompanied by thrush and Bednar's aphthæ, traumatic palatal ulcers, pterygoid ulcers, (Fig. 70), *i. e.*, gray exudations on the mucous membrane of the palate which may change into superficial eroded sores which are typical in their location upon both sides on the pterygoid process and on the median raphe. On the process, they are usually round and on the raphe elongated. Of the various attempts at explanation of these ulcerations, but one is correct; they are nothing more than mechanical erosions caused by attempts at cleansing the mouth. Bednar's aphthæ always point to ignorance in the treatment of mouth conditions. At the time of the first dentition a special predisposition to catarrhal conditions of the mouth exists. The so-called "epithelial pearls," yellowish white masses, the size of a pinhead, are frequently seen on the median line of the hard palate. They are supposed to be inclusions of nests of epithelium formed by the closure of the structure.

The prognosis of the disease is good. With proper treatment and care of the all-important general disease, healing may be expected in a few days. On the other hand, there is the possibility of complications, especially of a septic nature.

The most important and successful prophylactic measure in infants is the omission of the customary mouth cleansing. Epstein has shown that washing the mouth causes traumatic aphthæ and that the omission of this routine scrubbing avoids catarrhal conditions. So long as a child is not given anything to chew and nothing becomes lodged between the teeth to decompose, the mouth should be left severely alone. This is also the best therapeutic measure in successful treatment. With older children, the condition of the teeth must be looked after. Under certain circumstances, measures used for the aphthous forms of stomatitis apply to this type.

Septic Stomatitis.—Very frequently in infants, less frequently in older children, we find septic catarrhal conditions of the mouth. These are characterized by severe purulent inflammation and often by circumscribed or flattened fibrinous plaques with a tendency to ulcer formation. Accompanying these severe general symptoms, high fever and, not uncommonly, the picture of general septic poisoning appears. Locally, spreading necroses and gangrenous disintegration are occasionally seen. In other cases, the dental germs may become inflamed and form abscesses. Complications, in the way of lymphadenitis, inflammation of the salivary glands, phlegmon, erysipelas and septic metastases, are not uncommon.

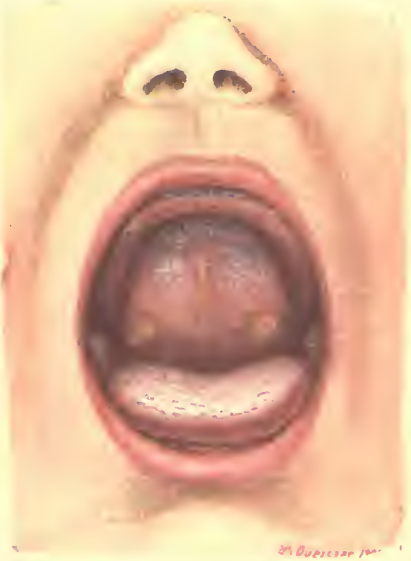


Fig. 70.—Catarrhal stomatitis, thrush and Bednar's aphthæ in an infant. (Berlin Children's Asylum.)

An especially appalling condition is seen in the pseudodiphtheria of the new-born (Epstein). In feeble children during the first few weeks of life, and usually arising from aphthæ, tough, flat, necrosing deposits, following confluent fibrinous exudates, appear which frequently attack the underlying soft parts and bones. They spread rapidly to the pharynx, nose, larynx and esophagus. At autopsy, this disease may be mistaken for lesions caused by the ingestion of caustics.

The cause of septic stomatitis is to be found in virulent pyogenic organisms, most commonly streptococci. Diphtheria may, at times, be the cause. The lowering of resistance due to an existing or intercurrent disturbance of nutrition may be the fundamental cause and gives an especially bad prognosis. The necrosed and gangrenous forms are doubtless found only in

generally run down children. A suitable feeding therapy often gives the first and most surprising results, when all other measures have had only a palliative effect.

Aphthous stomatitis (canker sore mouth), is characterized by the appearance of white or yellowish, round, lenticular plaques slightly raised upon an hyperemic base, found especially on the anterior portion of the mouth and tongue, while the posterior portions, the soft palate and the tonsils are much less frequently attacked. These "aphthæ" may become confluent here and there and form large patches or areas. They are the result of the effusion of a fibrinous exudate into the upper layers of the epithelium.

The eruption occurs with local symptoms, pain, consequent difficulty of feeding, salivation, fetor, and fever. The duration of the disease is usually from one to two weeks. In most cases recovery then takes place. With feeble children, however, a disturbance of nutrition or a pneumonia may be added to this, or a severe local disease of long duration may be ushered in by the aphthæ. With high and continuous fever, disseminated membranous exudations, ulcerations, extreme swelling and fissuring of the lips and irritation of the surrounding skin may occur. Septic complications are not uncommon. These are probably the result of a septic stomatitis implanted upon the soil of the aphthæ. In the diagnosis, one must consider true diphtheria; and, aside from the bacterial examination, the appearance of typical lentil shaped plaques is diagnostic.

Herpetic stomatitis, which is not uncommon in children, gives a similar exudation when the small vesicles have broken. The mode of onset, the group formation of the efflorescence and the absence of infective organisms make the differentiation possible. The cause is unknown. Some observers connect the disorder with the foot and mouth disease of cattle. It is undoubtedly transmissible and the companions of the child should be protected against infection by special measures (isolation, separate utensils, etc.). In the treatment, energetic local measures should be avoided. Rinsing or irrigating with warm infusions or with antiseptic solutions, as potassium permanganate, hydrogen peroxide, or boric acid solution may be employed. With tractable children, the disease spots may be touched with a 2 per cent. solution of silver nitrate or phenol. For the pain, penciling before eating with anesthetizing solutions (novocaine, 1 per cent. or 3 per cent. eucain lactate) or dusting with sterilized bolus alba, to which one of the above anesthetics has been added, is useful. The anesthesin candies may also give relief at times. The nourishment must be liquid; highly seasoned soups are to be avoided because of the pain they cause.

Ulcerative Stomatitis.—Ulcerative inflammation of the buccal mucous membrane is found only when teeth are present and most commonly in children after the sixth year. It begins as a purulent gingivitis at a molar tooth, recognizable by a yellow line, and soon identifies itself by a discolored necrotic sloughing. By the destruction of the gums, the roots of the teeth are bared and the teeth themselves loosened. In milder forms the inflammation does not spread; in the more severe, it may cover the whole alveolus and be carried to neighboring parts of the mouth and tongue, and at times

even to the palate (see ulcerative angina). The intense fetor is characteristic. External adjoining tissues, as well as the regional lymph nodes, become swollen. Fever and other general symptoms usually occur with these lesions and with the pain and the far-reaching influence upon feeding, a severe disease-picture is produced.

In spite of this, the prognosis is usually not bad. Normally, the process ends in one to two weeks and scar formation begins. But in weak children, deep necroses, similar to noma, and local and general septic complications may set in and cause death.

The occurrence of ulcerative stomatitis only when teeth are present and especially when these are uncared for or carious, gives direct indication of the cause. In fact, we find in smears from the ulcers, fusiform bacilli and

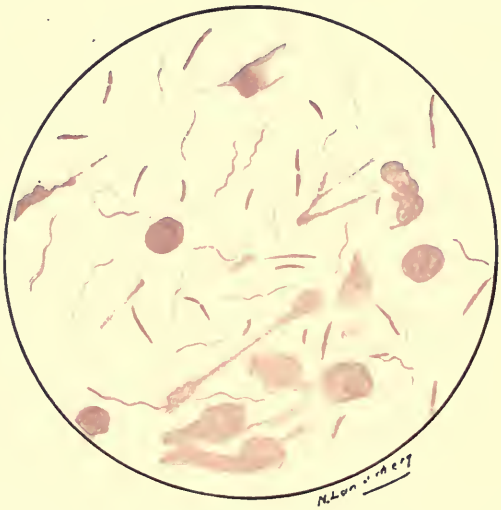


Fig. 71.—Fusiform bacilli and spirilla in ulcerative stomatitis. (Berlin Orphan Asylum.)

spirochætes (Fig. 71) associated, as they are in carious teeth, and their etiologic significance can hardly be doubted. Of course, they may not be looked upon as primary disease producers, and that their pathogenicity demands a previous lowering of the general health is shown by the fact that ulcerative stomatitis occurs most frequently in cachectic children or those weakened by other infections. The microscopic identification of the bacteria is important for diagnosis. For differentiation, mercurial stomatitis and scorbutic stomatitis must be considered.

The treatment is that of all stomatites. The feeding, especially, is of extreme importance. For local treatment, cauterization, once or twice daily, with a 5 per cent. solution of chloride of zinc, or penciling with tincture of iodine or with 95 per cent. phenol by glass rod, is recommended. Dusting with antiseptic powders, xeroform, iodoform, bismuth-iodogallate, in small quantities to prevent poisoning, is also useful. Ulcerations in the recesses of the mouth are favorably influenced by applications of

iodoform gauze saturated in a solution of aluminum acetate. Recently intravenous injections of salvarsan and local applications of a 10 per cent. solution of the same in water, glycerin or oil, have been used in adults with surprising success and this treatment should also receive consideration in extreme cases in children.

Hemorrhagic stomatitis in infancy and early childhood is usually a symptom of scurvy. Other infectious hemorrhagic stomatites, also of a serious nature, occur but rarely at this age.

In older children we occasionally see a condition resembling scurvy, without its usual history, which may end in death by uncontrollable bleeding.

Noma.—Gangrenous stomatitis, also called noma or water cancer, occurs, with few exceptions, only in children who are initially feeble and have been affected by other diseases (chiefly measles, typhoid, diphtheria, disturbances of nutrition, etc.). It usually begins opposite one of the premolars with a small discolored infiltration on the mucous membrane of the cheek, which soon pierces the cheek and is then seen externally as a small brownish spot. This spot rapidly takes on the black color of necrotic tissue; it enlarges and while the central portion disintegrates, the peripheral lines spread with startling rapidity, so that a large part of the cheek or even the whole side of the face may be affected in a few days. After the sloughing of the foul, putrid mass the interior of the mouth is exposed. The process is not terminal and continues to necrosis of the bones. At the borders, toward the normal tissue, we find edematous swelling with little or no inflammatory reaction. The general well-being is soon affected; fever, cachexia, and diarrhœa consume the strength of the patient and usually cause death. Most cases last but a short time; only a few extend over several weeks. Spontaneous healing does occur, but very rarely. Scar formation in the lesions naturally takes a long time.

In the etiology of noma, cachectic conditions are of the same importance as in ulcerative stomatitis. The agent which acts with such fatality upon susceptible soil is not yet definitely known. In many cases, a massed grouping of a cladothrix-like micro-organism, growing, in threads at the border between the diseased and normal tissue, has been found and there are many indications that this may be of etiologic importance. An occasional case, perhaps may be caused by diphtheria.

Treatment.—But little is to be expected from internal medication. The antiseptics (H_2O_2), as well as the caustics (40 per cent. chloride of zinc paste), are useless; nor is the actual cautery any better. The best results have been reported from energetic surgical interference, the excision of all diseased tissue, bony structures as well as soft parts, followed by the cautery. Plastic operations are necessary after healing.

Thrush.—Thrush is peculiar to infancy. In older children it occurs rarely and only accompanies extreme cachexia, as in adults. In the infant, however, it may occur upon very slight disturbance, the severe forms only appearing with more marked cachexia.

The disease appears first in the form of pinhead, or larger, white, and

slightly raised patches (see Fig. 70), which later become confluent into thick flat deposits which may occupy the whole mouth and take on a yellowish or reddish discolorization from blood-stain. They are quite adherent and consist, as is shown by smears under the microscope, of thread-like mycelia and round shining gonidii of a fungus which, according to Plaut, is closely related to the monilia candida, which anchors itself by the growth of its mycelia between the epithelial cells.

It was formerly supposed that thrush demanded severe general disease for its development. Today, however, it is known that it is due to a comparatively harmless nosoparasite and that diarrhœas and other severe or fatal general diseases, in connection with which it is found, are not its results, but rather create a predisposition which permits its growth as an intercurrent malady. However, even children with such diseases may remain almost or entirely free from thrush, so long as the abuse of mouth-washing does not produce lesions of the buccal epithelium and a traumatic stomatitis which appears to be absolutely necessary for the growth of the fungus. The consensus of experience of all institutional physicians has taught us that with the prohibition of mouth-washing, thrush, which formerly could not be exterminated, disappears from the wards.

Thrush itself is not dangerous. With a very rare involvement of the larynx, however, a stenosis may occur, or when the pharynx is affected there may be difficulty in swallowing. There is reason to believe that the fungus may get into the general circulation, causing embolic abscesses or even severe general disturbances (Heubner).

The most certain prophylactic against thrush is the omission of mouth-cleansing; and, added to this, the increase of the resistance of the child by proper nutrition. The normal mucous membrane of the mouth is not a favorable soil for the fungus. Thrush will heal if the mouth is left alone and the general health is improved. All penciling or rubbing hinders healing, or at least delays it. An aid to healing may be secured from the sucking of pacifiers consisting of gauze-covered cotton tampons saturated or powdered with fungus killing substances [boric acid powder and benzosulphinide (saccharin) or 20 per cent. boroglyceride].

ANOMALIES OF THE TEETH AND TEETHING

The presence of teeth at birth is not uncommon. Frequently the central incisors are present, although the bicuspids or canines have also been observed. They are probably caused by a misplacement of the dental germ and in this event are without roots and being loose in the gum should be removed. Or they may be developed from the alveolus as a result of an early germ centre or an accelerated growth. In the latter variety, inflammation of the peridental membrane is frequent. This may cause necrosis of the alveolus if the tooth is not extracted.

Early appearance of the teeth is noted occasionally as a family peculiarity. It may occur in connection with general and sexual precocity. The chief cause of retarded and irregular dentition is rickets. Of course, other

facts (severe disturbance of nutrition, myxedema, mongolism, etc.) may enter into its causation.

In most cases, dentition occurs without symptoms. The old doctrine of difficult dentition, which taught that fever, convulsions, mental symptoms, cough, dysenteries, eczemas and the like may be caused by the appearance of the teeth, is today accepted, if at all, in but a very limited and circumscribed sense. Restlessness, disturbance of sleep, local pruritus, slight stomatitis may possibly be caused by dentition. It is possible that variations of temperature and crops of "tooth rash" may be directly connected with it, especially in neurotic children. This also applies to the occurrence of convulsions, which must be considered as manifestations of spasmophilia. All other "teething symptoms" are more than questionable. According to Heubner, the phenomena of growth in the jaw during dentition are of sufficient importance to cause a higher demand upon the total energy of the body and may thus make the child more prone to exhibitions of spasmophilia.

Disturbances of the dental follicles also occur with the development of the permanent teeth and appear in early years in the form of erosions, cracks, notching, softening and tendency to early caries. Rickets plays a major part in their causation as, also, does congenital syphilis which produces the so-called Hutchinson's teeth. A circular green deposit at the base of the temporary teeth or, more commonly, circular caries is especially frequent, but not pathognomonic, in scrofulo-tuberculous children (H. Neuman). It justifies, to a certain extent, a suspicion of this condition. The temporary dentition should receive the same care as the permanent set. Brushing with a soft brush with a non-gritty dentifrice is an easily acquired habit. The early loss of teeth from the jaw delays its proper growth and also affects the permanent teeth. Anomalies of position favor the occurrence of caries and its complications and early orthodontia is advisable.

DISEASES OF THE SALIVARY GLANDS

Of the acute diseases of the salivary glands (chiefly the parotid) other than mumps, it is necessary to discuss only those that are secondary to purulent inflammations of the mouth. The most frequent of chronic conditions is ranula, the congenital or acquired retention cyst of the sublingual gland. As rarities, many others may be mentioned, especially in the parotid; new growths, swellings of intermittent character, ptyalolithiasis, tuberculosis and a chronic inflammation belonging to the symptom-complex of Mikulicz.

DISEASES OF THE TONSILS, THE PHARYNX AND THE ESOPHAGUS

Angina

Angina is the name generally applied to inflammatory diseases of the pharynx and nasopharynx which occur in the child, as well as in the adult, with extreme swelling of the adenoid tissue involving the "lymphatic ring" (Waldeyer), which surrounds the fauces and the nasopharynx,

resulting in the hypertrophy of the tonsils and adenoids to tumor-like masses. This region is usually involved in its entirety; so that the customary differentiation of palatal angina (tonsillitis) from retronasal angina is not always justified, even though the different parts are affected in varying degree. In the first year of life, the affection of the faucial tonsil is much less frequently encountered than in later years, while the retronasal form is more common.

CATARRHAL ANGINA AND EXUDATIVE ANGINA

The cause of anginæ, if we except those which are symptoms of general infectious diseases (scarlet fever, influenza, cerebrospinal meningitis, etc.), are the usual pyogenic organisms (such as streptococci, pneumococci, micrococcus-catarrhalis, etc). In many cases it is an auto-infection in which an accidental cause (exposure to cold, vocal strain, or other diseases), lowers the resistance of the body and gives the pathogenic organisms inhabiting the mouth opportunity to grow. On the other hand, the disease may be carried from person to person, so that true epidemics occur. Occasionally, the possibility of *scarlatina sine eruptione* must be considered. Aside from this, an individual predisposition seems to exist because of which some children are especially prone to anginæ. More important even than predisposition in the local sense (hypertrophy, the tonsillar crypts acting as reservoirs of infective material), is the general predisposition, dependent upon lessened immunity, which is probably connected with lymphatic conditions.

The general symptoms are those of an infection; acute onset, with chills, fever, headache, malaise, convulsions in the spasmophilic and, frequently, initial vomiting and diarrhœa. Older children, usually but not invariably, complain of pain in swallowing. The submaxillary and cervical glands swell and become painful. A foul breath is noticeable and, with severe grades of swelling, muffled speech and probably stridor. Upon inspection, the cause of these symptoms is found to be one of the various forms of angina.

Catarrhal angina is an inflammation characterized by hyperemia, mucous secretion and enlargement of the lymph nodes. The epithelium has a lack-lustre appearance; small hemorrhages occur, but no membrane appears. This form includes the larger number of mild attacks, but may be accompanied by severe symptoms. Its duration is usually only from two to three days, but it may last longer.

Follicular angina is to be differentiated from the catarrhal form by a marked swelling of the lymph follicles of the tonsils which show gray and, later, yellowish, round, flat or raised lesions. These are equally distributed over the tonsil and may drop out or cause superficial ulcerations.

Lacunar angina differs from the above form in the appearance of a gray or grayish-yellow mucopurulent exudate which, at first, may cover the whole tonsil, but later is rubbed off from the exposed parts so that the exudate is found in the crypts only, and therefore gives a lacunar appearance. The general and local symptoms are usually more severe than in the

former types. When there is a recurrence, or the spread of infection from one side to the other, the disease may be of long duration.

Circumscribed, multiple, fibrinous exudations upon the tonsils, the palate, etc., are not uncommon in grippal angina. Widespread infiltration may occur in septic forms.

The inflammation of the pharyngeal tonsil (retronasal angina, adenoiditis, pharyngeal angina, pharyngitis superior) is of great importance. The severe interference with nasal breathing, the rather nasal than palatal voice sounds, the involvement of the ear (sharp lancinating pains, slight deafness), the purulent discharge from the nose, are diagnostic of this condition. Upon inspection, the nasopharynx may be seen to be covered with mucous and pus. Digital or rhinoscopic examination shows the tonsil inflamed and greatly enlarged. The cervical glands are swollen. The course and duration resemble that of lacunar angina, but in this condition long continued fever is common.

Variations of the usual course are frequent. In some cases, excessive gastro-intestinal symptoms may simulate typhoid, and in patients of excessive nervous irritability meningismus may arise. In retronasal angina especially, we have fever of long duration and at times with very slight local manifestations. Fevers of one, two or more weeks duration, of a remittent or intermittent type, which lead one to suspect the presence of pus, but usually end benignly, are also of importance. Further, we may have chronic recurring anginæ, the attacks of which follow one another so closely as to cause serious disturbance.

The number of complications is very large indeed; tonsillar abscess, otitis media, exanthemata, diseases of the respiratory organs, lymphadenitis with pus formation, in particular, and others.

Glandular fever (Filatow and E. Pfeiffer) is the term applied to a disease which is recognized by a reddening of the pharynx, acute swelling of the cervical glands and frequently by involvement of other lymph node groups, even the mediastinal and mesenteric, and which, in spite of severe symptoms, usually goes on to recovery, without abscess formation, after a duration of weeks. In this disorder, we probably have to deal, not with an entirely separate disease but with a retronasal angina with very slight local symptoms. Glandular fever frequently occurs in epidemics and probably stands in close relation to la grippe.

When we consider that angina represents a local septic infection, it may be readily understood that its sequelæ present many conditions which appear as symptoms of rheumatic or general septic infection. To this class belong erythema nodosum and exudativum, purpura, nephritis of hemorrhagic type, serous and purulent inflammation of the joints, the heart and body cavities, osteomyelitis, etc.

The diagnosis of angina is made by inspection. This should be done, even when no subjective symptoms call attention to the throat. The consideration of an inflammation of the regional lymph nodes is important. The differentiation from scarlet fever may cause great difficulty in cases with erythematous eruption. In these instances, the indefinite demarcation

of the reddening of the throat and the absence of the rasberry tongue is of importance. In diphtheria the exudate is in patches and is not lacunar, although we may have a real lacunar angina with diphtheritic cause, which may be distinguished by bacteriologic examination or by the sequelar paralysis alone.

Treatment.—Because of the undeniable infectiousness of many anginæ, isolation is advisable. Treatment consists of rest in bed, careful feeding, cold applications to the neck, even of ice packs; gargling with salt water and glycerin, lemon water, weak solutions of aluminum acetate, lime water, etc. Internal remedies, *e. g.*, potassium chlorate 2 per cent., 1-2000 cyanate of mercury, are better avoided.

Sweat-producing measures are often useful. For the swollen lymph nodes, hot applications, causing local hyperemia, give good results both in reducing the swelling and aiding abscess formation. To prevent recurrence, a change of constitutional conditions should be brought about by diatetic and hygienic prophylaxis.

Membrano-ulcerative Angina—(Vincent's angina, Plaut's diphtheroid angina, *angina a' bacille fusiform.*) In membranous angina, we find tough, spreading membranes which are due to an extension of the necrotic exudate into the mucosa. After the sloughing of these necrotic membranes, distinctly circumbscribed ulcers of varying depths are seen. The entire pharynx is swollen and there is a tendency to hemorrhage from the mucous membrane. A strong fetor, resembling that of ulcerative stomatitis, is characteristic.

Distinction is to be made between the milder diphtheroid form, with more superficial membrane formation and smaller ulcers, and the membrano-ulcerative form with deeper and more widespread lesions.

In many cases, the fever and general symptoms, as well as the objective signs, are extremely mild, so that only the routine examination of the throat brings out the cause of the illness. The contrast between the mild general symptoms and the severe local changes is, in fact, quite characteristic of the disease, although cases are seen with high fever and typical anginal symptoms.

The course of the diphtheroid form resembles the ordinary anginæ. Transitional forms exist. In these, the long duration, the slowness of the sloughing process and the tendency to recurrence are quite characteristic. The disease usually ends in recovery; only exceptionally, and probably in cachectic children, have extended necroses, with a fatal result, been reported. Complications are hardly to be feared.

The same fusiform bacillus and spirochæta that cause ulcerative stomatitis, may be assigned as probably etiologic factors. A certain infectivity undoubtedly exists in grouped cases observed in families, houses or barracks; but the transmissibility is evidently not very great.

Together with the throat findings and the characteristic fetor, the demonstration of the fusiform bacillus and spirochæta in a smear stained with fuchsin, is diagnostic (Fig. 71). In the differential diagnosis, diphtheria and syphilis must be considered. This form of angina often resembles the latter disease so much that French authors have applied the term

"chancriform" to its lesions. The absence of other signs of syphilis, the negative results of cultures for diphtheria bacilli and, finally, its course are diagnostic.

The treatment is that usual for anginæ. The cleansing of the ulcers may be attempted by touching with hydrogen peroxide and by the application of antiseptic powders. Of late, good results have been obtained, as in ulcerative stomatitis, with salvarsan.

Rare forms of gangrenous angina, of unknown origin and of doubtful prognosis, with extremely foul-smelling, progressive disintegration of the entire throat and a tendency to hemorrhage are seen. The very fatal serous or seropurulent pharyngeal phlegmon and pharyngeal erysipelas may also occur in the child.

HYPERPLASIA OF THE TONSIL

Tonsillar hyperplasia, leading to a globular or longitudinal tumor, frequently extending far downward into the pharynx or developing into a ragged polyp-like growth, is closely related in its etiology and course to adenoid growths (*q. v.*). Treatment should not be too energetic. It is generally well to await a gradual physiologic recession. Tonsillectomy is to be advised only in case of large protruding tumors. The hope of curing a frequently recurring angina by snipping off the tonsil is very slight. A harmless diphtheroid membrane is always formed upon the wound after the operation. It must not be forgotten, however, that true diphtheria may occur there.

Tumors of the pharynx are usually congenital or are formed upon a congenital base; and among them we must consider dermoids, lipomata, fibromata and fibrosarcomata (so-called nasopharyngeal polypi). The most serious is the lymphosarcoma of the tonsil, which must be considered in cases of rapidly growing unilateral swelling of the tonsil.

RETROPHARYNGEAL LYMPHADENITIS AND RETROPHARYNGEAL ABSCESS

The lymph channels of the nasopharynx pass, in part, on their way to the cervical nodes, through lateral pharyngeal groups which lie at the level of the atlas behind the tonsils. These in turn communicate with the deep cervical nodes. They may also be connected with small, inconstant nodes which become obliterated during childhood and which lie in the median line, at the level of the odontoid axis, imbedded in the fascia. In the event of inflammatory conditions in the territory drained by these glands, they may enlarge, become inflamed and finally suppurate. This is the cause of the occurrence of retropharyngeal lymphadenitis or retropharyngeal abscess.

The primary process is usually a coryza or a retronasal angina. The condition may also be connected with lues, measles, or scarlet fever. Further, wound infection from rhagades and ulcers must be considered. Idiopathic lymphadenitis, of which older physicians speak, does not exist. Streptococci, and less frequently influenza bacilli or other micro-organisms are etiologic factors.

The disease occurs especially in the first two years of life. Later on it is less common, probably because of the obliteration of the nodes involved. A simple swelling of the lymph nodes is quite frequent; the severer forms, on the contrary, are comparatively rare.

The first stage of the disease causes no symptoms aside from fever and only by digital examination can the movable swelling, which may be of the size of a bean or hazel-nut, be discovered. More severe symptoms appear when the swelling becomes larger and the infiltration of the surrounding connective tissue more extensive. Among these may be mentioned dysphagia, rattling and gurgling in the throat due to the collection of mucus above the obstruction, a muffled voice, and pharyngeal stridor. The rigid pose of the head, at times resembling torticollis, taken to guard the painful swelling from pressure, is notable. The superficial glands are frequently involved. The condition may cause attacks of suffocation or, extending far down, may produce edema of the larynx. The swelling of the throat may then be noted upon inspection; but better information is given by palpation, which shows whether there is still a hard infiltration or a fluctuating abscess.

Course.—Many simple swellings and tissue infiltrations recover spontaneously; only a few go on to abscess formation. In the latter cases, recovery may set in after evacuation. Frequently, the neighboring nodes are affected and, becoming suppurative and confluent with the retropharyngeal group, form a large abscess. Extension into the mediastina and later, pyemia may occur. Spontaneous rupture may cause death by inspiration of the pus.

With early recognition and treatment the prognosis is favorable.

The diagnosis, frequently missed by beginners, is made upon the signs of stenosis and the discovery of swelling by palpation. Differentiation from laryngeal diphtheria, without recourse to digital examination, should be possible by the pharyngeal tone of the stridor, the rattle of the mucous and the rigid position of the head.

The treatment of the non-suppurative stage is that of angina in general. Abscess demands immediate incision, either with an ordinary scalpel wrapped with adhesive plaster to the point, or with a special knife (Schmitz or Carstens). Perhaps it is still more satisfactory to use a slender, curved, sharp, rat-toothed forcep. The incision is made with the guidance of the finger, the child being held in an upright position. As soon as pus appears, the body should be bent forward to prevent aspiration and the opening should be gradually increased. This does not insure absolutely against an attack of asphyxia. The wound usually heals rapidly and only exceptionally will it be found necessary to reopen it. In case of large confluent abscesses pointing outward, the opening should be made from without.

Occasionally a *chronic tuberculous, retropharyngeal glandular swelling, glandular abscess* or *mediastinal abscess*, due to gravitation, may occur. Their treatment is that of tuberculosis in general. Incisions are to be avoided. In cases with symptoms of obstruction, aspiration with a syringe is recommended. *Retropharyngeal abscess due to acute osteomyelitis of the bodies of the vertebræ* is very rare.

CONGENITAL ANOMALIES OF THE ESOPHAGUS

The congenital anomalies of the esophagus are chiefly stenoses or atresias. *Congenital atresia*, which may occur in many different forms, as entire absence of the organ, absence of the upper or lower portion, a cul-de-sac, a circular closed channel, communication with the trachea or bronchii, is recognized immediately at birth by the fact that even the smallest quantity of nourishment is vomited at once in an unchanged condition. This vomiting may occur with attacks of suffocation which lead to the conclusion of a connection between the esophagus and the respiratory passages. The esophageal bougie brings up against an impassible barrier at very short distance. The unsuccessful attempt to aid such infants by gastrostomy has always proved fatal. The rare *congenital stenoses*, to be differentiated from acquired forms by the fact that the narrowed portions show a normal structure of the connective tissue, may exist for months and years, and even be latent until old age. Only the severe forms cause symptoms of difficulty in swallowing, gurgling of mucous, rumination, and temporary occlusion. The bougie shows the presence of an obstruction. Congenital diverticula have occasionally been reported.

ACQUIRED DISEASES OF THE ESOPHAGUS

Corrosive Esophagitis.—Numerous cases of burning of the esophagus with strong acids or caustic alkalies (lye and soap powders) which children have swallowed or which have been accidentally administered by adults, still occur. The action of the caustic, in milder forms, is shown by a croupous necrosis of the epithelium which may heal without scar formation. With more severe lesions, the entire thickness of the mucous membrane is eroded, deep sloughing occurs, followed by circumscribed ulcers which may perforate. Healing, with stricture-forming cicatrices, results.

The symptoms of corrosive esophagitis are local pain, vomiting of bloody masses and pieces of the mucous membrane even to the extent of the entire esophagus. Added to these, is a tendency to collapse. Many cases end fatally, while in others convalescence sets in. But in convalescence a new danger threatens at the end of several days, in the erosion of, and hemorrhage from neighboring blood-vessels, perforation with mediastinitis, and pyopneumothorax. If these later symptoms do not occur, those of stenosis may appear subsequently. Of those poisoned with sulphuric acid, over one-half die; of those with caustic alkali, one-fourth. The survivors have strictures, more than one-half of which are severe.

The stricture is usually located in the upper third, less frequently in the middle or lower third of the esophagus. Above the obstruction, the esophagus is widened and hypertrophic, the mucous membrane being frequently inflamed and covered with ulcerations. The nature and severity of the condition depends upon the grade of the burning. The symptoms of the stricture appear two or three weeks after the poisoning and are those of obstruction to the passage of the swallowed food.

The diagnosis is made from the history and the condition of the cicatrix is

established by examination with the sound or the esophagoscope. The prognosis is better than in adults. With proper treatment, recoveries are reported in from fifty-six to sixty per cent. of the cases.

The treatment of the poisoning consist in efforts to neutralize the caustic. Organic acids, chalk, calcined magnesia are employed; carbonates, because of the formation of CO_2 gas, should not be used. The pain may be controlled by morphin hypodermically or by the application of local anesthetics (10 per cent. anesthesin in oil, novocaine, or alypin, 2 per cent. solution 5-10 drops). Liquid nourishment should be given. The treatment of the sequelæ may usually begin by the third week and consists in gradual dilatation with the bougie and of injections of thiosinamin.

Corrosive esophagitis with its sequelæ is the most common lesion of the organ in childhood. All other conditions are great rarities. Cases of nervous cardiospasm, with symptoms of regurgitation and rumination, as well as stenosis following syphilitic ulcers, are known.

NUTRITIONAL DISTURBANCES OF INFANTS

Introduction.—The understanding of the disease conditions discussed in the following paragraphs has undergone enormous change within the last three decades. Formerly physicians recognized in them nothing more than diseases of the stomach and intestine which could be considered analogous to the fermentative dyspepsia, fermentative catarrh, gastro-enteritis and diarrhœa with vomiting, in the adult; the physiologically slight resistance of the infant predisposing it to a severe course. Example of this is seen in the teaching of the Vienna school, which was able to obtain general recognition through the classical presentations of Widerhofer. From the study of anatomical findings, this school distinguished severally, the purely functional, the catarrhal, and the inflammatory ulcerative lesions of the intestine; each of which could be recognized clinically by the varying consistence of the stools and the degree of constitutional disturbance. This conception further enabled these authorities to recognize (1) acute and chronic dyspepsia, as subdivisions of the purely functional disturbances; (2) histologically and clinically severe entero-catarrh; (3) follicular enteritis chiefly of the large intestine, belonging to the inflammatory type and (4), the extreme form of entero-catarrh, formerly known as cholera infantum, as final subdivision of this group.

Useful as this system was in its time and excellent as the observations were upon which it was based, further advance of knowledge has made its abandonment necessary. With great difficulty could cases in actual practice be classified under these sharply differentiated forms of disease, in view of the frequent occurrence of intermediate and transitional forms. In infancy, inflammatory conditions occurred which resembled the intestinal affections of the adult; but these, again, were greatly exceeded, alike in frequency and importance, by disturbances which had to be considered purely functional. Identical and entirely normal findings in the bowel were shown in cases presenting the most variable clinical symptoms in which, according to previously accepted teachings, severe anatomical lesions were

to be expected. It has become clear that in infancy, as in later life, functional alterations of the gastro-intestinal system underlie pathological conditions, but these, in the infant organism, give rise to so many and such important changes in the general health that we may no longer speak of them as merely organic, but rather as general diseases; and, further, as general diseases so exceedingly grave that frequently the entire symptom-complex is characteristic of, and the prognosis dependent upon the constitutional status rather than upon the intestinal lesion. Again, it has been found that the bacteriolcgic etiology of these diseases does not play the essential rôle formerly attached to it. Both acute and chronic disorders, either of the mildest or the most severe form, may be of purely alimentary origin, and numerous diagnostic symptoms, formerly supposed to be caused by bacterial toxins, may be the results of a direct food poisoning. In a word, disturbances created by intestinal lesions are commonly seen to induce so complete and so severe derangement of the entire metabolic process that the term gastro-intestinal disease has become too narrow. In its place has appeared the phrase "disturbance of nutrition."[1]

In view of this conception the necessity of distinguishing the nutritional disturbances of the breast-fed infant from those of the artificially-fed at once becomes obvious. The very feeding of a food other than that intended for the infant by nature is a pathologic condition. Such feeding lowers the resistance and may very readily be the cause of disease. Only rarely does the bottle-fed infant thrive anywhere near as well as the breast-fed. The tendency to rickets, spasmophilia and anemia is very great. The immunity against infection is reduced and the tendency to the conditions, to be discussed *i. e.*, the disturbances of nutrition, is exceptionally great.

DISTURBANCES OF NUTRITION OF THE ARTIFICIALLY-FED INFANT

General Etiology and Pathogenesis

1. Milk.—The increased tendency to disease, and the actual causation of disease in infants fed upon milk other than that intended for them in the natural process of nutrition, *i. e.*, mother's milk, is not dependent to any great extent upon such accidental factors as the bacterial contamination of the milk or the improper combination of the food elements, etc. It may be accepted as proved, rather, that this tendency to and actual increase in the morbidity of bottle babies is due primarily to the milk itself, simply because it is not suitable for the human young, falling short in many essential requirements. The morbidity is exceedingly great even in those artificially-fed infants in whose feeding no criticism can be brought against the technic or the food mixture. For an explanation we must accept the theory that the specific peculiarities of cow's milk are directly or indirectly the causative factor. And this brings us to the outstanding problem of all artificial feeding, *i. e.*, what is there in cow's milk which, though it be modified to resemble mother's milk in every respect, still leaves it so inadequate a food for the human young?

[1] Ernaehrung Stoerung, Czerny.

The first attempts at explanation were made by Biedert when he put forth the proposition of the indigestibility of the casein of cow's milk. He claimed that the absorption of the products of protein digestion was very poor in the bowel and this formed the basis for the injury. At present, this explanation seems to be successfully controverted by the consistent findings of more careful chemical examination of the stools, the digestive processes in the intestine, metabolism experiments and clinical observation. Similarly the theory of Hamburger that the injury is due to toxicity of the foreign protein is not born out clinically. Indeed, no definite proof has been brought forward to show that the protein is the prime factor in the etiology. There is no doubt, however, that fat and sugar have a harmful action. But even these substances are not primarily the cause of the nutritional disturbance except when fed in improper dosage. This fact is demonstrated by the more recent researches, which show that by changing certain other constituents of the food, as for instance; increasing the percentage of protein, reducing the amount of whey or replacing the carbohydrate by one less fermentible, immediately relieves the disturbance. Others have attempted to show that the injurious factor lay in certain chemically undemonstrable substances in the whey. According to Schloss, it is hardly probable that the whey has a distinctly injurious action on the intestinal function. According to the theories of Marfan, Concetti, Escherich, Pfaundler and others, the great advantage of breast-milk is due to certain specific ferment-like substances which act as a stimulant to growth and development. The absence of such substances in cow's milk or rather, the fact that these substances in the milk of various species are not interchangeable is the cause of the failure of artificial feeding. If these substances are contained in the whey, the deleterious action of whey is easily explained. Even without bringing to our aid the theory of such hypothetical ferments, it can be conceived readily that the physical and biochemical structure of the whey of cow's milk is such as to make it a fluid in which the digestive function of the intestinal mucosa is much more difficult than in the medium of breast-milk, which also has a certain catalytic action (Finkelstein and Meyer). Strong, lusty infants can call up the reserve strength necessary to overcome this hampered action of the digestive apparatus, while weak infants succumb.

Some authors do not believe in the theory which blames the injury to the specific action of any one foreign substance in the milk, but lay the difficulty to the varying relation of organic and inorganic components.

2. Tolerance.—In infants with strong metabolic powers, overfeeding will cause merely an excess of fat (Fig. 72) which can hardly be rated as a disease. Very commonly the symptom of excessive fat is combined with manifestations of rickets, spasmophilia or exudative diathesis. Very few infants, however, have a digestive system which will withstand overfeeding for any length of time; signs of dystrophy or dyspepsia supervene and, if the excess of food be continued, even more serious conditions result. In some cases, indeed, the actual excess of food does not have to be so very great, for there are infants that become ill when the amount of food does not sur-

pass or is below the physiologic requirement. Such infants may be said to have a low tolerance. This injurious excess may be due to too great volume of a properly modified food as is seen in the "overfeeding dyspepsia" of the breast-fed infant or it may be due to too much of one ingredient in the mixture. The injuries of high sugar or high flour feeding serve as examples of the latter.

3. Composition of the Food.—Closely related to the above category of causes of nutritional disturbances is that group in which an improper rela-

FIG. 72.—Adiposity in an overfed eight-month-old child, weight 9.4 kilos (21 pounds). (Gisela Children's Hospital, Munich, Prof. Ibrahim.)

tive balance of the individual food components forms an essential factor; "*nutritional disturbance ex correlatione.*" The correlation of the ingredients of cow's milk are, in themselves, not adapted to the infant. In addition to this the infant is rarely fed with natural cow's milk but with cow's milk to which innumerable other things, such as water, flour, fat and various carbohydrates, have been added. As a result, the correlation of the elements of the food becomes even more abnormal and resultingly more liable to produce disturbances. Such an etiology is probable, for example, in those cases in which a persisting dyspepsia is relieved by the addition of a small amount of casein to the formula without other change. Or in the reverse, infants that have not thrived may be made to develop normally by reducing the milk

and increasing the carbohydrate. Very slight changes in the correlation may cause marked changes in the nutritional status of the infant either for better or for worse.

According to our present conception, the transition from the tendency to disease, to disease itself, results from a disproportion of the demand upon, and the ability of, the metabolic functions leading to irregularities of the digestive and resorptive powers which in turn results in pathologic bacterial growth and putrefactive or fermentative processes. This adds a new factor to the complex for harm in the shape of changed endogenous fermentation. According to Moro, Tobler and Bessau the ascent of this fermentative process into the normally almost sterile small intestine is of greater importance than the increase of this process in the large bowel. For here, at the very seat of the digestive function, slight changes in the chemical process are greatly magnified in their importance to the organism. The form of the decomposition depends largely upon the composition and correlation of the food. Usually the disturbance affects the digestion of the fat and carbohydrate. This results in the formation of acids of fermentation which are usually looked upon as pathogenetic of these conditions. Recently several authors have added to this "fermentation injury," the conception of a "putrefaction injury" which is caused by an excessive decomposition of protein on the basis of the acid formation (fat and sugar). This condition is supposed to cause diarrhœa and other symptoms of bowel irritation in its acute form. In its chronic form it is characterized by far reaching general manifestations and putrefactive stools.

4. Inanition.—Underfeeding also plays an important rôle in the causation of nutritional disturbances. New-born infants are often greatly underfed because of the fear that a cow's milk mixture might be harmful, and for the same reason children with intestinal disturbances are often underfed for a long time. Aside from simple underfeeding, that is the feeding of a perfectly satisfactory food in too small amount, we must also consider qualitative underfeeding. This condition obtains when the caloric requirement is fully covered, but one or more essential elements of the diet are below requirement. Normal development can occur only when the requirement of every food element including the inorganic substances is fulfilled. Frequently quantitative and qualitative inanition may be combined as in the continued feeding of thin gruels.

The usual results of moderate inanition are failure to gain in weight or slight gradual losses. Persisting inanition leads to subnormal temperature and slowing of the pulse. The condition may be quickly relieved in the early stages by the addition of liberal amounts of suitable food. In the cases of long standing, however, the tolerance is markedly lowered and any sudden increase of food may bring on extreme diarrhœa and toxic manifestation and death. In new-born and young infants, inanition may cause vomiting and frequent loose bowel movements, a hunger dyspepsia.

5. Infection.—In contrast to the etiologic factors of disturbances of nutrition of purely alimentary origin and without other provocative influences, we have another category of nutritional disturbances which must be

considered as secondary to other forms of injury to the general infantile organism and affecting the gastro-intestinal function especially. The digestion is so definitely injured that food which was well accepted can no longer be borne. This form of reduction of the tolerance as a result of exogenous weakening may be, to a certain extent, contrasted directly with the reduction of tolerance by excess feeding. The most characteristic member of this group is the disturbance of nutrition resulting from acute or chronic infection. The bacterial poisons injure the entire cell structure of the body and, of course, this also affects the organs of digestion. As a result their functional capacity is reduced, and, unless the food is promptly changed to meet the altered tolerance, a complicating alimentary disturbance is added to the infectious process. The complication may become grave enough to govern the entire clinical picture and obscure the original infection. Recovery from the nutritional disturbance may take place before the infectious process disappears or the two may be relieved coincidently or, finally, the digestive trouble may persist and become chronic as an independent disease.

6. Milk Infection and Milk Toxins.—The etiology of a nutritional disturbance caused by the bacterial content of milk or by milk toxins is identical with that of the condition above. Formerly the bacterial content of cow's milk or the chemical products of their activity before ingestion was held to be the chief etiologic factor in the digestive disturbances of artificially-fed infants. At present, however, but slight stress is laid upon this phase. The majority of milk bacteria are harmless saprophytes, which may be ingested in large quantities without harm. It must be admitted that pathogenic organisms are sometimes found in dirty milk. Furthermore, in weak infants fermentation processes occur more readily if huge numbers of fermentation producing organisms are added to intestinal flora, than when the food is sterile. It is also possible that injury may come from substances occurring in the milk as a result of improper feeding of the animals, or from the products of decomposition of the milk such as acids, peptones, etc. This possibility is limited, however, by the fact that if these substances occur in any appreciable amount, the taste, odor and even appearance of the milk are such that the mother will not use it or the child itself will refuse it.

Until recently, two important points of argument were urged in favor of the etiologic relation of the contaminated milk, *viz.*, (1) the increase of gastro-intestinal disease and of the death-rate from this cause in the hot months of the year (the summer apex of infant mortality); and (2) the fact that these prevalent conditions affect principally artificially-fed infants while breast-fed babies remain almost wholly immune. The parallel between the statistical evidence and the increase in the contamination of milk during the summer months appeared to be undeniable evidence of the serious pathogenicity of spoiled food. At present, however, even this conclusion is denied and there is an inclination to accept a belief in the harmful influence of heat upon the child, although the exact nature of this influence has not yet been determined. In some cases the summer heat may cause overheating (hyperthermia) producing disease and death which may be considered

closely related to the phenomena of heat stroke. Such disease begins suddenly and runs an acute or subacute course, characterized symptomatically by convulsions and hyperpyretic coma. The conditions may be prevented or cured by the prompt application of fever-reducing measures. In other cases, the gradual effect of the summer heat is to reduce the limit of tolerance, so that alimentary disorders occur very readily and tend to run an especially severe course. Moreover, bacterial infection, entering through the skin eroded by excessive perspiration, appears to play an important rôle during the hot weather and death may frequently occur through such an intercurrent invasion of pathogenic bacteria. Not until we have eliminated all these causes may we entertain the etiologic relations to summer diseases of contaminated milk.

A plausible explanation of the immunity of breast-fed infants from heat injury is also suggested. It appears that an infant with impaired nutrition is much less resistant of heat influences than the normal babe. Since most breast-fed infants are well and very many artificially-fed babies have some slight disturbance of nutrition, the difference in the effects of heat is possibly explained.

In considering the relationship between high temperature and infant mortality, the prevailing custom of burdening young children too heavily with clothing may add to the injuries caused by the heat of summer. How and to what extent these several factors of diminished tolerance produce secondary disturbances of nutrition, is again a question of relation between the degree of resistance and the strength of the attacking forces. In feeble children or in children weakened in any way, especially in those with impaired nutrition, a slight overheating, a coryza, a small phlegmon, or a vaccination, may give rise to serious symptoms. A healthy, normal child, on the contrary, may resist even severe infection, without the appearance of any gastro-intestinal disorder.

7. Constitutional Anomalies.—There are infants that will thrive on any form of artificial food and apparently never have a nutritional disorder even though subjected to severe infections. Others require the most exacting study of the feeding formula, are extremely prone to intestinal and nutritional disturbances, their nutritional function suffering severe upsets as a result of the mildest infection. Every pediatrist sees children that are extremely hard to raise because they respond to the slightest irregularities of the food or to every mild infection with the most stormy general symptoms. These differences in the nature of the individual can be explained only on the basis of a congenital, constitutional tendency that acts in the form of a greater or lesser tolerance. We have no definite knowledge of the basic etiology of such a constitutional difference and no great advance in the understanding is made by looking upon it as a manifestation of "neuropathy" or "hypersensibility" or as a part of the "exudative diathesis." It seems more satisfactory to consider the one group as "trophostabile" and the other as "tropholabile" and leave further discussion for a time when more study has given a clear understanding.

GENERAL SYMPTOMATOLOGY

It has been shown in the introduction to the Chapter on the Disturbances of Nutrition that symptoms of these conditions may be much more varied than has been hitherto supposed. To appreciate the complexity of this symptomatology, we must recall the several phases of the process of nutrition. It includes not only the digestion and the absorption of food from the bowel, its assimilation, the repair of the waste of tissue incident to functional activity, and the process of growth, but also the maintenance of the normal concentration of the tissue-cells, the function of internal secretion and the regulation both of heat production and of heat loss. Accordingly the symptoms of nutritional disturbance are not confined to pathologic alterations in the gastro-intestinal tract, to loss of body-weight, or to an arrest of growth. It is to be expected that variations of body-temperature and perversions of function in many different organs will be met with. The relationship of such symptoms to disturbances of nutrition is shown in the fact that they are often favorably influenced by changes in the form of feeding. There is a type of alimentary fever which disappears almost immediately when feeding is discontinued; an albuminuria of similar origin, and even cerebral and spinal symptoms, with anomalies of cardiac and pulmonary function, which may be relieved at once by changes in diet.

Recognition of the symptoms of disturbances of nutrition presupposes a knowledge of the "normal conditions of nutrition and of the normal metabolic processes."

As superficial evidences of normal health may be noted: the proper development of the musculature and the fat layers of the body, the turgor of the skin and the skeletal tissues, and the normal color of the skin and visible mucous membranes. Broadly speaking, health means the normal functioning of all organs, and particularly of the kidneys, lungs, heart and nervous system; a general development proportional to age; and a due degree of agility and of static and dynamic power.

As indications of especial bearing upon nutritional integrity, the normal activity of the gastro-intestinal tract, the proper adjustment of body-temperature, a progressive increase in weight and the maintenance of natural immunity may be cited.

With a fairly equable atmospheric temperature, the curve of body-temperature of the infant is remarkable for its slight variations (monothermia). The weight-curve shows the same continuity. So small variations are seen with daily weighings that the curve shows an almost evenly rising line. The active formation of antibodies is to be recognized by the infrequency of infections and their ordinarily mild course.

As a final and a most important indication of normal nutrition, we note the standard of tolerance for food; meaning thereby the exercise of a wide range of tolerance toward the food-stuffs ingested. This tolerance is exhibited in two ways; in the quick adaptation of the unimpaired digestive

functions to increased quantities of food without consequent harm, a quantitative normal tolerance; and in the successful response to food in the most varied mixtures, whether of mother's milk or cow's milk, whether in preparations rich in fat and poor in carbohydrates or *vice versa*, in so far as they are adapted to continuous feeding. This response may be defined as the normal reaction to food.

Equally, the healthy child exhibits a normal reaction to and endurance of those external influences which tend to reduce tolerance; *e. g.*, in the large measure of resistance it shows to infectious disease or to abnormal temperature changes, to which it does not succumb unless the attack from without is more than ordinarily severe.

On the other hand, symptoms of disturbance, in the direction of lowered nutrition, are seen in loss of weight, emaciation, increased or diminished tonus, abnormal dryness or increased fluidity of the tissues, and pallor or injection of the skin and mucous membranes. Numerous signs of functional insufficiency are shown in the form of muscular weakness, decreased or increased nervous irritability, the disordered functional activity of various important organs, and especially of the nervous system. Important indications of pathologic changes in the gastro-intestinal tract are to be found in an abnormal curve, characterized by slow rises and frequent drops, in abnormal temperature changes, and in the lowering of the power of resistance to bacterial infections.

It should be observed that either subnormal temperature or fever may be caused by dietetic disturbances. Fever is frequently associated with other symptoms characteristic of fevers due to infections such as cardiac weakness, albuminuria, leucocytosis, etc. "Alimentary fever" or "dietary toxemia" can be distinguished from the results of infection only by their etiology and by their quick response to changes in diet.

Different combinations of the symptoms briefly described above, and variations in their course give us clinical pictures, and sometimes symptom-complexes of contrasting significance. The form which any individual case may take, whether indicated by arrest of development and loss of weight or by symptoms resembling acute toxemia, depends largely upon the kind and amount of food given. Infants with a primarily high tropholability and infants severely injured by alimentary or infectious processes react more definitely to the influences which cause only mild disturbances in children less liable, influences which in strong infants may even be favorable. This peculiar irritability in response to food and other stimuli is known as the paradoxical reaction.

The reduction of the tolerance is not only shown by the variation in the metabolism but is reflected in an unusual susceptibility to external influences. In such patients, infection occurs more readily and tends to serious results, or minor ailments now readily lead to secondary disturbances of nutrition. Summer heat or excessive artificial temperature produces variations in body-temperature or other disease symptoms which may assume a severe type.

CLASSIFICATION

The classification of disturbances of nutrition, into acute and chronic dyspepsia (the latter also called atrophy), entero-catarrh, cholera infantum and enteritis, until recently accepted, is no longer tenable. Since it has become apparent that in these several conditions we have but variant forms and degrees of general alimentary disorder, and since it is known that one form may be transformed into another form by changes in diet, it is no longer fitting to use terms which bring the intestinal symptoms into the foreground and, at the same time, encourage the view that each is to be looked upon as a distinct disease.

The classification of Czerny and Keller gives the alimentary element in the disturbance its proper place. It distinguishes (*a*) disturbances of nutrition due to food (*b*) disturbances due to infection, and (*c*) those due to congenital constitutional defects. Under the first class, we have as subclasses, the various "food injuries," such as those caused by an excess of milk or of flour. Under the second, disorders due to contaminated milk and diseases due to enteral and parenteral infection, such as dyspepsia or cholera infantum under the older classification. Under the third, the diatheses associated with the nutritional problem.

The authors are convinced that an etiologic classification is not only unessential but impossible. In the group as a whole, and even more so in the individual case, the causation is not clearly recognizable nor devisible into its separate factors. Take, for instance, the child that does not thrive on a cow's milk mixture, with the result that the immunity is decidedly reduced. To this primary nutritional abnormality add an infection which will bring on manifestations of dyspepsia. In this case it is obviously impossible to lay the cause to any one definite etiologic factor. In chronic cases leading gradually to atrophy, all sorts of etiologic factors have come into play, either together or in sequence, so that, again, a systematic classification is impossible. These difficulties are avoided if we accept a simple clinical grouping such as the following.

A. Nutritional disturbances without toxic manifestations.

I. Dystrophy.[1] Disturbance of qualitative and quantitative growth.

(a) Without diarrhœa, or slight diarrhœa.

1. Dystrophy as a result of excessive milk feeding (the milk-feeding injury of Czerny and Keller).
2. Dystrophy as a result of excessive flour feeding (the flour-feeding injury of Czerny and Keller).
3. Dystrophy as a result of inanition.

(b) With diarrhœa.

4. Dystrophy with dyspepsia (chronic dyspepsia).

[1] The authors have replaced the older term "Disturbance of balance" by that of "Dystrophy" which seems to bring out the changes noted in the clinical picture and especially, the delayed development more clearly. Langstein suggests the term "hypotrophy" but this is almost synonymous with the term "hypoplasia" which refers rather to a failure of the germ cell.

II. Decomposition, loss of body-weight as a result of imbalance of the metabolic processes with the loss of essential body elements, especially water, because of the advanced degree of alimentary and dyspeptic injury.

B. Nutritional disturbances with toxic manifestations.

I. Acute dyspepsia; acute gastro-intestinal disturbance resulting from abnormal endogenous decomposition of the food, characterized by free purgation and leading to intoxication.

II. Intoxication, severe general disturbance of a toxic nature with terrific losses of weight, probably the result of poisoning by intermediate products of metabolism in combination with severe dehydration.

It is hardly necessary to say that the individual headings in each group do not represent definitely separable disease entity in each case. They should be considered rather as various stages which may at any time progress from the milder to the more severe. Such transition may take place if the food is changed or if the patient be subjected to an external injury which reduces his resistance.

A. Nutritional Disturbances Without Toxic Manifestations.

I. Dystrophy.

(a) Without diarrhœa.

1. Dystrophy as a result of excessive milk feeding. (Milk-feeding injury of Czerny and Keller. Mild form of atrophy.)

Symptoms.—This condition is characterized chiefly by delayed development both in a qualitative and quantitative way without other distinct indications of illness.

Even at the outset, daily weighings show great variations, both upward and downward, so that the curve becomes very irregular. Later, periods of variable duration occur in which there is no change of weight or a continuous loss, again equalized by a rise (Fig. 73). On the whole, the weight falls gradually below the normal average and, although not actually emaciated, the child gives the impression of great debility. Retardation of growth is noticeable, so that the infant is generally smaller than a healthy child of the same age.

The evidences of reduction in the qualitative conditions of nutrition are to be especially noted. The tone of the tissues is reduced, the muscles are relaxed, the abdomen is distended, the skin dry, the color pale, dynamic and static power are below the standard to be looked for at the given age. Disposition and sleep are affected. The reduction of immunity is seen in the tendency to secondary infections, chiefly of the skin, which do not usually extend beyond their local limits.

The stools may be normal or, again, dryer and lighter than usual, or even white in color. Rarely do we see the fat-soap stools, the so-called "gray obstipation." The temperature is more variable than in health. With the exception of the meteorism and occasional vomiting, other evidences of disease of the gastro-intestinal tract are absent.

Etiology.—The origin of the condition described lies solely and primarily in an excess of milk in the diet with an insufficiency or absence of carbohydrate. In many cases the milk was given in too large amounts; in others, the error in the diet appears after a short time even though the food was apparently quantitatively correct. In these latter, it may be supposed that a constitutional peculiarity of the digestive function of the infant plays a part, for innumerable infants thrive on unaltered whole milk for a long time. This inherent peculiarity of the infant is further demonstrated by the fact that the symptom group is often encountered in infants that are receiving as high as five per cent. of carbohydrate and that such children

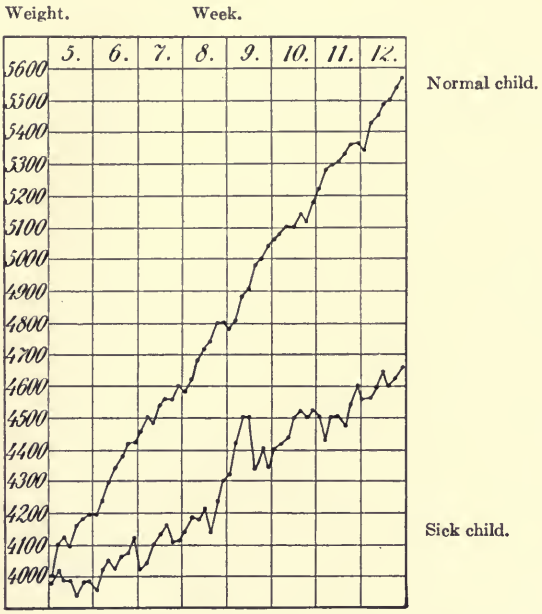


FIG. 73.—Weight curves showing the increase of a healthy infant as compared with one suffering with dystrophy.

often develop satisfactorily if the starch is increased to as high as ten per cent., and even higher.

Pathogenesis.—The explanation of the failure of development in spite of food of sufficient caloric value is usually sought in a disturbance of the metabolism of some one constituent of the milk. Most commonly, the blame has been laid upon the fat metabolism. Recently the suggestion has been made that increased putrefaction in the intestine might possibly be the cause. It seems more probable, however, that the disturbance of the fat metabolism as well as the intestinal putrefaction is secondary to the low carbohydrate. In this case, the condition would be explained as a carbohydrate requirement. Such a condition is characteristic of rickitic infants, of those with spasmophilia and the exudative child.

Metabolism.—The metabolism is apparently not greatly altered so far as the absorption of organic substances is concerned, although there may be some slight reduction in the absorption of the fats. In consequence of altered intestinal conditions, an increased secretion of alkalies into the bowel occurs, partly in combination with the abnormal quantities of higher and lower fatty acids and partly by increased secretory activity. The balance of the earthy alkalies may even become negative. This, however, is not so important in the matter of gain in weight as the coincident reduction of the sodium and potassium retention. The reduction of the available quantity of these mineral substances, so important for growth, is probably the cause of the decreased development. The irregularities of absorption and retention, due to the variation of alkali and water content, are the basis of the variation in weight. The beneficial action of increased carbohydrate is explained by the marked affect upon the alkali reserve and the water retention.

The fat-soap stools are distinguished from the normal by the difference in the partition of the fecal fat. They contain much more of the earthy-alkali soaps and much less of the free fatty acids. Their light color is caused by an extensive reduction of the bilirubin into colorless urobilinogen. A strong alkalin reaction in the large intestine is necessary for the formation of such stools.

Soap stools were at first looked upon as pathologic, *i.e.*, as evidence of the abnormally great excretion of bases into the bowel. It is certain now that these materials occur regularly in the feces under perfectly normal digestion if the alkalin reaction is sufficiently strong; so that the diagnosis of nutritional disturbance cannot be made from the soap stools alone. Some feeding cases are seen which fail to thrive because their caloric requirement of about 100 calories per kilo is largely supplied by milk. The history and a careful examination reveals no definite pathology and there is no diarrhœa. Such cases may be included under the group of dystrophy due to excessive milk feeding. In the differential diagnosis the group of dystrophies due to inanition and also the failure of growth due to constitutional anomalies must be considered. The latter is, of course, still a purely hypothetical condition and its action in delaying growth is far from being understood. Such an endogenous disturbance of growth resists all therapeutic intervention. Delayed development also occurs in cases where there is sufficient carbohydrate in the mixture. This is probably an early stage of dyspepsia in which the characteristic changes in the stool have not taken place.

Prognosis.—The prognosis is favorably influenced by proper regulation of the food mixture.

Treatment.—The treatment consists in a rational regulation of the quantity of food and the number of meals, in a reduction of milk and the addition of suitable carbohydrates to the diet. In disturbances of recent origin, the addition of gruel or flour, with a slight reduction in the quantity of milk, frequently produces immediate benefit. The following list of carbohydrate foods is suggested in order of value. Dextrin-maltose preparations, (malt soup, etc.); prepared or dextrinized flours; toasted breads, simple

flours. Sugar of milk is to be excluded and cane-sugar should be used only in combination with gruel or flour. The quantity of the carbohydrate should be five per cent. of the total amount of food given, although frequently one needs more. The sum total of the nourishment should, at first, be small; when the tolerance of the child has been ascertained, it may be increased until finally the child itself determines the quantity which will it take. Few and, at most, not over five feedings in twenty-four hours are to be given.

If the carbohydrate used does not give satisfactory results, one of higher value is to be substituted. In cases of long standing, it is well to give the most efficacious form from the beginning. Liebig's malt soup may be used, according to the formula of A. Keller, or buttermilk mixture.

Malt soup, with its nutritive value of about 700 calories per litre, is sufficient for children to the weight of 5 kilos, or 11 pounds, and to the age of seven or eight months. In older and heavier children, it should be mixed with more milk, less water and less flour; otherwise the mixture becomes too thick. Children under three months of age should be given only two-thirds of the original solution of carbohydrate.

Malt soup is prepared as follows: Stir 50 grams (2 ounces), of white flour into one-third of a litre (11 ounces) of milk, warming it gradually. In another dish dissolve 100 grams (3½ ounces) of malt soup extract (malt extract neutralized with potassium carbonate), in two-thirds of a litre (22 ounces) of water. Mix this with the milk and flour; boil and strain through a fine sieve.

Malt soup produces somewhat thin and frequent stools and is therefore to be recommended when constipation follows the use of other food.

Buttermilk intended for use in infant feeding should be produced in as cleanly a manner as possible. Its acidity should be about 7 c.c. normal NaOH solution.

Heat one quart of buttermilk. Mix five grams (⅕ ounce), or two level tablespoonfuls of flour to a smooth paste with a few tablespoonfuls of the buttermilk. Stir the paste into the remainder of the hot buttermilk. Continue stirring until it boils, withdraw from the fire a moment, and then reboil without stirring. Add 50 to 70 grams (1¾-2½ ounces) of granulated sugar, dissolving it thoroughly and bring the mixture to a boil the third time.

Changes in the relative quantities may be made to meet the tendency to either constipation or diarrhœa. The dextrin-maltose mixture may be substituted. Benzosulphinidum (saccharin), may be used if the sugar given does not make the mixture sweet enough. Beginning with two to three per cent., the quantity may be increased gradually.

When the child is given this therapeutic diet, an increase in weight should immediately follow and continue progressively; a noticeable improvement in the general well-being should be observed (Fig. 74); and the stools should be of good quality. If improvement is not seen and marked diarrhœa does not suggest the transition to the type of dyspepsia, it is necessary to make greater changes in the carbohydrate components of the food (see next division). Malt soup or buttermilk having been given

for six or eight weeks, an attempt should be made to return to an ordinary diet and to ascertain thereby whether a longer period of the therapeutic diet is necessary. The recovery may be considered complete when continued development is reëstablished upon ordinary milk mixtures.

The therapeutic dietaries above are better adapted to older infants. In new-born infants and those of four to six weeks, great care must be exercised in making changes. Gradually increasing amounts of gruels and sugars, preferably dextrin and maltose mixtures and granulated sugar up to seven per cent., or small amounts of buttermilk mixture with five per cent. sugar, may be cautiously employed. If immediate improvement is not noted,

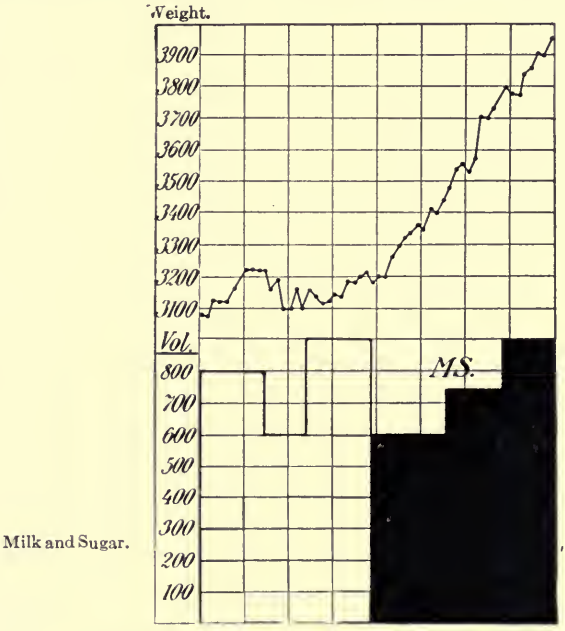


FIG. 74.—Typical curve of successful treatment of dystrophy with diet poor in fat and high in carbohydrate (MS. = Malt Soup).

there is danger of transition to dyspepsia and the treatment for this condition is advisable from the onset.

2. Dystrophy as a result of excessive flour feeding. (Flour-feeding injury of Czerny and Keller.)

Pap or flour paste feeding, with or without small additions of milk or other food material, is a method of infant feeding still encountered occasionally. Flour-feeding injuries may also be caused when a flour diet, ordered by the physician or selected by the mother herself to cure a diarrhœa, is continued for too long a time. The younger the child, the more rapidly does injury occur when flour is the chief constituent of the diet.

Symptoms.—In not a few cases, disease symptoms fail to appear for some time in spite of the improper diet. A normal development may be

simulated for a time for the great water-retaining power of the carbohydrate causes marked increase in weight. The general appearance of the child is good and the fat deposits abundant. But even at this time, certain abnormalities may be observed. The musculature may be slightly hypertonic, the complexion poor; a nervous irritability (latent tetany) may be demonstrated upon close examination. Later, the development of extreme symptoms is threatened. A severe flour-feeding injury develops which may take on varyingly graded forms, according to the exclusive or non-exclusive use of this article of diet.

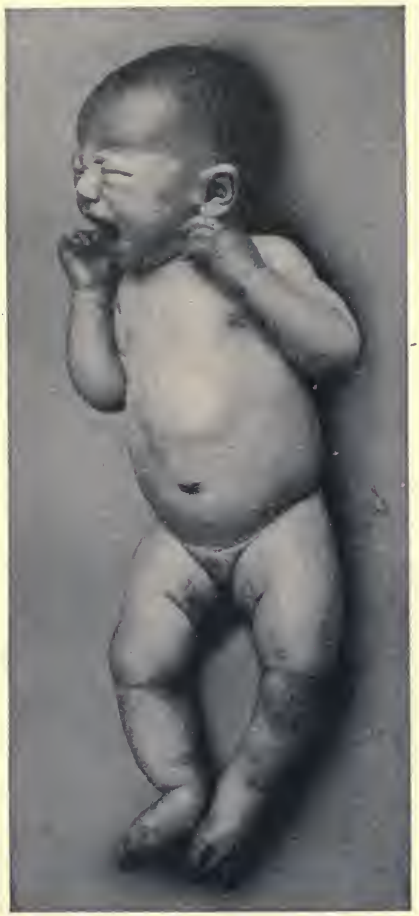


Fig. 75.—Alimentary edema. (University Children's Hospital, Breslau, Prof. Tobler.)

The atrophic type appears when the feeding has consisted of flour alone, without the addition of salt. It can hardly be differentiated from simple severe starvation. The only distinctive features are the hypertonicity of the muscles and the drying out of the tissues, which are particularly noticeable. A brownish-red coloring of the skin should attract especial attention.

In the hydremic form, which occurs in flour feeding with large additions of salt, the weight is increased markedly, in consequence of the retention of large quantities of water in the tissues. This may be recognized clinically by a pale, bloated appearance of the face, the spongy consistency of the skin and, finally, by the appearance of edema without evidences of injury to the kidneys (Fig. 75).

A hypertonic form is also described in which the chief symptom is the rigidity of the musculature. This hypertonicity may at times take so severe a form that not only the limbs but the entire body becomes stiff. This condition, however, is not confined to flour-feeding injuries alone, but may occur in other disturbances of nutrition.

Stools are formed or soft, are of brown or yellow color, alkalin or acid, according to the flour used and to the tendency to constipation or diarrhœa. If the digestion of the flour is impaired, they become pasty, slimy and foamy, due to the formation of gas, have a foul, acid odor, and contain

many undigested particles which will stain with iodin. Irritation of the large intestines, with symptoms of colitis, may result from the fermentation of the flour.

A characteristic peculiarity of flour-feeding injury is the great and sudden variation of the weight-curve which occurs spontaneously, or when the child is attacked by an additional nutritive disturbance, or by an infection. Then a fall of several hundred grams, or even a kilogram, may occur within a few days, accompanied by correspondingly severe general symptoms. Such events are the more frequent, because such children show a definite reduction of immunity which affords an especially responsive soil for infective organisms which produce bacterial injuries of the most variable sort; such as, pyodermia, inflammatory lung affections, and pyelitis. Of the non-bacterial complications, corneal and conjunctival xerosis may be mentioned, while manifest tetany (spasmophilia) is not uncommon.

Metabolism and Pathogenesis.—The disturbance of the organism occurring in exclusive flour feeding is to be considered a qualitative inanition, produced by the absence of one or more of the necessary tissue-building foods (fat, and in part, also, protein and salts); on account of which the formation of normal body tissue, if not the development in general, is impossible. Usually the caloric value of the food is insufficient, so that quantitative inanition also plays some part. The enormous quantity of water retained, because of the large salt content of flour food, stands in direct relation to the variations of weight. The reduction of immunity may be due rather to the scant formation of antibodies, as a consequence of inanition, than to the high water content of the body.

Prognosis.—The prognosis depends upon the age of the child and the duration of the period of incorrect feeding. The younger the child and the longer the continuance of flour feeding, the less hopeful is the final result. The great mortality is caused rather by unavoidable intercurrent infections, than by the feeding injury itself. The tetany and the spasmophilic convulsions resulting from it are very dangerous manifestations.

Prophylaxis.—The incidence of a primary flour-feeding injury is avoided by an appropriate diet. It is important to consider, the possibility of the occurrence of such an injury when flour feeding is resorted to for therapeutic purposes, whether for the purpose of alleviating symptoms of spasmophilia or for the treatment of dyspeptic diarrhœa. The danger may be avoided in the first instance by the addition of casein preparations, fat and small amounts of salts, and in the second case by continuing the pure flour diet for only a very few days; and overcoming the difficulties of a return to a milk diet by the methods to be described below.

Treatment.—In young infants, and especially in the severer class of cases, the most rapid recovery is to be expected from breast-milk feeding. Remembering that inanition may have caused a lowering of the limits of tolerance, it is well to begin, as in decomposition, with small quantities 200-300 c.c. (7-10 ounces *per diem*); and then to increase the amount as carefully but as rapidly as possible. Whole milk, slightly diluted, or milk with moderate carbohydrate additions (3-5 per cent.), are the artificial

mixtures to be recommended. With these infants it is well to begin as though decomposition were present. Protein-milk has given very good and certain results.

In all these cases—in so far as we do not have to deal with the purely atrophic type, a decided loss of weight is to be expected as a result of the feeding of mixtures poor in carbohydrates, because of the rapid loss of the water which has been retained during the flour feeding. Occasionally this may produce a serious condition and it is advantageous therefore to retard the loss by the addition of small amounts of flour or of dextrin and maltose preparations to the milk mixture.

If the change from flour to milk mixtures is made gradually by adding small quantities of milk to the gruel, as is customary when the period of flour-soup feeding for the relief of diarrhœa is to be ended, the diarrhœa often recurs; which leads to the further discontinuance of milk in the mixture, and to the conclusion that milk is not well borne by the child. Frequently this trouble is due to the fact that flour ferments more readily in the presence of milk; as, for example, with sugar, a strong solution of which may be taken care of in dyspepsia, while the same amount, added to milk, produces symptoms of severe intestinal irritation. In these cases, the desired results are often obtained if the change is made suddenly and the flour omitted at once. In this event, again, the best results are obtained by feeding protein-milk.

3. Dystrophy as a result of inanition.

In addition to the forms of dystrophy considered above, must be added the innumerable cases in which the failure of proper development is due solely to quantitative insufficiency of food. A common cause of this is a food of too great dilution, the caloric value is so reduced that the child cannot take enough to supply its requirement. Frequent vomiting and regurgitation is a cause. Inanition also plays an important rôle in those older children who have been much delayed in weight but are still being fed on the caloric requirement of that weight instead of the requirement of a normal child of that age. They have a much greater requirement than younger infants of the same age and will not thrive on 100 to 150 calories per kilo. At times, children are seen who, because of anorexia, do not take sufficient food of their own accord to cover their needs.

Diagnosis.—The diagnosis is made by an investigation of the actual amounts of food taken and the determination of the caloric value of the same, in relation to the age and weight.

Treatment.—In the milder cases, gain in weight and improvement in the general development may readily be obtained by changing to a more concentrated food or, if necessary, by giving seven or more feedings instead of the customary five. If the child is markedly underweight and does not take its food well, highly concentrated food must be given. In such a case, whole milk with twelve to seventen per cent. of carbohydrate, which nearly doubles its food value; concentrated protein-milk with fifteen to twenty per cent. carbohydrate; Sauer's farina mixture; and other thick gruels are valuable. Fat may be added to the food in the form of butter or cream.

These are the cases in which the "butter-flour mixture" of Czerny and Kleinschmidt is especially indicated (see feeding of normal infants in General Part).

B. Nutritional Disturbances Without Toxic Manifestations With Diarrhœa.

4. Dystrophy with dyspepsia (chronic dyspepsia).

Etiology.—In many cases, the cause of the failure of development, the dystrophic condition, is found in abnormal digestive processes. This may occur without producing extreme gastro-intestinal symptoms, both in primarily healthy children and in conjunction with acute dyspepsia or with flour or milk-feeding dystrophy. The condition is encountered when there is a distinct intestinal digestive insufficiency for the food offered. It may be the result of absolute or relative inanition; or of primary reduction of the tolerance, especially by infection. This insufficiency may become so great that pathological fermentation takes place and brings on the symptom-complex to be described. The products of fermentation cause increased peristalsis and resulting diarrhœa.

Symptoms.—The symptoms of disturbed gastro-intestinal function are the chief indication of the dyspeptic condition. The appetite may be diminished; regurgitation and vomiting may occur. The motor power of the stomach is reduced. The analysis of the stomach content usually shows the absence of free HCl. But the free and volatile fatty acids are increased, as can be recognized from the characteristic odor. Frequently the abdomen is distended. Increased peristalsis may be visible or may be determined by auscultation. There is a tendency to flatulence and colic, with a resultant restlessness of the child. The stools are more numerous and definitely abnormal. They become thinner, watery or lumpy and contain mucus. Their odor is abnormal, indicating either putrefaction or fermentation. Their chemical reaction is variable, usually acid. Their color is often green, in consequence of the oxidation of bilirubin to biliverdin.

The increased peristalsis interferes more or less with the absorption of the products of digestion, a fact which may be determined not only by metabolism experiments, but also by macroscopic, microscopic and chemical examination of the feces.

The stools contain fat soaps, seen as small white or yellowish lumps ("milk curds"), from which fatty acid crystals may be obtained by heating with strong mineral acids. Neutral fats in the form of fine granules or coarser globules, and fatty acids in the form of needles, clusters or droplets are also present. Usually present in small amounts only, both occur occasionally in enormous quantities. Microscopically, fatty stools are soapy or shiny in appearance, semisolid or fluid in form, yellow or green in color, and of markedly acid reaction. In the stained preparation, a large number of Gram positive bacilli are noticeable, resembling in this respect, the stools of the breast-fed infant.

A good conception of the distribution of the fats may be obtained by staining a smear with dilute carbol-fuchsin. Neutral fat remains unstained, soap-fat appears bright pink and fatty acid bright red. The fat globules in

the stools vary in the shade of red, so that we may conclude that the neutral fat is always mixed with fatty acids (Fig. 76).

Flour stools are pasty or foamy. With iodin, the undigested starches stain blue and the erythrodextrins red. Frequently, large numbers of idiophilic bacteria may be found.

For a long time, the question whether undigested casein appears in the stools has been of interest. The whitish-yellow flakes, or so-called "milk curds," have been erroneously considered as particles of casein which had escaped absorption because of their indigestibility. It is now an established fact that these alleged casein flakes consist almost entirely of the salts of fatty acids and of bacteria. Only under raw-milk feeding do large, tough, rubber-like casein curds pass undigested through the intestine. Their appearance does not permit us any conclusion as to their pathogenetic importance.

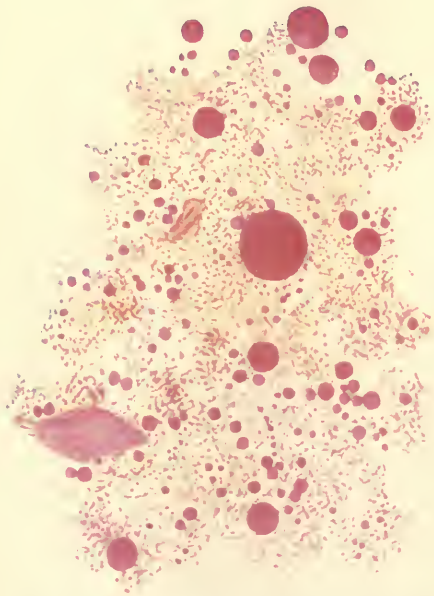


FIG. 76.—Microscopic preparation of stool in case of fat diarrhœa. Stained with carbol-fuchsin. Fatty acids red, soap fats pink.

The general symptoms—when sharply differentiated from other severe forms of disturbance of nutrition, *e. g.*, intoxication, decomposition, with acute incidence—are not of great variety. The patient is pale and restless, his sleep light and his disposition altered. The tone of the tissues is reduced. While the body-weight may increase in the milder cases, there is usually no increase or a slight loss of weight. The body-temperature is important; the daily curve varying between a slightly subnormal point and slight fever, (see alimentary fever).

Pathogenesis.—(See also general pathogenesis.) The local symptoms of dyspepsia are usually caused by the increased formation of the acids of fermentation.[2] In all probability, the pathologic decomposition of the carbohydrates (sugar, flour), must be considered the primary cause of the fermentation; while the decomposition of the fat, when given in ordinarily small amounts, is secondary to it. No satisfactory proofs of injury to the intestinal tract by the decomposed digestive products of casein, of which so much has been written, have been brought forward. Rather, has it been shown that the harmful decomposition of carbohydrates may be combated by large

[2] Cases are occasionally seen in which there is a poor gain in weight with increased slimy stools, and which are easily cured by the addition of a more readily assimilated carbohydrate. Evidently, there is an actual carbohydrate hunger in this condition. The chemistry is not understood.

quantities of casein which acts beneficially as a therapeutic agent. It is true that when large amounts of casein are fed, there is a possibility of the protein entering into the circulation with severe general symptoms, as frequently happens in premature and very weak infants. The main support of the view that the primary cause of dyspepsia is the carbohydrate, lies in the fact that it is nearly always possible to reduce the abnormal fermentation and the excessive peristalsis by sharp reduction of the sugars, eliminating, in certain cases, even the lactose of the milk itself. The different sugars show important differences in their susceptibility to fermentation. Sugar of milk is most readily fermented; cane-sugar less readily; while the dextrin and maltose preparations of commerce, dextri-maltose, etc., are the least liable to fermentation.

The results of clinical experiment indicate that the intestinal tolerance for carbohydrate and the greater or less tendency to pathologic fermentation are not constant, but are dependent, as is the tolerance for fat, upon the liquid in which the carbohydrate, in solution or suspension, is given. A given quantity of sugar in undiluted whey gives rise to symptoms of dyspepsia more readily than when added to water or diluted whey. Thus an increase of the protein content may counteract the tendency to fermentation, while an increase of the whey with reduced protein favors it.

The pathogenesis of the general symptoms of dyspepsia is akin to that of the symptoms common to the several grades of disturbance of nutrition.

A reduction of absorptive power, which may be demonstrated clinically, has been proved by metabolism experiments. Not only the organic, but the inorganic food-stuffs are involved in this reduction. However, in dyspepsia, in contrast to decomposition, a lessening of the mineral bases of the body is rare, because increased retention of the inorganic salts compensates for the diminished intake. So rapid a deterioration and so greatly reduced tone of the tissues is not seen, therefore, in dyspepsia.

Variations in the Course.—It is essential in every case to attempt to arrive at some conclusion as to the etiologic characteristics. This is especially true in those cases in which the dyspepsia is more or less directly the result of infection. In young infants, enteral and parenteral infections are very commonly seen in combination with secondary disturbance of nutrition and conversely a nutritional disturbance may often have a secondary infection added to it. There can be no doubt of the presence of an infective process when rises in temperature persist in spite of the correction of the food as required for the dyspepsia. Further infection must be suspected when, in spite of a diet practically free from sugar and definitely non-fermentation producing, the frequent stools still contain large amounts of mucous. Such a condition is due to a mild secondary enterocolitis. The pedantic differentiation between infection and dyspepsia is in itself of minor importance, provided that the treatment is not influenced to the extent of deviating from that to be indicated for dyspepsia in the following. There is danger, however, that the persisting intestinal irritation kept up by the local or hematogenous infection be interpreted as a continuance of the fermentative process that initiated the disorder. In a similar manner, the dyspeptic

symptoms that appear with hunger and inanition may lead the treatment astray. For this reason, the possibility of hunger dyspepsia should constantly be kept in mind. This especially in the new-born.

Diagnosis.—Diagnostically it is important to determine whether the diarrhœa is merely a manifestation added to simple failure of development (dystrophy), or whether it is a far-reaching nutritional disturbance with actual loss of body substance (decomposition). These two conditions differ not only in their prognosis, but require entirely different treatment. The differentiation is made from the clinical examination and from the history. Emaciation, sudden losses of weight and subnormal temperature indicate decomposition. A history of repeated diarrhœa with periods of loss of weight, and recurring injuries reducing the tolerance also indicates a reduction of the functional ability of the organism in the sense of a decomposition.

Prognosis.—The outcome of a persisting dyspepsia may still be favorable, as long as there is no loss of weight and the general condition of the child is not markedly affected. In recurring dyspepsia, the prognosis must be more guarded because of the great reduction of the tolerance. This is also true in cases of diarrhœa in infants during the first few days of life. On account of the very low tolerance at this age and the readiness with which the condition may go on to decomposition the prognosis is always grave.

Dietetic Treatment.—The safest food for cases of the dyspeptic form of dystrophy is breast-milk. Especially during the first few weeks of life, every effort must be made to give the infant the natural food, for at so early an age the results of artificial food are always doubtful. The full caloric requirement may be supplied in breast-milk but even with this food it is well to underfeed slightly at first.

In using artificial food in the treatment of dyspepsia, the cure must be affected by changes in the composition of the mixture and not by reducing the quantity. The diarrhœa may be stopped temporarily by cutting down the diet that caused it, to a starvation ration, but the intestinal fermentation flares up again as soon as the food is brought up to the requirement. The course to be chosen, therefore, is rather to change to a mixture containing less fermentable carbohydrate. The reduction of the abnormal fermentation is most rapidly accomplished (1) by the reduction of a possible excess of carbohydrate; (2) by replacing the more easily fermented sugar of milk or cane-sugar with the readily assimilated dextrinized flours or maltose and dextrin mixtures; (3) by the addition of calcium caseinate (protolac, larosan) up to two per cent.

In the milder cases, these additions may be made to mixtures of gruels and lean milk (Fig. 77). When the symptoms have persisted for a time, it is well to eliminate even the lactose of the milk by substituting buttermilk. Some authors ascribe a beneficial effect to the acid of the buttermilk.

The results of the treatment are evidenced by the reduction of the number of the stools and the improvement of their consistency. With the slowing up of the peristalsis, the weight as a rule begins to increase. The

temperature, which during the illness is extremely variable, returns to the typical monothermia. Nevertheless, it usually takes weeks before the general condition of the infant returns to the normal well-being.

With quite a few children, however, this method fails. While the loss of weight may be favorably influenced, the stools do not decrease in number or improve in consistency; or the loss of weight is continued and the diarrhœa persists. In these cases, we have probably to deal with a secondary infection or with a severe nutritional disturbance of a type transitional to decomposition. Nothing can be more mistaken or more disastrous than to continue underfeeding with the idea that the intestine will yet recuperate with continued rest. Children are killed in this way. Only a quick recourse to the treatment indicated in infective nutritional disturbances and in decomposition will avail. Therefore we would urge that while the schematic rules for the treatment of dyspepsia be first followed, under no circumstances should the underfeeding be continued in case of failure, but that the directions given for the treatment of decomposition be instituted at once.

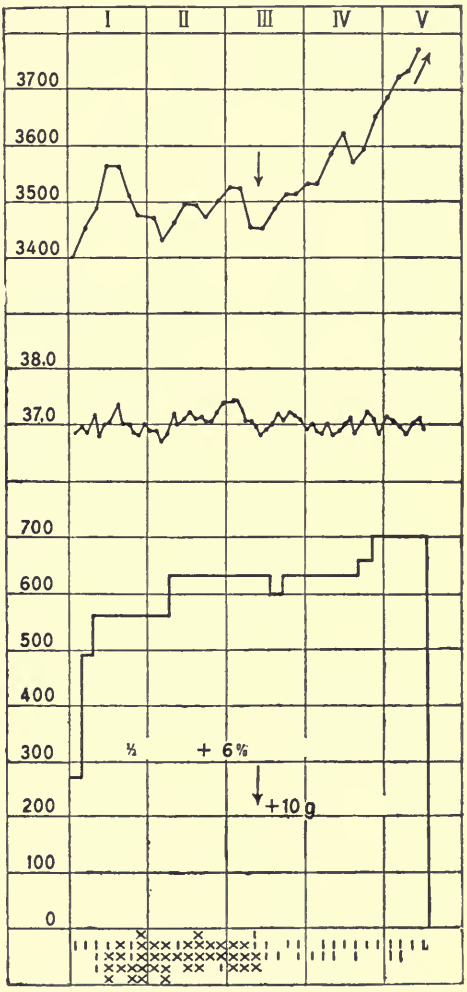


FIG. 77.—Dystrophy with dyspepsia in a young infant. Recovery after the addition of casein to the food.

One of the most successful methods of treating dyspepsia, in artificially-fed children, has been the use of protein-milk. While this is not necessary in many cases, the mixture protects those patients who do not respond to the ordinary therapy from the delay of improvement and the consequent serious loss of strength.

In the treatment of chronic forms of dyspepsia, there is no indication for underfeeding. Since we do not have to deal with a temporary injury, but with a chronic reduction of the limits of tolerance, the added trauma of hunger can only do harm. The feeding should be rational; the carbohydrates should be reduced to the absolute minimum (from 2-3 per cent.)

at once, and those forms more difficult of assimilation should be replaced by the more readily assimilated dextrin and maltose preparations. If this does not suffice to improve the condition of the stools, better results may be obtained with breast-milk or protein-milk. If these cannot be obtained, nothing remains but to continue the use of carefully measured quantities of the above food in order to keep the child alive in the hope that increasing age will produce an increase of tolerance and resultant recovery.

Medication is usually unnecessary. Astringents (tannigen, tannalbin, tannocol, etc., 5 to 10 grains four to five times daily; or bismuth salicylate, five grains four times daily), may be of use in long continued irritative cases with mucoid diarrhœa.

II. DECOMPOSITION [3]

(Pedatrophy)

The various degrees of nutritional disturbances discussed above, while serious do not have actual injurious effect upon the body structure. In the next grade, decomposition, however, there is such an effect with a true loss of body constituents. As a result of an extensive lesion of the intestine, there is a gradually increasing disturbance of the metabolism. This leads to destruction of body substance with grave pathologic loss of body fluids and body substance.

Symptoms.—Dystrophy is in the stage of transition into decomposition when marked losses of weight occur. These losses are, at first, gradual but later, and in severe cases, are sudden and large. As the case progresses, we may have extreme emaciation which causes the "old man" appearance and later the skeleton like body of the "atrophic infant" (Figs. 78 and 79). The abdomen is usually distended, even tense; the musculature flabby or hypertonic. The patient may be pale at first, but later has a characteristic pasty gray color, while the mucous membranes of the seemingly huge mouth are a deep red.

The urine is free from protein and sugar. The stools are usually dyspeptic, frequently diarrhœic; liquid and solid evacuations may alternate. At times, during periods of remission of the fundamental disease process, only formed stools are passed. In a peculiar condition known as fatty diarrhœa, the discharges are characterized by excess of fat. Often the stools are tarry or reddish black, due to bleeding from peptic duodenal ulcers (Fig. 80).

At first, the patients are irritable, cry a great deal and eat ravenously. Later, they become dull and lethargic. There is a noticeable tendency to slowing and irregularities of the pulse. The temperature may be subnormal

[3] "Decomponere" means to "separate into its constituents" as well as to "change the composition," (in an unfavorable sense). Both definitions apply to the condition in question. Not only do the tissues of the body break down, while their separate cell constituents are excreted in various ways, but the remaining cell contents are changed in composition, as shown in their seriously disordered functions. Although the term decomposition, and particularly alimentary decomposition, since it is traceable to the influence of feeding, has been criticised, the nature of the condition is better so described than by the ambiguous word "atrophy."

and is liable to great variation in distinction to the normal monothermia. Edema and cyanosis are common symptoms.

Aside from the emaciation and its accompanying phenomena, decomposition is characterized, symptomatically, by the sensitiveness of the patient, or rather by the sensitivity and severity of his paradoxical reactions to nutritive, infective, and other influences. A slight alteration in the quantity and quality of the nourishment may immediately cause a very threatening aggravation: a minor bacterial infection, a coryza, a bronchitis, etc., may lead to serious decline. A little overheating may produce high fever and collapse. By this very sensitiveness the diagnosis of the condition may be made when the child is in a remission, or is beginning to recover and even when the symptoms of the body loss are not apparent. The reduced immunity predisposes to infective complications (furunculosis and other pyo-

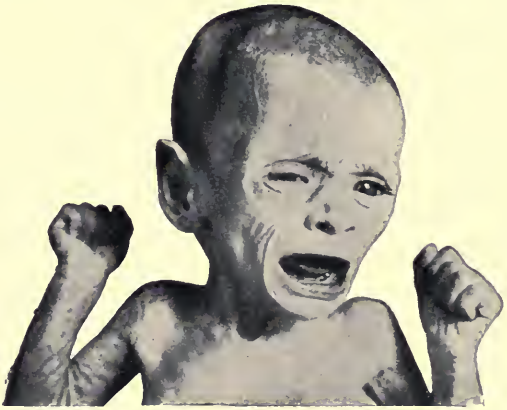


Fig. 78.—Facies in moderate decomposition. (Berlin Children's Asylum, Dr. Dessauer.)

dermatites, pyelitis, septic diseases, bronchitis, pneumonia, etc.), which occur frequently and run an exceptionally severe course.

A peculiar form of interruption of the course is often seen in decomposition and also at times in dyspepsia. To this the name of "Reversion" has been applied. The patient seems to be doing especially well for several days. There is a constant gain in weight up to several hundred grams and the attendant is greatly encouraged. This is not a true manifestation of growth, but is largely due to a retention of water. The slightest error in the treatment or the mildest external injury throws the patient back and the gain is lost in a few days. The patient has not gained anything by the period of improvement but has actually been weakened.

Pathogenesis and Metabolism.—Formerly it was believed that inanition was the cause of severe "atrophy" and was due to interference with food absorption in consequence of a chronic inflammation and destruction of the secretory mechanism. The foundations of this teaching are today, however, overthrown; for the concurrent reports of all observers show that the intestine of the atrophic child is anatomically normal. It is clearly a

question of a functional disturbance, leading to a reversive metabolism, recognized by Parrot many decades ago. In fact, clinical observations prove that we have to deal with a paradoxical reaction of the food material; the more food we give the more the patient loses, while in the milder cases an arrest of the disease may be secured by reducing the allowance of food. In severe cases, of course, even this measure will not stop the wasting. From the rapid loss of weight, it will be seen that the pathologic loss of water and salts is primary, because this alone can produce such sudden changes in weight, while other tissue losses must be more gradual.

Fig. 79.—Extreme form of decomposition.

The experiments in metabolism have cast some light upon this problem. It may be readily understood that the continual enteral fermentation gradually produces so high a degree of alteration in the processes of digestion that severe damage is done to the agencies of interchange between the intestinal content and the tissues. A radical alteration in the water and salt retaining function of the cells probably ensues. This results in the increased secretion of water and alkali into the intestine, which is not balanced by a compensatory reduction of the renal output as it is in the healthy or the only slightly-ill child, in whom a negative balance often exists. Doubtless abnormally increased quantities of water are given off by the lungs. As a result of the loss of salts by the bowel, a greatly increased excretion of ammonia in the urine follows, a condition of relative acidosis. The alkalies are lost, in part by increased secretion, and are, in part, consumed in the necessary neutralization of the great quantity of acids formed by fermentation. To cover these losses, the storage depots of the body are drawn upon. When these are exhausted, those stored in the constant constituents of the organism must provide the necessary quantity of water and salts by the decomposition of the cell substances. In addition to this, an actual inanition also ensues. The carbohydrates and fats are fermented in large quantities; the acids of fermentation prevent the normal splitting of the sugar preparatory to absorption; and as a result of the violent peristalsis large quantities of food material pass through the intestine entirely unchanged. To this is often added the semi-starvation of underfeeding

usually adopted for therapeutic purposes. The "decomposition" of the more important organs finally leads to so great an alteration of their cell conditions and, in consequence, of their functional activities, that not only because of the continued diarrhœa, but even after this has disappeared, the normal internal metabolism can no longer be maintained and an auto-intoxication results which produces the terminal symptoms of the disease.

Cases of decomposition of purely alimentary origin are probably found in new-born and very young infants only. In older children the infectious influence takes a very prominent place in the etiology. To this must be added the frequent starvation periods employed in the attempt to stop the diarrhœa, which undermine the strength of the patient more and more. Such starvation cause recurrences even in cases on the road to recovery and are often to be blamed for the fatal termination. In fact, numerous factors are combined in the pathogenesis of decomposition.

Course.—In young infants the course is often uninterrupted, the fatal outcome occurring in a few days or weeks, at the most. In older children,

Fig. 80.—Duodenal ulcer in decomposition. Fatal hemorrhage occurred from this lesion (Berlin Children's Asylum).

periods of improvement and periods of decline alternate. Serious catastrophies may occur anywhere along the course. In this manner, we may have an illness of weeks' duration, with exacerbations and ameliorations. The intestinal processes determine the occurrence of such remissions. As long as the stools are infrequent and formed, the weight remains stationary or there may even be slight gains. As soon as diarrhœa again appears, there is actual loss of body substance. A sudden change in the disease-picture is often brought about by hemorrhage from the peptic duodenal ulcers, which are generally fatal. The etiology of these ulcers is not known.

The termination of unfavorable cases is attended by a variety of symptoms. In many children, a narcosis develops with an absence of reflexes, general torpidity—and subnormal temperature continuing for a number of days. Others die with the appearance of symptoms which, with or without fever, resemble those of alimentary intoxication. Sudden death in collapse, in consequence of some exciting influence or after several hours of hunger, may occur. Complicating infections tend materially to increase the death-rate.

The prognosis depends to a very great extent upon the dietetic treatment. If errors are avoided and the treatment is instituted early enough, even the extreme cases may be saved. If, however, the loss of weight exceeds one-third of the original body-weight (the so-called Quest's quotient), recovery is apparently impossible.

Diagnosis.—The diagnosis of severe cases is clearly given by the clinical picture. The mistake of confusing the symptomatic emaciation of tuberculosis and other diseases leading to cachexia, or the weight-loss of severe starvation, must be avoided by careful examination and a study of the history. In milder forms, the differentiation from dystrophy and simple dyspepsia, without destructive losses, is of so much greater importance since the treatment indicated for these conditions, *viz;* a diet rich in carbohydrates and a period of starvation, may cause serious injury in children suffering with decomposition. In such cases, the history is of the greatest importance. Repeated diarrhœa, loss of weight, in contrast to the stationary weight incident to non-development in dystrophy, or in infective fever of varying degree, are suggestive evidences of decomposition. A final conclusion must be reached by the reaction of the patient to the prescribed diet. If with a fairly plentiful diet, the paradoxical reaction (diarrhœa, loss of weight, occasional fever, etc.), occurs definitely and severely, the diagnosis of decomposition is certain.

Treatment.—For the cure of decomposition, it is necessary that the fermentation which causes a continuance of the destructive phenomena be abated, so that the organism may again retain water and salts, and the digestion may again take care of the organic food-stuffs, in a normal manner. Therefore, the same problem is present as in dyspepsia and one might be inclined to inaugurate the same treatment as it demands; that is, a period of starvation, followed by a gradually increasing use of suitable food mixtures. In fact, this procedure is frequently adopted. One must realize, however, that success cannot rightly be expected, save in a certain number, only, of older infants; and among these only in cases where the debility has not advanced too far. For the methods recommended in dystrophy are absolutely contraindicated by the principles of treatment in any severe form of decomposition. These principles are (1) that the child with decomposition must not be starved; (2) that the child should be put to the breast, for it is to be feared that artificial feeding, under prevailing methods, will rapidly and progressively aggravate the disease.

In children with severe disturbances of nutrition, the dangers of a starvation period are very much greater than in well infants or those with slight disturbances. Hunger causes much more rapid loss of weight, marked slowing of the pulse, subnormal temperature and collapse and occasionally death. Repeated starvation periods at short intervals are especially dangerous. The second or third such period is usually fatal (Fig. 81). The special emphasis laid upon this point is necessary because starvation treatment is still very much in vogue and is recommended in text-books. Aside from the above, it is further true that subjecting the patient suffering from

decomposition to hunger periods can only result in a further loss of strength. Persistent underfeeding makes the prognosis very grave even in cases in

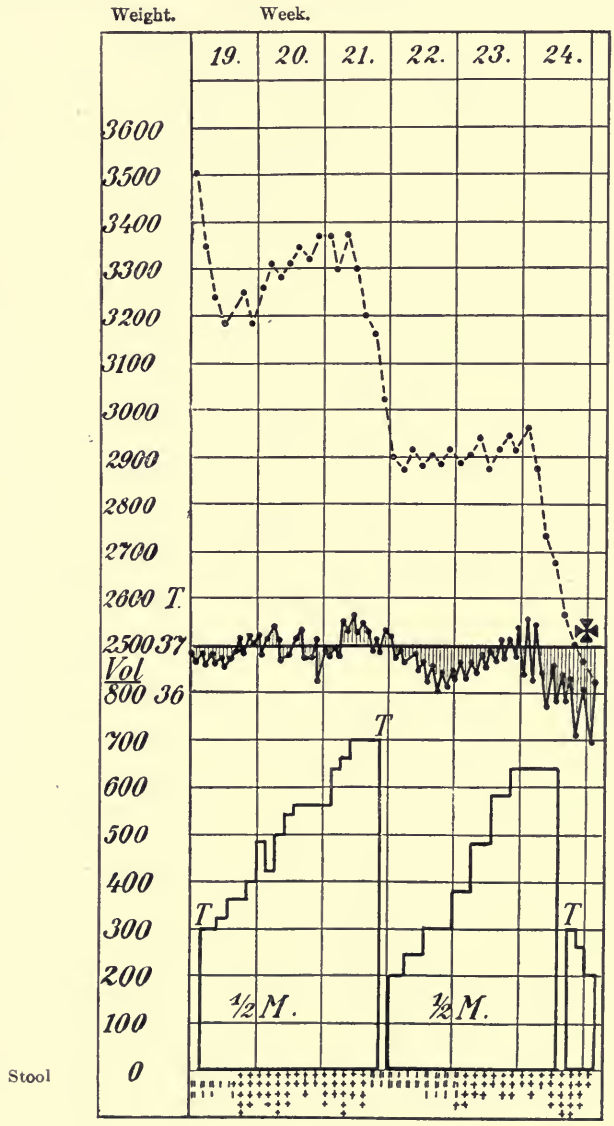


FIG. 81.—Showing the injury caused by repeated starvation periods in cases of disturbance of nutrition. After the first starvation period the weight is stationary for a time but there is no improvement of the intestinal condition. Soon the temperature rises again and further losses of weight occur. Death resulted on the third day of the second starvation period.

which there was some hope of recovery. It is essential that the caloric requirement be supplied from the very beginning of the treatment. In the large majority of the cases, this is not possible with the customary artificial

foods and, for this reason, breast-milk is to be preferred to all other forms of food.

In the use of breast-milk, certain points must be taken into consideration; and first, the matter of dosage. The curative influence of breast-milk, apparently due to the constituents of the whey, is not at first so active as to preclude the injurious effect due to the fermentation of its large amounts of sugar and fat. This danger is increased as more food is taken; yet if too little is given, the danger of renewed injury from inanition again confronts us. The best plan is to give a daily total of 200-300 c.c. (7-10 ounces) without any preceding starvation period. It is better to give the breast-milk from a bottle so as to relieve the weakened child of the labor of sucking. In addition to the food, large quantities of water sweetened with benzosulphinidum (saccharin) should be given. Eight to ten feedings may be offered in twenty-four hours, for experience has shown that the same quantity in small doses is less liable to cause injury than in large ones. In the shortest possible time, about every other day, the quantity should be increased until by the seventh to the tenth day 100 calories (130-150 c.c.), per kilo of body-weight are being given and in less frequent feedings. By this time, the child may be put directly to the breast.

It is further to be remembered that even with breast-milk the severer type of cases will, at first, show an aggravation of symptoms; the patient becomes more pale and more dull; a subnormal temperature and a slow pulse may appear; while the loss of weight continues. Only after several days, or even during the second week, does the weight-curve become stationary and the other symptoms begin to disappear (Fig. 82).

The danger of breast-milk feeding is greatly reduced and favorable outcome enhanced by adding 200 to 300 c.c. of buttermilk or fat-free milk without carbohydrate addition to the daily allowance of mother's milk at the outset. This prevents the continued loss of weight since the high whey content favors water retention. As the amount of breast-milk can be increased, the additional fat-free milk can be gradually reduced. It is better, however, not to stop it entirely for slight losses of weight may result.

Now follows a period of varying duration, sometimes continued for several weeks, during which the weight remains stationary, while the improvement in the general condition continues. The inexperienced advisor concludes, therefore, that the milk of the wet-nurse is not good and recommends a change. This is a mistake. We have to deal with a period of repair during which the body is rebuilding without being able to attain any appreciable increase, partly because of still inadequate absorptive power and partly because breast-milk, poor in protein and salts, affords so little material for cell-growth. Only after this period does an increase of weight occur. The period of repair may be shortened by giving suitable additional food; the best, probably, being buttermilk to which carefully regulated proportions of dextrin and maltose preparations or malt soup are added. This supplemental food should not be given before the fourth week and then at only one meal a day. The addition of powdered casein is often beneficial.

Complete recovery cannot be expected in less than two to three months. Not until this period of time has passed should a return to artificial food be considered. It is advisable to precede the discontinuance of breast-milk with small artificial feedings, for there may be an idiosyncrasy for cow's milk. If it is impossible to obtain breast-milk for the patient suffering from decomposition, the treatment to be followed should be that recommended for dyspepsia. Buttermilk, or milk poor in fat, diluted with gruels should receive first consideration. But while with breast-milk the intestine soon recovers to such an extent that it is possible to give adequate quantities of

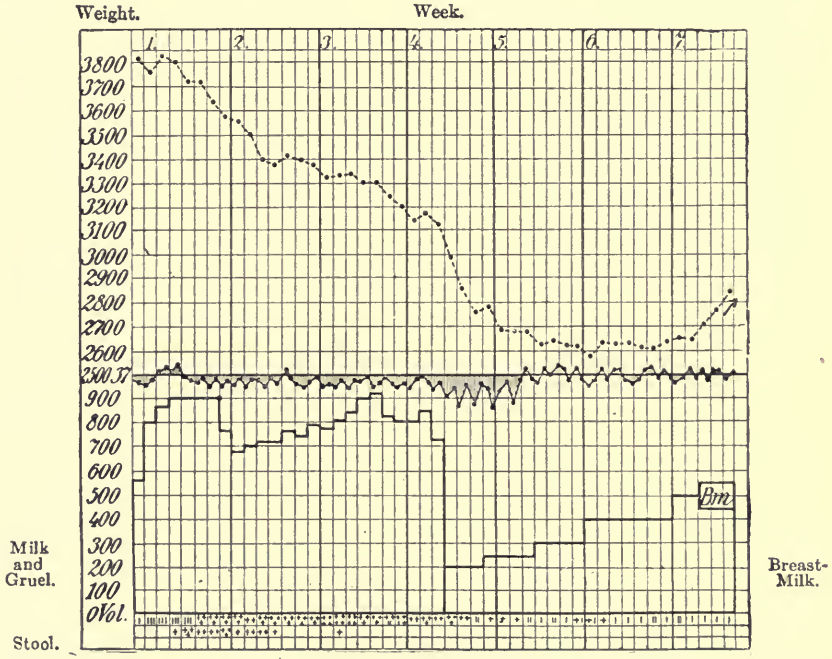


FIG. 82.—Recovery of a case of decomposition treated with breast-milk. Aggravation when breast-milk was first given shown by subnormal temperature and disturbances of weight. Then for two weeks no change in weight. Increase occurred after addition of buttermilk (Bm). + = pathologic stool; | = normal stools.

food, the slightest increase in artificial feeding may produce a recurrence of the diarrhœa or an entire arrest of improvement. No gain is to be hoped for from the continuation of the infant's former food, or from the feeding of gruel soups, which are hazardous because of the danger of inanition. The prospect of good results under artificial feeding especially with younger children, is slight. Recent experience, however, has shown that the possibility of recovery is considerably enhanced by the use of protein-milk or like preparations.

The object of protein-milk (Eiweiss Milch), is to avoid as fully as possible the occurrence of injurious acid fermentation. This is accomplished by the reduction of the milk-sugar content of the milk by a dilution of whey, which, in itself, improves the tolerance of the intestine for sugar, and

by the subsequent addition of large quantities of protein which tend to counteract the acid fermentation by the development of an alkalin reaction.

This preparation is made in the following manner: One litre (1 quart) of milk is warmed over a water-bath to 98° F. and curdled with one tablespoonful essence of pepsin. The whey, containing the sugar, is separated from the curd by straining through cheese cloth (one-half hour). The curd, mixed with one-half litre (1 pint), of water, is then rubbed through a fine wire milk strainer two or three times without excessive pressure; and to it one-half litre (1 pint), of good buttermilk is added. Finally the required amount of a malto-dextrin preparation (dextri-maltose), is added and the mixture is brought to the boiling point stirring constantly. The curd should not form lumps, the mixture resembling a very thin porridge when shaken.

The preparation is sterilized by boiling with constant energetic stirring, which is best done with a "Dover" egg-beater. If insufficiently stirred, the protein becomes tough and the mixture is useless.

Separate feedings must be warmed gradually and high temperatures should be avoided.[4] Benzosulphinidum (saccharin), may be added to sweeten.

Protein-milk contains 3 per cent. casein; 2.5 per cent. fat; 1.4 per cent. sugar of milk; and about 0.5 per cent. ash. Its food value is four hundred and fifty calories per litre.

A number of food mixtures based upon the principle of protein-milk have been devised. It has been our experience that some of these have the certain effect of the original protein-milk. Among others may be mentioned: larosan and protolac, casein calcium preparations. Twenty grams (2-3 ounces), of this are added to one litre (1 quart), of equal parts whole milk and water. Feer's protein-cream-milk is prepared by adding 50 grams of 20 per cent. cream, 10-50 grams dextri-maltose, and 15 grams calcium caseinate to 500 c.c. (1 pint), of whole milk and 600 c.c. (18 ounces) of water. The protein-milk itself may be most easily prepared according to the directions of Engel or those of Mueller-Kran. Engel accomplishes the fine division of casein which is so important, by using a special rennet tablet to curdle the milk after it has been boiled. Unfortunately, the coagulation is not always complete with these tablets; Mueller and Kran, therefore, recommend the following process: Mix one litre, (1 quart), of buttermilk with an equal quantity of water, boil and make up to two litres (2 quarts), with water. Let the mixture stand for thirty minutes until the casein settles to the bottom. Remove 1125 c.c. (36 ounces), of the fluid from the top by means of a dipper. To the remainder add 125 c.c. (3⅔ ounces) of 20 per cent. cream and make up to a litre again with water.

The advantage of protein-milk feeding lies in the fact that a return to the full amount of nourishment required may be made more rapidly than with

[4] Difficulty is frequently experienced in the preparation of protein-milk. In a poor preparation, the curative action is often lost, the child refuses it, or quite frequently vomiting results. For this reason, it has been prepared commercially and may be obtained in the powdered form.

any other food, without causing a recurrence of the fermentation processes. The danger of inanition is thus excluded and repair is hastened.

In early decomposition and in dyspepsia it is customary to begin, after twelve hours of starvation, by giving 300 c.c. (10 ounces), of food, with at least 3 per cent. or even 5 per cent. dextri-maltose, in five or six feedings. Milk-sugar is not advisable, nor is the cane-sugar likely to produce good results. The additional fluid required is given in the form of weak tea.

The quantity of protein-milk is increased in the succeeding days without regard to the condition of the stools, giving an additional 100 c.c. every second day, or even more frequently if the stools are formed, until a daily total of 180-200 c.c. per kilo is reached. The total feeding should not exceed

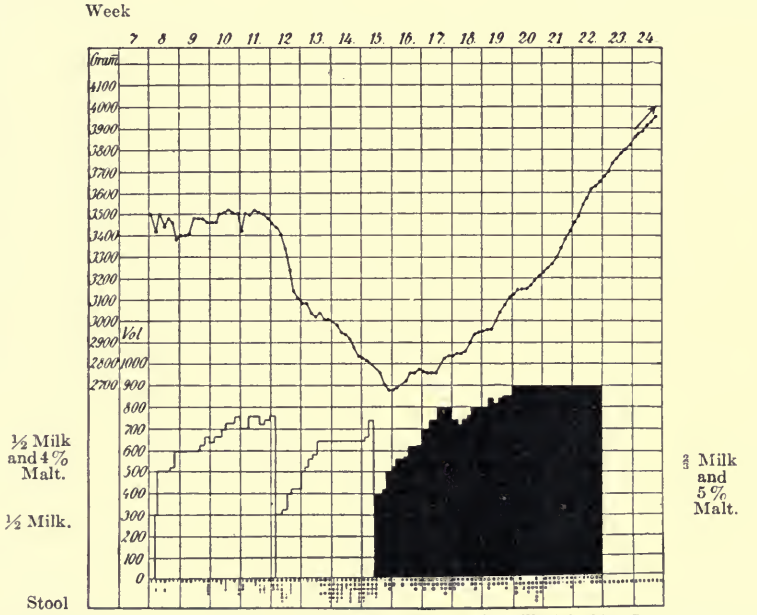


FIG. 83.—Typical recovery of case of decomposition on protein milk (black). Rapid disappearance of the diarrhœa (+) with the appearance of soap stools (□) while the weight remains stationary. Later undisturbed growth on ordinary milk mixture.

1000 c.c. In typical cases, the dry-soap stool is formed on the first or second day; the weight soon remains stationary, and the child is moving on to uninterrupted recovery (Fig. 83).

When the quantity of food has been brought up to the required amount, without regard to whether the stools are infrequent or still numerous, the addition of carbohydrates should be made and increased gradually to five per cent. If possible, the carbohydrate addition is increased up to five per cent. in the course of ten days. If the weight remains stationary and the stools are homogeneous, even though they are not formed, the sugar may be increased to even more than 6-10 per cent. and 1 per cent. to 2 per cent. carbohydrates in the form of flour may be added.

In advanced cases of decomposition, the intestine must be emptied as rapidly as possible. In spite of the danger of starvation, a hunger period

of from six to twelve hours cannot be avoided. After this 200-300 c.c. of protein-milk is to be given in frequent feedings (eight to ten in number), during the first twenty-four hours with a rapid increase in the quantity of food and a decrease in the number of feedings; to which, within a week, carbohydrates, in quickly increased amounts, should be added. If the loss of weight does not cease within the first three or four days and there is a tendency to subnormal temperature and exhaustion, even though the stools are frequent, the carbohydrates should be further increased, in the effort to stop the condition.

Treated in this manner, the number of unsatisfactory cases is happily a small one. It is, of course, impossible to save children in whom the terminal comatose symptoms have developed. Experience has shown that several common mistakes are made in the treatment with protein-milk, which are responsible for seeming failures. They all arise from further injury to the patient through inanition or carbohydrate starvation. Among these errors may be mentioned: (1) the too gradual increase of quantity, thereby lengthening unduly the period of underfeeding and aggravating the condition; (2) the tardy addition of the carbohydrates and their use in insufficient quantity; (3) an arrest of the increase of food quantity and especially of the carbohydrates when the stools do not immediately improve; (4) a decrease of the amount of food and particularly of the carbohydrate when diarrhœa reappears, or when temperature rises, or a loss of weight occurs. All these errors are to be avoided. Only when a sudden decline in weight occurs and severe diarrhœa sets in, should the volume of the food be slightly reduced and the carbohydrate lowered to three per cent., just as is often necessary in dyspepsia or intoxication. After the disappearance of these symptoms, the return to full feeding should be made as rapidly as possible. In feeding the protein-milk the food-stuffs are less frequently responsible for such remissions than are the accidental infections.

During the first part of the period of treatment with protein-milk, tendencies to temporary aggravation, similar to those which occur when the infant is fed upon breast-milk, occur; but this should not hinder us from increasing the quantity of food. Rapid improvement usually follows, provided enough carbohydrate is supplied.

Protein-milk feeding may be continued for six to eight weeks in young infants; in older children from four to six weeks. After these periods, the repair of the disease condition will have progressed so far that milk mixtures, suited to the age of the child, will be taken care of. It is best to discontinue the protein-milk at once, replacing all the feedings with milk mixtures. If a relapse occurs, it may be necessary to return to the protein-milk treatment.

In a child who has suffered a disturbance of nutrition we may be assured of a complete recovery only when through a sustained period of feeding with ordinary milk-mixtures, within a normal range of quantity, the development continues its uninterrupted course (Fig. 83).

In the child with decomposition, medication is necessary only when a tendency to collapse occurs. Stimulants may be used, *e. g.*, citrated caffein;

caffein with sodium benzoate, in 0.5 to 1 per cent. solution, teaspoonful doses, four to five times daily; camphor, a 10 per cent. solution in oil, five to ten minims subcutaneously, every two hours; brandy, ten drops, several times a day. During the early period of the illness, artificial heat, by means of hot water bottles or warm baths, 36°-40° C. (96°-104° F.) should be applied; or, if possible, the child may be kept in an incubator. Care should be taken to avoid overheating, for the child with decomposition is especially prone to heat injury. The attempt to replace losses of water by means of normal salt solution is not efficacious in this condition, because the salt does not cause water retention, and because, in decomposition, it may produce edema.

For hemorrhage from duodenal ulcers, the treatment recommended for bleeding of the new-born in that section, is effective.

B. Nutritional Disturbances with Toxic Manifestations

I. Acute Dyspepsia

Symptoms.—Acute dyspepsia is distinguished from the dyspeptic form of dystrophy by its sudden onset and severe clinical manifestations. An infant that has been doing very well and apparently is developing normally, suddenly develops severe gastro-intestinal symptoms. Nausea and vomiting may precede the more marked signs, but very soon the bowels become extremely loose. The stools are watery with much gas and frequent. The intensity of the diarrhœa is usually greater than in the chronic dyspepsia. In other respects, the difference between the two conditions is rather one of degree only. All the symptoms of chronic dyspepsia are also seen in the acute, but are more severe. This is especially true of the alimentary fever which may be very high in the acute. Because of the greater severity of the symptoms of this form of dyspepsia, it may be considered as a forerunner of intoxication and treated as such. Improper treatment very frequently completes the transition.

The remaining symptomatology, metabolism and pathogenesis is identical with that of dystrophy with dyspepsia.

Etiology.—In most cases, a parenteral infection is probably responsible for the sudden disturbance. When no infectious process can be demonstrated the cause may lie in an excess of carbohydrate in the mixture or may be found in a sudden overfeeding.

Diagnosis.—The question whether the sudden serious condition arises in a previously normal infant or whether it is an exacerbation of an old disturbance of the nutritional function, or whether the patient is one of the not uncommon constitutionally "tropho-labile" infants, must be carefully answered, for the treatment depends entirely upon this.

Prognosis.—The result of proper dietetic treatment in a child that has been well up to the time of the acute attack is good. In very young infants, the danger of transition into decomposition is constantly present. In older children, the possibility of transition into chronic dyspeptic dystrophy must be kept in mind.

Treatment.—In the acute form of dyspepsia in previously healthy children the treatment may be based upon the fact that the patient has an uninjured tolerance and that the cause of the sudden diarrhœa is due to an acute discrepancy between the food ingested and the metabolic capacity. In such a case, rapid recovery may be expected after a short period of free purgation. Accordingly, the following procedure is recommended for the typical case. (1) A starvation period of not more than six to twelve hours. During this time liberal amounts of water sweetened with benzosulphinidum (saccharin) are given. The emptying of the gastro-intestinal tract may be made more complete by the use of gastric lavage and high enemata. If the diarrhœa is moderate, castor oil, one tablespoonful may be indicated. (2) After this, the food is again given but in greatly reduced amounts. Usually only about one-third of the caloric requirement is supplied. Large amounts of fluids are essential and the quantity may be made up by diluting the food or by giving water flavored with tea and sweetened. (3) Rapid increase of the food quantity should follow, so that the full requirement is again given by the second or third day. The underfeeding period must be limited to the shortest possible time.

As to the food, it must remembered that the diarrhœa will disappear no matter what food is used, if the quantity is small enough. This is true even though the food that caused the disturbance is continued. As in chronic dyspepsia, however, those mixtures containing the less fermentable carbohydrates give more certain results. But the carbohydrate additions cannot be omitted entirely except for a very short period of time. Mixtures high in fats are contraindicated because of the unfavorable action of fatty acids on the intestine already injured by the fermentation. Accordingly, in mild cases the feeding is best begun with a simple gruel, to which may be added small amounts of partially skimmed milk with the addition of a less fermentable carbohydrate using flour, dextrinized flour, maltose and dextrin preparation but no milk-sugar. Skimmed lactic-acid milk or buttermilk is useful, and calcium caseinate may be of benefit. Protein-milk is especially good. In fact the foods used in the chronic form of dyspepsia are also indicated in the acute stage.

Gruels or flour soups may be used in infants older than three months. Several objections can be raised against this treatment, however. In the first place, there is distinct danger of qualitative inanition (flour-feeding injury) due to the lack of protein and fat, and the danger that the food will be continued longer than was intended. In the second place, the change to a mixture containing milk is often very difficult because of the recurrence of diarrhœa when even small amounts of milk are added. If the gruel treatment is used, it is well to add a small amount of calcium caseinate and a few spoonfuls of meat broth for the mineral salts, and even then the mixture must not be continued for more than three or four days.

Cases treated according to the above method of procedure recover rapidly (Fig. 84). After a sharp decline of the weight-curve, as a result of the starvation period, the line gradually becomes less steep and after a day or so remains stationary. At the same time, the temperature also

becomes normal and the stools, less frequent, are more formed. As soon as the caloric requirement is completely covered, the weight increases. At times it may be necessary to add more carbohydrate to produce an increase, and this may be done even though the stools are not entirely formed and still more frequent than normal. If improvement is delayed, or there is still some loss of weight, no further time can be lost before instituting the treatment for the severe forms of dystrophy with dyspepsia, or for decomposition. No greater harm can befall these patients than persisting in underfeeding.

In those forms of acute dyspepsia arising from parenteral infection, such as coryza or grippe, strenuous interference is contraindicated and the case should be treated expectantly. As long as the infection does not produce marked loss of weight, it will hardly be found necessary to change the food.

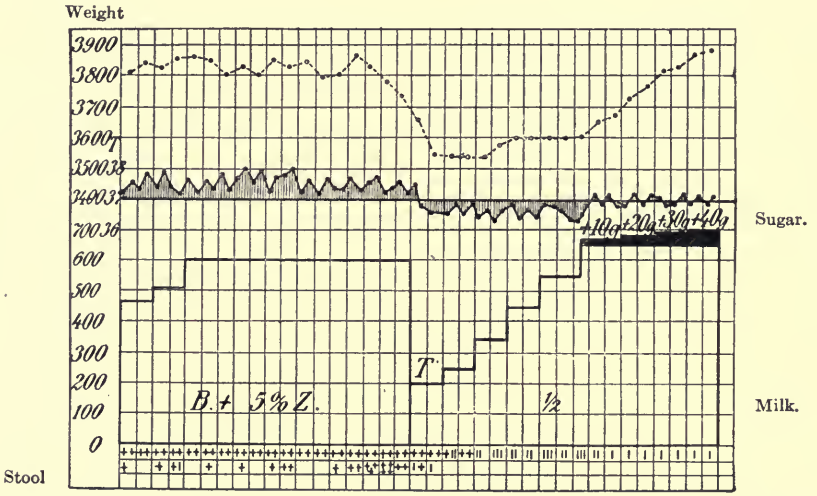


Fig. 84.—Dyspepsia with alimentary fever after feeding with sweetened buttermilk. Typical course of recovery. + = pathologic stool; | = normal stool.

After the infection has disappeared, the digestive function frequently returns to normal without therapeutic alterations of the diet. Only when there are severe losses of weight, or when the diarrhœa persists after the infection has been overcome, will it be necessary to institute the procedure described.

II. Intoxication

(Alimentary Toxicosis, Entero-Catarrh, Cholera Infantum, Etc.)

Intoxication may arise gradually from the acute form of dyspepsia, or it may at any time appear as an acute catastrophy in the chronic course of a disturbance of the type of dystrophy or decomposition. Furthermore, it may occasionally develop repeatedly in the same infant. In the previously comparatively normal child, the development of intoxication requires a rather severe injury, but in infants with decomposition, a very slight irregularity of the diet or a mild added infection brings on the dire symptoms very quickly.

Symptoms.—The first sign of the toxic action of food is the fever. In its mildest forms there is a slight rise above the daily maximum of temperature; at other times, subfebrile temperatures are seen; or even high fever may occur.

Alimentary fever, with dyspeptic stools continuing for a long period, may be the only symptom of the toxic nature of the disturbance. This condition does not necessarily preclude increase of weight. In most cases, however, additional indications are seen, such as loss of weight, weakness, and symptoms of disorder of the kidney. From these prodromes, complete intoxication may develop more or less rapidly and even acutely.

Typical and fully developed alimentary intoxication may be recognized by the following symptoms: Fever, collapse, severe diarrhœa, disturbances of the sensorium, sighing respiration, albuminuria, casts, glycosuria, leucocytosis, and abrupt loss of weight. Disturbance of the sensorium is seen early, in the form of abnormal lassitude and drowsiness. The patient is unusually quiet and lies very still or relapses rapidly into lethargy after being aroused. When he opens his eyes, the look seems vacant and can be engaged only with extreme difficulty. The usually lively expression is covered by a mask-like stare. Slight shadows lie about the eyes. In place of the ordinarily rapid movements of the healthy child, infrequent, slow, listless, apathetic and indirect gestures are observed. The normal pose of the limbs is replaced by unusual attitudes continued for a long time and due to a cataleptic condition. The so-called "boxer's position" is very frequent among these peculiar poses (Fig. 85).

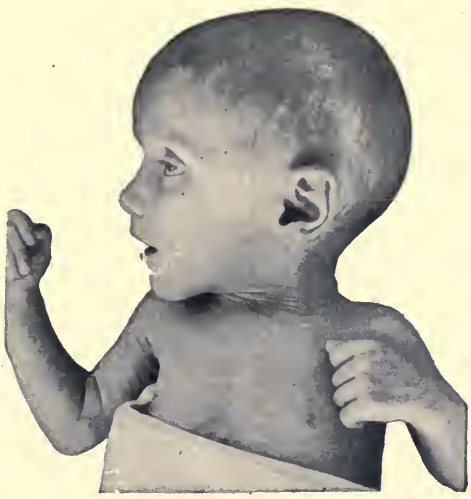


Fig. 85.—Facial expression of child with intoxication. Indication of "boxer's position" (Dr. Dessauer).

In severe cases true coma may follow this stage. From this the child awakens with severe jactitation and loud shrieks. As the condition progresses, these become less frequent and the child lies moaning in a deep stupor. Convulsions, other symptoms of meningeal and cerebral irritation and paralyses frequently occur.

Pyrexia or even hyperpyrexia is common; normal or subnormal temperatures occurring only in children with decomposition and intoxication.

The respiration is of that peculiar type frequently designated as "toxic breathing." The rhythm is long, deep, without pause and of increased rapidity, at times resembling that of the "hunted beast."

During the prodromal period, the stools resemble those of dyspepsia or decomposition. At the height of the illness they become very numerous,

watery, of a greenish-yellow color, wanting in substance and containing flakes of mucus. The reaction, strongly acid in the beginning, may change to an alkalin one on account of the excessive intestinal secretion.

Vomiting is frequent and, in definitely established cases, very violent. It may take so important a place in the symptom-complex that the designation of diarrhœa with vomiting is probably justified. In the severest forms, the vomited matter consists of dark, "coffee-grounds" masses which indicate gastric hemorrhage.

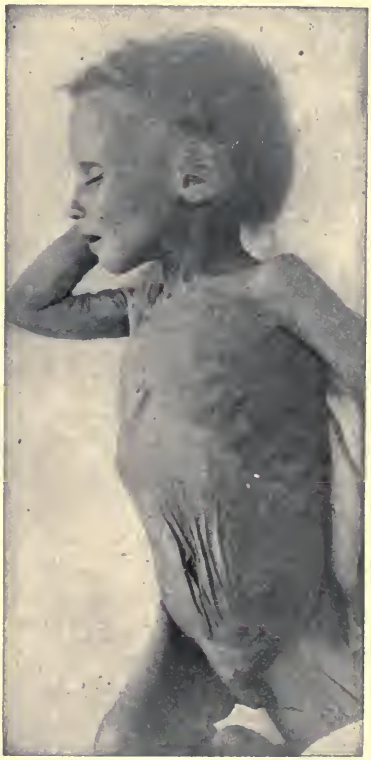


Fig. 86.—Severe dehydration, showing the loss of elasticity of skin. Cholera infantum. Two-year-old child.

Sharp decline in the weight-curve is caused by the excessive loss of water. Five hundred to one thousand grams (1-2 pounds) or more, may be lost in a few days. The skin becomes dry and plastic, so that if pinched the impress remains (Fig 86); the features are sharp and the fontanelle sunken. There may be muscular hypertonicity and painful cramps or contractures, especially of the leg muscles.

The drying out of the tissues is probably the cause of collapse, which is accompanied by a small pulse, dull and faint heart sounds, and cold and cyanotic extremities. The polycythæmia produced by the extraction of fluid from the blood is probably the cause of the characteristic pale bluish-yellow color of the skin. The urine contains albumin and usually much sediment with hyalin and granular casts. The occurrence of glycosuria[5] is purely dietetic; that is, it disappears upon the withdrawal of food. The sugar in the urine is of the form ingested. When the infant is fed mixtures containing sugar of milk, lactose and galactose are found in the urine; with other foods, other sugars are present.

There is always a leucocytosis, the maximum count being as high as thirty thousand.

In severe cases, fat sclerema, that peculiar hardening of the skin and subcutaneous tissues, beginning on the legs and buttocks and finally extending

[5] The demonstration of sugar in the infant's urine is not a simple matter. In doubtful cases, the osazone test should be used, in addition to the Trommer's and Nylander's tests. In the Trommer's test the urine should be boiled for quite a long time, since the cuprous oxide is not precipitated, in the presence of much ammonia, by simply heating. The exact identification of the sugar can be made only by microscopic study of the precipitated osazone.

over the whole body, may develop. The nature of this change has not been determined. The explanation, formerly given, of a solidification of the infantile fat of high melting point by the subnormal temperatures, is contradicted by clinical observations.

The large number of symptoms suggests the complexity of the disease. The clinical picture is varied by the prominence of one or another indication in the symptom-complex. Soporific, choreiform and cerebral types may be differentiated; the latter probably corresponds to the hydrocephaloid type of the older authorities.

Pathologic Anatomy.—The structural changes are not great and do not aid in the understanding of the severe clinical findings. A serous or serohemorrhagic catarrhal condition is found in the stomach and intestine. The gastric mucous membrane is covered with tough bloody mucus. The intestinal walls are injected, of a deep red color, and edematous. In the jejunum, there is a disseminated macular hyperæmia, with punctate or streaked hemorrhages. Peyer's patches are swollen and surrounded by a slight hyperæmia. Microscopically, the intestine may appear practically normal. More frequently, however, we see, besides a round cell infiltration, mucoid degeneration of the goblet cells. In severe cases there is destruction and marked loss of epithelium. In other organs, mild parenchymatous changes occur. In the liver, a capillary hyperæmia is a common occurrence and degeneration of the endothelial and liver cells is noted. Hyperacidity of the liver tissues and of the muscular tissues may be demonstrated by special staining methods.

Etiology.—The great resemblance of these symptoms to those of real cholera and cholera nostras in the adult, described in the older terminology, was the reason for the acceptance of an infectious etiology in the intoxication of the infant. Even though toxic symptoms may raise secondarily in enteral and parenteral infections of the young child, this explanation is hardly tenable. Not only has it been impossible to demonstrate specific organisms, but the most careful clinical observations show that we have to deal with a condition of alimentary poisoning in which the question of specific pathogenic germs is not to be considered; but in which fermentation products of the food or constituent parts of the food itself are the etiologic factors. The importance of preformed poisons in the food (exogenous milk decomposition), must also be excluded, since intoxication may arise with absolutely aseptic food.

Cases are seen in which the symptom-complex is of purely alimentary origin. In other and probably more frequent cases, there may be a mild infective process causing dyspeptic manifestations from which the intoxication develops as a secondary alimentary complication. After the infection has been relieved, the intoxication persists and forms the essential part of the picture. A third large group shows no relation to the feeding and must therefore be considered purely infectious.

The etiologic rôle played by the food is convincingly shown by the results of the withdrawal of food and the inauguration of the water-diet for therapeutic purposes. In all pure cases, uncomplicated by infection, we observe

a critical drop in temperature upon the cessation of feeding. In almost all such cases, which are not *in extremis* a critical detoxication also occurs If the food quantity be increased too rapidly after the hunger period, a relapse follows. Thorough investigations have shown that the causation of the diarrhœa and the fever is to be found in the action of the carbohydrates of the food, combined with the whey constituents (Fig. 87). If these are given in sufficiently large quantity, their pyrogenic action may be augmented by the toxic effect. Large additions of fat alone may exhibit a toxic action; but a primary fat poisoning is possible only when the metabolic functions have been previously damaged by a severe sugar injury, or by an existing decomposition, or by severe infection.

The Nature of the Toxic Condition; Its Pathogenesis and Metabolism.—So far as the results of investigation of the metabolism go, at present they show that intoxication is due to an insufficiency of all of the inter-

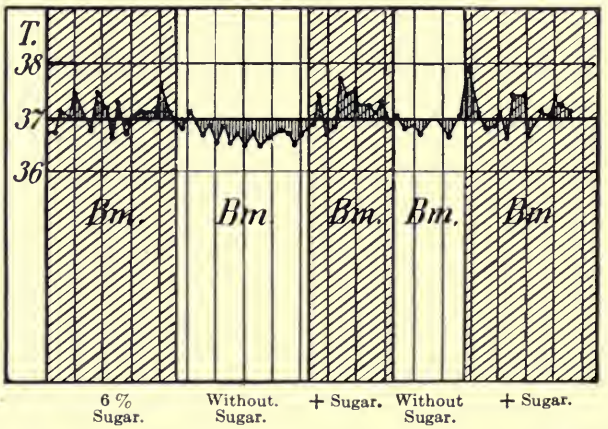


Fig. 87.—Alimentary fever during buttermilk feeding, produced by the addition of sugar (shaded days) and relieved by the omission of the sugar.

mediate metabolic processes, in which the evidences of acidosis are especially prominent. The present knowledge of the metabolism of these severe general disturbances permits the following statements as to the etiology. There is, no doubt, a far reaching injury to the intestine which permits abnormally free osmosis of the products of the perverted digestion from without inward and also, unquestionably, acts in the opposite direction. This condition is caused by the extreme decomposition of the ingested food, the products of which affect the mucosa. According to the older theory this abnormal permeability of the intestinal lining permits the absorption of bacterial poisons, the cause of the fever and the toxic manifestations. More recently, however the blame has been laid on the sodium salts, sugar and protein, for clinical observation on the pyretogenic and toxic action of these substances make it apparent that the physicochemical disturbance must be taken into consideration. It is possible that the portal blood carried to the liver is in a condition of osmotic imbalance, this injures the liver cells resulting in catabolic products which are causative factors of free purgation. Some experimental foundation for this theory has been brought out. If the diarrhœa becomes

very severe, there is a great dehydration and ultimately a lack of water in the tissues. In such a condition of dehydration, the oxidation processes would naturally be impaired. Very probably poisonous derivatives of food proteins or of body protein catabolism carry on the toxic condition. This would place great emphasis on the importance of the acute loss of water in the etiology of intoxication. This conception is supported by the fact that detoxication always occurs as soon as water retention is established.

Diagnosis.—The alimentary fever is to be distinguished from fever due to infection in that it is always abolished when food is withdrawn, or when a marked reduction of food is made. In a general way, this is also true in case of alimentary intoxication. But cases with severe toxic symptoms are either relieved very slowly or not at all by this reduction. Here, no doubt, the continuation of the symptoms is due to the persisting loss of water. If it is possible to cause water retention detoxication at once begins. These obstinate forms are unquestionably ones in which the intoxication is complicated with infection, the condition being infectious rather than purely alimentary. In other cases the carbohydrate and salt of the diet are so low as to prevent water retention. Finally, the bowel condition may be so serious that the cessation of feeding alone will not result in sufficient repair to prevent further loss of water.

Prognosis.—The prognosis is influenced less by the severity of the clinical findings than by the duration of the toxic condition, as well as by the status of the child at the incidence of the intoxication. Acute attacks, in previously healthy children, frequently encountered in those who are overfed upon buttermilk with large additions of sugar, if promptly and energetically treated by correct methods, have, in spite of the severity of symptoms, a good prognosis. Long duration of the toxicosis is naturally detrimental even to such children. A clinically mild attack of intoxication in children suffering with decomposition is very dangerous; because it is liable to overcome the remaining tolerance and because the unavoidable hunger-period is particularly serious. Every infection causes an irreparable complication of the condition. The difficulty of dietetic treatment of intoxication caused by infection depends upon the severity of the infection. The weight-curve gives the best index of prognosis. On account of the effect of water retention upon the weight-curve, the prognosis is more grave the longer the time required to bring the weight to the horizontal.

Treatment.—In severe cases, the complete withdrawal of food for the removal of toxic conditions cannot be avoided. The free supply of fluids must not be interrupted and this is best met by giving large quantities of weak tea. Since the greater danger of sugar intoxication is avoided by discontinuing all other feeding, it is well to sweeten the tea with benzosulphinidum (saccharin).

In order to arrest the drying out process as speedily as possible, salt solutions have been recommended instead of tea; [*e. g.*, physiologic salt solution, Heim-Johns' solution (NaCl 5.0, $NaHCO_3$ 5.0, Aq. 1000),

Méry's vegetable bouillon, or Moro's carrot soup[6]]. All of these solutions have the disadvantage of frequently prolonging the pyrexia through the pyogenic action of the salt. In certain circumstances, they even aggravate the toxic condition. They should, therefore, be well diluted (1:2 water), and should be employed only after the elimination of the poison has begun.

No objection can be made to subcutaneous injections of a physiologic salt solution, if contamination of the water is avoided by distillation. The 0.3 per cent. solution of the so-called detoxicated salt solution (7 gms. NaCl, 1 gm. KCl and 0.2 gm. CaCl in 1000 c.c. water) is preferable to the physiologic solution. This solution is also absorbed very rapidly from the peritoneum and may be given under conditions of ordinary surgical asepsis in amounts of 500 to 750 c.c., repeated daily.

The intestine may be emptied rapidly by washing the stomach and colon. This, however, is not absolutely necessary. Cathartics should not be used in severe diarrhœa; while stimulants, caffein salts, camphor, digalen, ½-1 drop every three hours; epinephrin, 1:1000, 0.5 c.c. (7 minims) with pituitrin 0.25 (grs. ii) intramuscularly every three hours: or brandy cannot, be dispensed with. Tepid baths should be used to control the high temperature; while frequently repeated cold packs should be avoided because of the danger of collapse. If the skin is cool, a warm mustard bath may be considered. Jactitation, convulsions, and severe attacks of pain make the exhibition of narcotics desirable; chloral, however, must be avoided because it may produce a soporific condition lasting for days. Veronal 0.075-.015 gm. (1-3 grs.), or sodium diethylbarbituate, 0.05-0.1 gm. (1-2 grs.) per dose, is better. Stomach washing and local anesthetics are most useful when there is vomiting. (See Pylorospasm.)

In favorable cases of simple intoxication, the poison is completely removed in from twenty-four to thirty-six hours of starvation (Fig. 88). Although the child grows weak and thin, it is lively, its eyes are clear and the bowel movements are less frequent. After such a period of starvation, it is absolutely necessary that feeding be resumed. The prime object of treatment should be to keep the symptoms of intoxication in abeyance by the smallest possible amounts of food; increasing it very gradually during the first few days, while a plentiful supply of liquids is given.

The course of treatment most to be commended is the feeding of breast-milk. Even with this, it is best to feed small quantities frequently. It is well to begin by giving the child 5 c.c. five times during the first day, 10 c.c. five times or 5 c.c. ten times on the second day; and 10 c.c. ten times on the third day; thereafter, at first slowly, and then more rapidly, giving larger quantities in fewer feedings. The sooner the danger of inanition is removed the better; on the other hand, great care must be exercised to avoid an aggravation of the condition by too rapid increase of food. This aggravation is shown in the renewed appearance of the toxic symptoms. It is better

[6] Carrot soup is prepared as follows: One pound of carrots scraped, cut into small cubes and boiled one to two hours. The soft carrot is passed through a fine sieve, into bouillon made with one pound of beef in one litre of cold water; to this is added one teaspoonful of table salt.

to feed the expressed breast-milk from the bottle at first; and often it is better cold than warm. After a few days of feeding by this method the child may be returned to the breast.

With the use of artificial food, detoxication may also be accomplished if small enough amounts are fed. The mixture must naturally be one in which there is a minimum of fermentable carbohydrate and one which affords the maximum water retention. A food containing very little fat and sugar and a

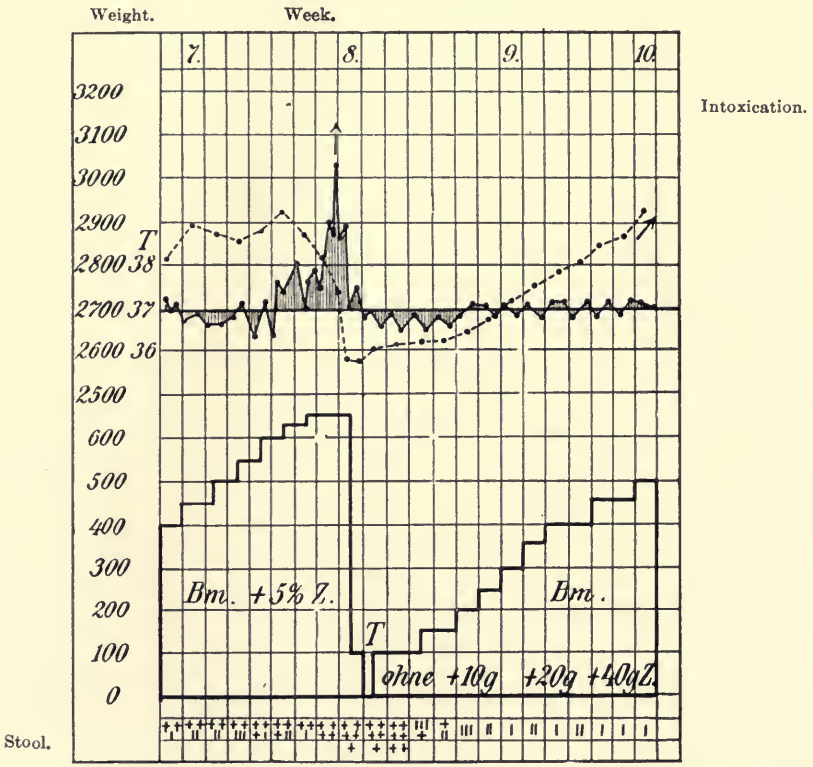


FIG. 88.—Typical intoxication in feeding diet rich in sugar and whey (buttermilk). Reduction of temperature and removal of poison by starvation period after which the amount of food is very gradually increased. + = pathologic stools; | = normal stools.

liberal amount of whey fulfils this requirement and buttermilk or skimmed milk without additions, are ideal. Whey has also been recommended but has no advantage over buttermilk. It is, in fact, less satisfactory because of the lack of casein which counteracts fermentation. The dosage and number of feedings should be as recommended for breast-milk. Even before the weight-curve becomes stationary, carbohydrate may be added and then the buttermilk gradually replaced by a whole milk mixture. After this, the treatment is that described for dyspepsia.

In those cases in which the poison is not fully eliminated, cases which were formerly classed as forms of mixed alimentary intoxication and infec-

tion, or those which should be included in the severe type of decomposition, the feeding must approximate that recommended above. Further starvation undoubtedly causes death. The only possibility of recovery lies in giving the small remnants of functional ability an opportunity to recuperate with a dietary as large as they can utilize. With children in the severer stages of decomposition this merely prolongs the trouble, while with infections, on the other hand, the disorder is frequently overcome and convalescence sets in. It is better, therefore, to rise very gradually to food quantities which are just sufficient to sustain life and to await results. Hopeful progress with these children may be expected from buttermilk with the addition of breast-milk or protein-milk, with a five per cent. carbohydrate addition. Protein-milk in itself seems to fulfil all the requirements and may be used without fear, if given in the same dosage recommended for the other foods.

INFECTION AND NUTRITION

Attention has been called already to the greater predisposition of the child with disturbance of nutrition to infections. As an actual fact, in but very few cases does the disturbance run its course without bacterial complications. Of most frequent occurrence are the infections of the skin, furunculosis and other pyrodermias, phlegmons and erysipelas; next in frequency are diseases of the air passages and lungs; sepsis and pyemia with their varied courses follow; pyelocystitis, otitis, and infections of cerebral localization also occur. These infections are not only incurred more readily, but because of the reduced resistance they run a more severe course, tend to spread and may become serious. A furuncle may increase to a phlegmon which extends with remarkable rapidity; severe pneumonia may follow la grippe, etc. The slow healing of the infected wounds in nutritionally disturbed children is very noticeable.

In turn, the course of any disturbance of nutrition is unfavorably influenced by each infection. A further reduction of functional energy is the inevitable result of the intercurrent injury. Every degree of effect may be noted, according to the resistance of the patient and the virulence of the infection.

An infection does not necessarily produce a secondary disturbance of nutrition, or one, at least of a severe nature. In healthy children, it frequently passes without loss of weight or even with a continued gain in weight. The stools may remain normal (Fig. 89). The bowel movements vary in different infections; hard, formed stools may occur in pyemia; while la grippe and colon bacillus infections produce thin, mucoid movements, the infectious nature of which is recognized by the fact that they are uninfluenced by dietetic therapy. Loss of weight in these cases is usually due rather to diarrhœa and vomiting. On the other hand, in constitutionally weak children and in those with a disturbance of nutrition, unimportant infections (*e. g.*, vaccination, Fig. 90), produce serious alimentary injuries. Of course, healthy children may be nutritively affected, either early in the course of a highly virulent infection, or later because of the gradual exhaus-

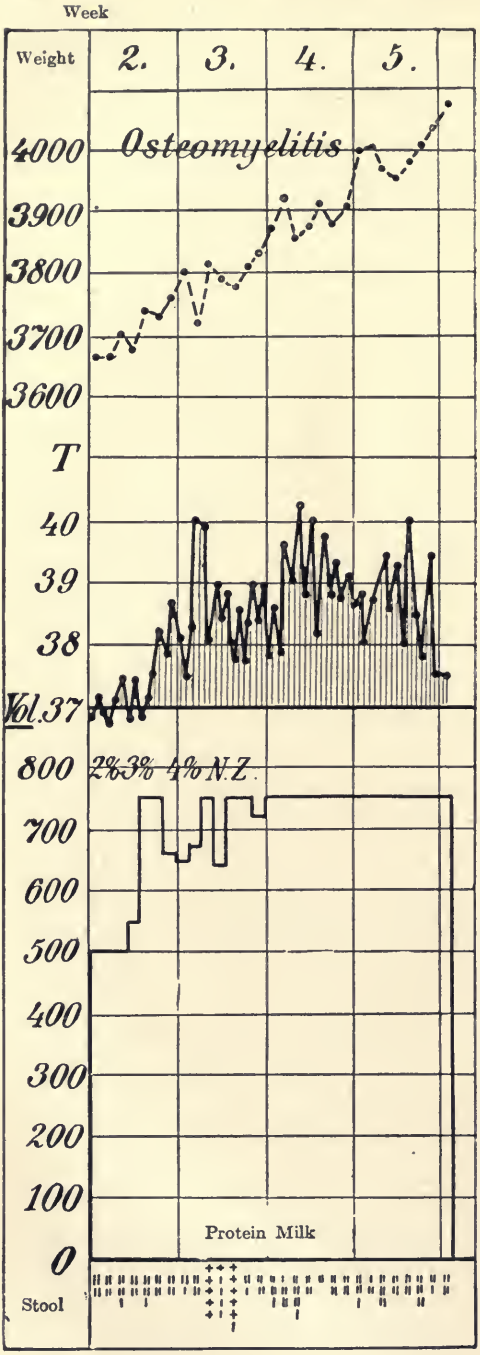


FIG. 89.—Course of severe infection in patient on protein-milk. No marked disturbance of nutrition (continued gain in weight, stools but little more frequent and but slightly changed).

tion of their original tolerance. The diet is to a certain degree important. Other conditions being equal, a given infection will produce a secondary disturbance of nutrition more readily in children fed upon those diets which tend to produce dyspepsia (*e. g.*, milk dilutions with carbohydrate additions, or buttermilk mixtures rich in carbohydrates), than in those fed with mother's milk or protein-milk. The conception of a relationship between infections and disturbances of nutrition has been already reviewed.

Symptoms.—The symptoms of mild secondary disturbances of nutrition resemble those of dyspepsia; that is, besides the signs of infection, dyspeptic stools occur, which may be improved by changes in diet while the fever continues. In contrast to simple inanition we have a marked and sudden loss of weight even when a fairly large quantity of food is being taken. This indicates a severe disturbance of nutrition, the character of which gradually comes to resemble more and more closely alimentary intoxication. The threatening loss of weight may be overcome, in many cases, by the same methods employed in a primary alimentary intoxication. The manifestations characteristic of the infection, however, remain. It is quite certain that a large percentage of cases running a severe toxemic course and formerly classified as general septic intoxications, were, in fact, no more than com-

plications of a severe secondary intoxication which could have been cured by dietetic therapy.

The combination of infection and disturbance of nutrition, that is of

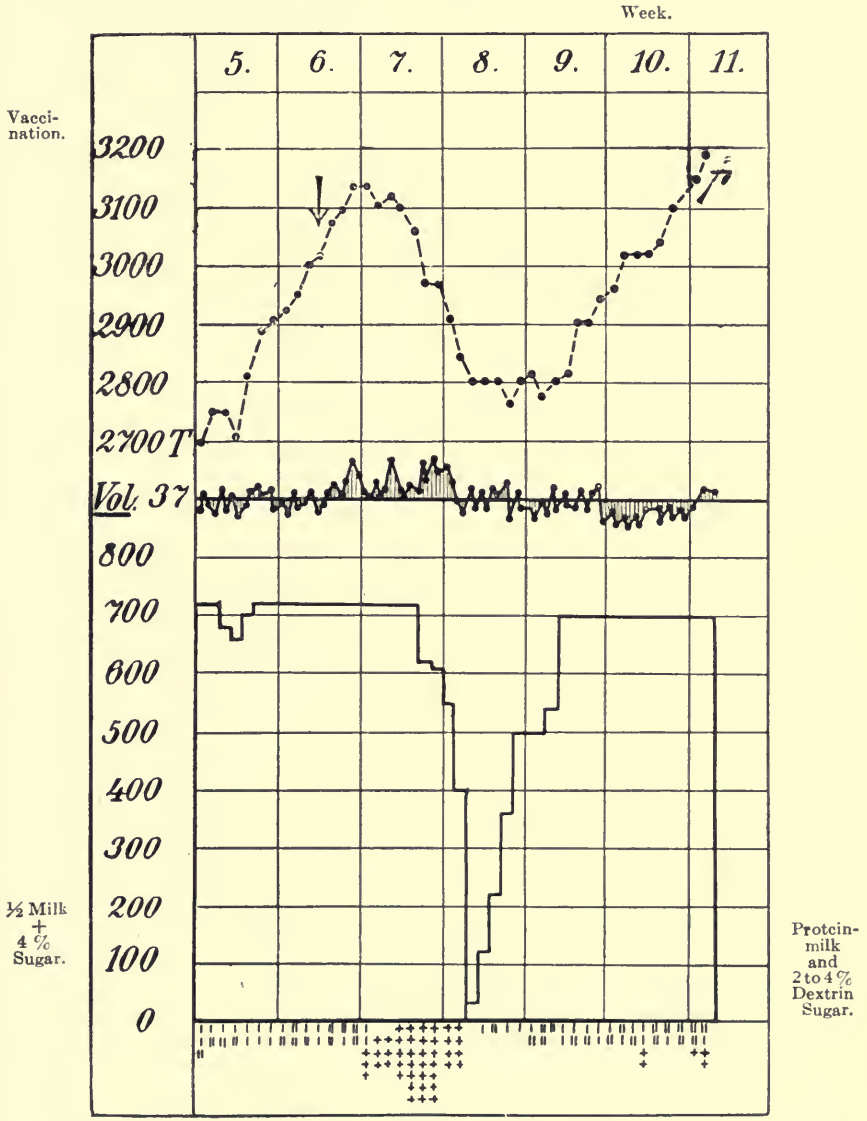


FIG. 90.—Severe secondary disturbance of nutrition resembling intoxication occurring as the result of vaccination. Recovery on diet of protein-milk. + = pathologic stools; | = normal stools.

alimentary fever and alimentary intoxication, produces many interesting complications. For instance, a fever may be partially due to infection and in part to alimentary causes; and the necessary changes in diet will then cause a lowering of the fever by eliminating the alimentary factor, so that

the hyperpyretic course becomes moderately febrile. Or, that which starts as an infective fever may lapse into an alimentary fever, without recognition of the change, since the infection has been overcome while the added secondary disturbance of nutrition sustains the fever, which yields only to a reduction of the food supply. Finally, the child may be so severely injured by the infection that the existing toxic condition is maintained, even in starvation, by an autotoxicosis.

After successfully combating an infection, many children, easily nourished before the infection, remain in a state of exhaustion which is exactly like the condition produced by a primary transgression of the limits of tolerance. The post-infective disturbances of nutrition, therefore, resemble in their symptoms and their reaction to food the picture of dystrophy, dyspepsia or decomposition.

Diagnosis.—The most important symptom of a secondary disturbance of nutrition, aside from the diarrhœa, the significance of which is not always clear, is the continuous loss of weight. If an infected child continues to lose weight rapidly, the cause must be a disturbance of nutrition, unless, indeed, the child absolutely refuses food. The loss due to slight underfeeding would cease in several days, or would be gradual. The reaction to the withdrawal of food or to other changes in diet would in itself show what symptoms, dependent upon the dietetic influence, could be considered alimentary.

Treatment.—With young infants the hope of overcoming an infection, and especially one complicated by disturbances of nutrition, is much greater if the child is fed at the breast than it is with the customary methods of artificial feeding, so that one must urge the use of breast-milk if it may possibly be obtained. Recent experiences have taught us that quite successful results may be obtained by the use of protein-milk (with a 3 to 7 per cent. addition of sugar). Under no circumstance, should the child be reduced to a state of inanition. Even with the most satisfactory diet, to say nothing of the rather generally used flour food, the patient is seriously handicapped by an insufficiency of food. As soon as loss of weight ceases, or even when the loss is slight, the food may be increased and should always be sufficient to allow an excess over the absolute necessity of repair. Only when a sudden fall in weight occurs and toxic symptoms appear should the food be discontinued for a half-day and then resumed in small quantities, as indicated in pure alimentary intoxication. In using protein-milk, a reduction of the carbohydrate content below three per cent. should be avoided. When the poison is not entirely eliminated by these methods, the food quantity must be increased in the same manner as heretofore advised. By this means, many children are saved who would certainly be lost under a starvation method. Diarrhœa alone is not sufficient reason for reducing the quantity in either form of feeding.

If the child is taking other forms of food at the outset of its sickness, and especially within the first three months of life, the change to breast-milk or protein-milk is to be recommended. If this is not possible, starvation is still to be avoided. If we are forced to reduce the quantity of food because of severe diarrhœa, sudden loss of weight, or symptoms of intoxication, the

case is extreme and the prognosis very questionable; at least among very young children but a few can be saved without the change of food. Of course, children suffering with infections may recover successfully with other food mixtures, but the percentage of unfavorable cases in very young infants is, other things being equal, considerably reduced with either the natural food or protein-milk.

THE DISTURBANCES OF NUTRITION OF BREAST-FED INFANTS

With the exception of actual underfeeding, the most important form of disturbance of nutrition of the breast-fed child is indicated by diarrhœa, with a "dyspeptic" condition of the stool. We should guard against making a diagnosis of a disturbance of nutrition upon the least variation of the bowel movement from the usual picture of the normal breast-milk stool, which is of a golden yellow and an inoffensive odor. Many children fed at the breast have occasionally or continuously green, thin, slimy evacuations containing fat flakes, incorrectly called "curds," which may be increased in number or otherwise changed without any special effect upon the general well-being. The causes and conditions which bring about such a changed consistency of the stools have not been wholly explained, but in no case do such stools justify the intervention by the physician and certainly not a departure from the method of feeding, either in a change of wet-nurse, a reduction of quantity, or, least of all, weaning. The child is to be considered diseased only when, besides seemingly abnormal evacuations, other certain signs of disturbance of development and of general well-being are found.

A distinction must be drawn between the exogenous diseases, *i. e.*, diseases due to causes extrinsic to the child itself, such as excessive feeding, improper composition of the food, heat, or intercurrent infection—and the endogenous diseases, due to the constitutional peculiarities of the child which cause a pathologic reaction to mother's milk. The former may be recognized clinically by the fact that they always occur more or less acutely after a period of normal development, while the latter appear immediately after birth and are chronic. It goes without saying that a disease of the first category may occur during the first few days of life.

Etiology.—The diseases due to improper composition of the milk play a large part as representatives of the first group. Formerly it was supposed that the milk of certain women could not be safely taken by infants. In other cases, the breast-milk was thought to be so affected by passing disturbances in the condition of the nursing mother, such as acute and chronic diseases, errors of diet, menstruation, psychic irritation, and even substances directly transmitted from the mother's food, as to do serious injury to the child. These beliefs, as a whole, must be classed as superstitions. Only in the event of menstruation or of intercurrent pregnancy is it possible that vomiting, restlessness, dyspeptic stools, may occasionally occur; but even then with but a small number of children. These disturbances are under no circumstances so serious as to warrant interference with, or change in diet, unless, of course, the breast ceases to functionate.

Of far greater importance, as causes of the injuries belonging in this category, are overheating, exposure to cold, neglect and, especially, infections. The parenteral infections, occurring with enteral or gastro-intestinal symptoms (coryza, la grippe, cystitis, stomatitis, etc.), may develop with such clear evidences that a diagnosis is readily made. Very frequently their course is so mild that, aside from the dyspeptic manifestations, only the most careful observation, as may hardly be had in the home, will give a good understanding of the conditions. Such cases are diagnosed incorrectly as the result of the harmful action of the breast-milk.

Overfeeding is another cause of dyspepsia. It seldom occurs with regular and infrequent nursing, but rather when the nourishment is irregularly given and at frequent intervals. The total quantity of the food is probably none too great in these cases.

Finally, hunger, that is, underfeeding, and probably only in certain individuals peculiarly susceptible, may bring about the symptom-complex of dyspepsia.

The symptoms of a disturbance of nutrition in the breast-fed child are practically the same as those of dyspepsia in the bottle-fed. Severe conditions are much more infrequent than in children under artificial feeding; but they do occur, sometimes with the picture of decomposition and more frequently in a transitional form between dyspepsia and intoxication; that is, with fever, apathy, lactosuria, etc. It is very improbable, however, that in these serious cases the food alone has had an unfavorable influence. They would rather appear in the first instance to be due to unskilful attempts at therapeutic starvation, and, second, to be incident to added infection.

Diagnosis.—The diagnosis of the dyspepsia of the overfed is made by the history and by determining the excessive quantity of milk. In other forms, it must be made by a careful observation of the environment as well as of the symptoms and the course of the disease.

Treatment.—Interference in the latter conditions is generally not only unnecessary, but strongly contraindicated. It is better to encourage the mother and to wait patiently until recovery takes place spontaneously. But with the dyspepsia of overfeeding, treatment must be more active. Stringent rules of nursing are necessary and, in severe cases, at times a marked reduction of the food quantity is required. If the child, being used to a large quantity of food, becomes restless, tea with benzosulphinidum, (saccharin), may be given; or, if necessary, during the first few days, a mild sedative, such as sodium diethylbarbiturate (medinal), chloral hydrate one teaspoonful of a one per cent. solution, two or three times a day or, in two per cent. solution, one teaspoonful, every two or three hours. Dyspepsia due to hunger recovers on the contrary, if sufficient food is given. In cases of other etiology, it is usually advisable to wait. Only when severe loss of weight occurs and slight somnolence, cyanosis and other indications of a more severe condition appear, is it necessary to give the breast-fed child a short hunger period and then to increase the food quantity gradually, as in the case of the artificially-fed babe.

DISEASES DUE TO ENDOGENOUS (CONSTITUTIONAL) CAUSES

Symptoms.—Children are occasionally seen who, from birth, do not develop well with breast feeding. In severe cases, there may be loss of weight, due to the lack of appetite, and with this distinct and frequently marked signs of dyspepsia. In typical instances, there is anorexia, severe flatulence, attacks of colic, frequent eructations, hiccough and regurgitation and withal a great general restlessness and emaciation. The stools frequently show the characteristics of a fat diarrhœa. By a combination of all these signs with eczematous and intertriginous changes, serious disease-pictures may arise. With many of these children, other signs are found which are indicative of a neurotic diathesis, such as: loss of muscle tone or hypertonicity, ptotic manifestations (diastasis of the recti, floating tenth rib, visceral ptotic, hernias, etc.), a tendency to be readily frightened, an increased muscular irritability, vasomotor pallor, etc. Furthermore, the history may reveal hereditary nervous stigmata.

Etiology.—Formerly there was a tendency to blame the unfit composition of the breast-milk for the disease-picture we have described: that is, the milk was supposed to be too rich. While it must be admitted that in exceptional cases improvement is had by a change of wet-nurses, it is the common rule that such children do not develop properly with any breast-milk. Considering, moreover, that the very food which is of so little advantage in these cases is satisfactory for other children, it is clear that not the food is to be blamed, but rather the constitutional peculiarity of the child which causes the paradoxical response to natural feeding. In fact, we have to do, on the one hand, with neuropathic individuals; and, on the other hand, with representatives of that anomalous constitutional quality which, at present, we prefer to designate as an exudative diathesis. Both of these conditions may appear separately or together. No clear conception of the relation between the constitutional disturbance and the symptoms referable to the digestive apparatus has yet been formed.

Treatment.—In the treatment of these disturbances, the customary change of wet-nurses is not to be recommended. Only rarely is it possible to find a wet-nurse whose milk will be better for the child than its mother's and very probably the expected result will not be obtained by even a number of changes. An especial warning must be given against the attempt to treat dyspeptic symptoms in these cases with starvation. Hunger never does any good and is always harmful. On the other hand, we may resort to satisfactory measures, the action of which is not to be entirely explained, in the way of additions to the human milk of protein preparations, or in the complemental feeding of cow's milk. One dram of calcium caseinate in one ounce of mineral water, may be given three to five times a day from the bottle, or with a spoon. Of the cow's milk mixtures, the buttermilk mixture gives the best results. In mild cases, the addition of one meal may be sufficient, while in more severe, two or three may be necessary with a corresponding reduction in the quantity of breast-milk. The results of both of these methods, in the increase of weight and of appe-

tite and in the improvement of the general condition are almost always astonishing (Fig. 91). In many cases, however, the stools remain slightly dyspeptic for a long time.

Idiosyncrasies.—Occasionally a dyspepsia, due to constitutional causes, reaches such a grade of severity that we may speak of an idiosyncrasy to breast-milk. The patient may even die despite of mixed feeding or weaning. This may, of course, be considered as the result of a long continued inanition due to the spontaneously inadequate nursing, or to the misdirected attempts at remedy on the part of the physician. Another rare form of idiosyncrasy to breast-milk is seen in the fainting spells which occur from birth at every nursing, or at least several times a day, and which disappear after weaning. It is not certain whether the composition of the breast-milk itself produces

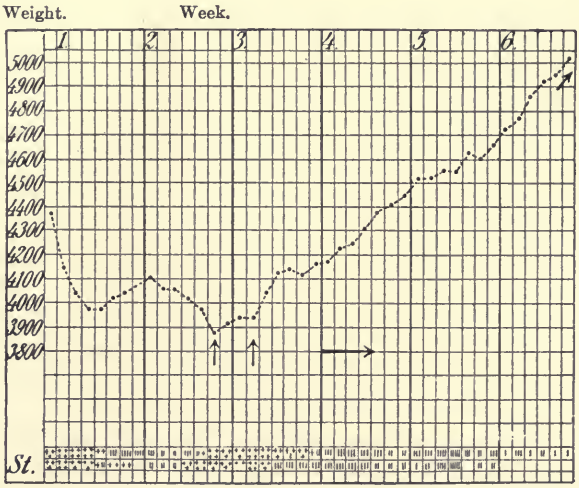


Fig. 91.—Recovery from severe endogenous constitutional dyspepsia by the use of breast-milk with addition of dextrin and maltose and mineral water.

the injury in these cases or, as seems more probable, that the exertion of sucking, incident to an abnormal vasomotor irritability, is at fault.

The idiosyncrasy of some breast-fed children to cow's milk is also of importance. These children, having acquired a disturbance of nutrition under artificial feeding, when given breast-milk develop satisfactorily, but when the attempt at weaning is again made respond to the first addition of cow's milk with an abnormal reaction. This has been observed in mixed feeding with doses of cow's milk as small as 5 c.c. or less. The symptoms vary according to the doses and the sensibility of the child. In mild cases, they resemble a slight dyspepsia, with fever, which appears a few hours after the cow's milk has been given; in severer cases, the complete picture of an intoxication, with even a fatal ending, may be produced.

Such children must be fed at the breast for a long time until they gradually lose their sensitivity. Sometimes it is possible to develop a tolerance by increasing the quantity of cow's milk drop by drop. Severe toxic reactions

must be treated by prompt withdrawal of the food and, later, by such careful feeding as in intoxication; for even though the cow's milk be wholly discontinued and the child be permitted to take as much mother's milk as it wants, an unfavorable result may be unavoidable.

DISTURBANCES OF NUTRITION OF OLDER CHILDREN

After the first year of life, more particularly after the second year, the constitution of the child is, as a rule, so firmly established that the severe disturbances of nutrition which so frequently develop in the nursling are hardly ever encountered. Its disturbances are usually classified as acute or chronic dyspepsias. They correspond in fact, to those conditions which have been described above for dyspeptic infants within narrower lines, in that their symptoms are entirely those of local fermentation and irritation, while the general well-being and the metabolism at large are not altered in any noticeably severe degree. Nevertheless, occasionally, even in older children, symptoms appear which in their form and mode of onset are distinctly those of alimentary fever and alimentary intoxication (see acute dyspepsia); and, similarly, chronic disturbances develop of so severe a nature that they fit completely into the picture of decomposition (compare chronic digestive insufficiency). The probability of a pathogenetic identity, with slight variations between the disturbances of nutrition in these older and younger children, is the more readily conceivable when we consider that in both the same therapeutic principles give identical results.

ACUTE DYSPEPSIA AND DYSPEPTIC COMA

Symptoms.—Acute dyspepsia has a sudden onset and is initiated by headache, loss of appetite and malaise, with nausea, vomiting, and fever and frequently, marked prostration. The tongue is coated, there is strong fetor from the mouth, frequently a distinctly acetone odor, slight distention of the abdomen, constipation and often, later, diarrhœa. The pulse is rapid and may also be variable, or occasionally slow and irregular. Albumin and casts are usually found in the urine.

With proper treatment this condition does not last long; convalescence may set in after two or three days, or even less. In other cases, it may drag on for a longer time and a condition which may be called "*status gastricus*" develops.

In a considerable number of cases, other symptoms are associated with those enumerated which make the disease-picture more impressive. These symptoms in their variety and intensity are those recognized in the intoxication of infants, and, when sufficiently developed, produce dyspeptic coma. Before the disturbance of consciousness goes on to coma, symptoms of spinal and cerebral irritation may appear, occasionally severe convulsions continuing for hours; deep respiration is observed, and sugar, or at least strongly reducing substances, may occur in the urine. The acetone odor is especially strong and unusually large amounts of acetone can be demonstrated in the urine.

The prompt results which follow in one or two days after emptying the

intestine, show that the cause of these symptoms, as of simple dyspepsia, is an intoxication arising in the digestive tract. Nothing further is known about the poison. It is very doubtful however whether we have to deal with acetone poisoning, as was formerly believed. The clinical resemblance, also, to intoxication in the infant indicates that similar conditions are causative. A dietetic error, an overloading of the stomach or some similar circumstance is usually the cause of the dyspepsia; but frequently such a cause cannot be established. Then, doubtless, an infection of some kind has given rise to the appearance of secondary alimentary disturbance, the symptoms of which still remain after the removal of the signs of dyspepsia. This frequently occurs in the course of influenza.

The diagnosis is not always easy at the beginning. Typhoid and paratyphoid, meningitis and similar conditions may come up for consideration. Many disturbances diagnosed as dyspepsia are certainly nothing more than an infection exhibiting gastro-intestinal symptoms. The surest differentiation is given by the prompt results of a thorough emptying of the stomach and intestine, after which any disturbance that can be classified as dyspepsia must disappear; if symptoms remain, it is certainly not primary dyspepsia, but only dyspepsia accompanying some other disease.

The treatment consists in the removal of the gastro-intestinal contents as rapidly as possible. Calomel 0.05-0.1 gm. (1-2 grs.), repeated two or three times; castor oil, rhubarb, etc., are used for catharsis; enteroclysis, or glycerin enemata are useful. Occasionally gastric lavage may be employed. Emetics, such as syr. ipecac 4 c.c. (1 dram), wine of antimony a teaspoonful, are not of much use. This treatment should be followed by a liquid diet; for several days small amounts of food should be given and finally a tonic.

An "asthmatic dyspepsia" must also be described. This is a condition which begins suddenly with dyspnœa, lasting for hours or days, and is relieved by emptying the intestinal tract. How much of the effect is mechanical and due to meteorism, or the high position of the diaphragm, and how much is due to reflex action and intoxication imposed upon a nervous constitution is not clear.

CHRONIC DYSPEPSIA

(Chronic gastro-intestinal catarrh, chronic intestinal and colonic catarrh.)

In the majority of cases, chronic dyspepsia develops from an acute intestinal disturbance, usually of infective origin which may be either enteral or parenteral. A primary acute dyspepsia is less frequently causative. In certain patients the onset is very gradual and neither the period of incidence nor any external influence is recognized. In these cases, constitutional faults are of probable importance, since there are no dietetic errors to explain the congenital or even the familial disability of the digestive functions.

The greater number of cases occur during the second to the fourth year. At later age disorders of this class are more infrequent. Many

chronic intestinal disturbances of older children have their beginning, however, in infancy. Various forms of chronic dyspepsia are seen.

Chronic dyspepsia of gastric origin is infrequent compared with other forms. It occurs in lean, pale, capricious, morose children where the one symptom indicative of gastric disorder is a marked anorexia. To this may be added occasional eructation and vomiting and sometimes the abdomen is distended in its upper portion. With the test-meal, we find abnormal chronic conditions in the way of a slight catarrh and a particularly marked motor insufficiency of the stomach which, in severer cases, is combined with atony or even gastro-paresis.

Gastro-paresis is a condition of hypotonicity, in consequence of which distention of the stomach becomes abnormally great even with moderate amounts of food. The most extreme degree is atony or atonic dilatation, in which the stomach cannot regain its normal size even when empty. It contrasts with the mechanical distention of organic stenosis in that it exhibits no anatomic changes in the gastric walls. Mild degrees of gastro-paresis are frequently found in cases of general debility and improve when this is removed. Only those cases are of importance to the physician in which the insufficiency of the organ is so great as to be the chief cause of existing symptoms.

Constipation is common, although gastric fermentation may produce intestinal irritation and chronic diarrhœa. This must always be considered as gastric in its origin.

Severe grades of the disease may lead to marked exhaustion and may even result in death. With careful treatment, the prognosis of even the more advanced cases is quite favorable. A considerable time may elapse, however, before complete recovery results. In its diagnosis, nervous anorexia must be considered. Usually it may be differentiated without the use of the stomach-tube, since children with nervous anorexia are lively and the appetite for some kinds of food is good. With existing diarrhœa, the differentiation from other forms of dyspepsia must be made with the stomach-tube.

In the common form of chronic dyspepsia disturbance of the gastric function is not so prominent a symptom. In the disease-picture the intestinal symptoms, and especially diarrhœa, overshadow all others. Lancinating and colicky pains may occur. The appetite is variable; it may even be good. The evacuations are commonly not very numerous and contain more or less mucus. The stools present variable special findings. Their reaction may be either acid or alkaline. Undigested food particles may consist of fat, starch and vegetable shreds. Connective tissue shreds are found only after the ingestion of raw or partially cooked meats; muscle fibres are rarely seen. Foamy fermentation is present—a fermentation so intense that it continues after the evacuation of the discharges. The general health is affected in proportion to the difficulty of feeding; nutrition is more or less unsatisfactory. Mild anemia is common and, in younger children, rickets. The majority of cases show symptoms of nervous irritability and in infants latent spasmophilia is common.

The repeated occurrence of exacerbation is very characteristic of this disease. An aggravation may be brought on by other intercurrent diseases (respiratory catarrh, etc.), or by overfeeding or improper food. A repetitional attack may be accompanied by severe diarrhœa with blood and mucus, accompanied by fever, with serious impairment of the general health. Even choreiform or typical attacks of tetany may occur. Many undigested particles, or even larger masses of food which have not properly been broken up, may be found in the stools at this time.

Of the cases belonging to this group, quite a large number are in the nature of fermentation dyspepsia, due to imperfect digestion of carbohydrates (A. Schmidt). In these, the evacuations are thin or pasty, of varying consistency, containing variable amounts of mucus; they are light in color and filled with bubbles. Their reaction is acid and the odor, sour. The iodine test gives a strong starch reaction. Microscopically, one may discover, besides the starch cells, many iodophilic bacteria. Careful chemical analysis shows that, on the average, the carbohydrate content of the feces is more than doubled. In the saccharimeter, fermentation occurs after twenty-four hours.

Typical cases of this kind can be readily diagnosed; indeterminate forms in which the consistency of the stools is not so characteristic are probably more common. In these, one may have stools of darker color, of changing reaction, with no response to the starch test. In spite of these findings, it must be remembered, in seeking proper therapeutic measures, that some carbohydrate is the initial cause of the fermentation dyspepsia. While the carbohydrate may have been so well digested that no free starch is found in the colon, we may still have acid formation. The acid reaction may be neutralized by the secretion of the large quantities of the alkaline intestinal fluids.

It is probably true of older children, as well as of infants, that a disturbance of carbohydrate digestion is the primary cause of dyspepsia; that the disturbance of fat and protein digestion is secondary to the amylolytic and glycolytic failure, and that the absorptive and peristaltic failure are caused by the acids of carbohydrate fermentation. The supposition that a primary fat or protein digestive disturbance is the basis of this lienteric condition is, in general, unjustified.

Mucous Colitis.—Only in one special form of disease which should be classed not as a dyspepsia, but rather as a catarrh due to local irritation, does the protein digestion play a particular rôle. This is mucous colitis. This disease, which runs its course without important suggestive symptoms, is characterized by the passage of large quantities of mucus, at times resembling casts of the intestine and again covering the fecal masses. In this condition, children have usually been fed large quantities of meat and eggs, while the vegetables and carbohydrates of the diet have been reduced. The constipation thus produced is usually the cause of the catarrh of the colon which results in this excessive mucoid secretion. Mucous colitis must not be mistaken for membranous enteritis or mucous colic, a condition in which long white shreds and tubular casts of the intestinal tube are passed under extremely severe pain. These shreds and casts consist of mucin; the

disease involves no inflammation of the mucous membrane. Their evacuation occurs at intervals of weeks or months. The disorder is probably in the nature of a secretory neurosis.

SEVERE CHRONIC DIGESTIVE INSUFFICIENCY IN OLDER CHILDREN

Those not infrequent cases which are termed *severe chronic digestive insufficiency in older children* must be regarded as chronic dyspepsia in its most severe forms. Children affected with this condition usually come from families with a severe neuropathic taint and often show neuropathic symptoms themselves. While some of them have passed infancy without noticeable disturbances; others have shown, even at this time, lowered resistance, and digestive failure. The actual disease begins insidiously or as a sequel to acute dyspepsia or to an infection. Its chief characteristic is the marked liability to digestive disturbances. Slight dietetic errors, or unimportant infections promptly produce intestinal symptoms severe in themselves and of severe effect upon the general well-being. It is not uncommon to see a loss of several pounds in weight within a few days in connection with a slight coryza, a vaccination, or following the ingestion of some unaccustomed food. Extreme weakness and debility are cause for anxiety. A choleric alimentary intoxication with all its typical symptoms may develop. A second characteristic peculiarity is the stunted growth and retarded gain in weight. The occurrence of long periods with no gain in weight, even though there is no intercurrent disease, show how greatly the growth suffers. Children of four years, but with the weight of a twelve months old infant and with size corresponding to weight are not uncommon; so that we may be justified, to a certain extent, in speaking of this as a type of infantilism. A third characteristic may be mentioned in the abnormally reduced capacity for repair.

While it is possible in ordinary forms of dyspepsia to restore the intestine to normal functional ability after several weeks of careful dieting, in these severe forms a sensitivity remains, even after the bowel movements have been normal for a long time. A slight change in, or increase of diet may immediately cause an aggravation of the disease.

The number of evacuations, excepting during these times of intercurrent aggravation, is normal or but slightly increased. The massiveness of the stool is especially noticeable; it may weigh a pound or more; the reaction is usually acid and there is a strong tendency to fermentation, which frequently continues after evacuation. The food substances are poorly digested; that is, large percentages of fat and, in severe cases, of the starches also, are excreted. The protein digestion, on the contrary, is not noticeably reduced, excepting at the time of acute aggravation, when large amounts of undigested meat shreds are found in the stool. Protein digestion is also unfavorably affected in those children in whom the test-meal shows an existing achylia.

The nutritional condition of the patient is poor. The abdomen is always distended (Fig. 92). During the periods of diarrhœa, we may have a

picture of pseudo-ascites, because the convolutions of the bowel, filled with fluid, are drawn to the dependant portion of the abdomen and cause dulness and fluctuation.

The disease may continue for years; periods of improvement and aggravation alternate; growth and weight are retarded for months and even years. With puberty, the disease may recover spontaneously, excepting for a slight sensitiveness. Of course, growth remains below the normal in a large number of cases. A fatal outcome is not infrequent.

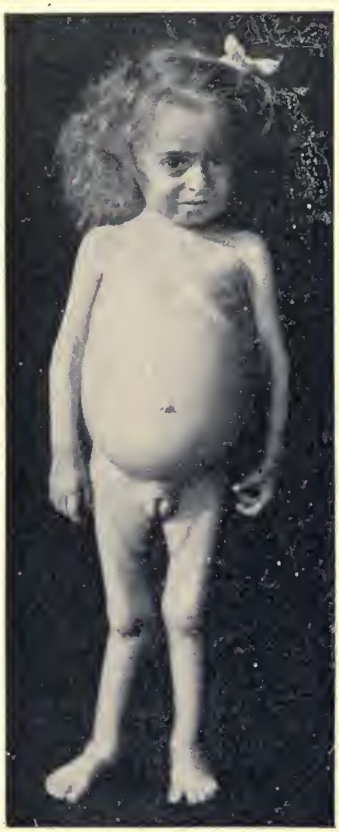


Fig. 92.—Intestinal infantilism. Girl of three years, 76 cm. (30 inches) tall, weight eight kilos (17.6 pounds), large abdomen, old face. (Children's Hospital, Zurich, Prof. Feer.)

The diagnosis of chronic dyspepsia is based principally upon examination of the stools. The reaction and the test for starch with iodine are, aside from simple inspection, the chief methods of macroscopic diagnosis. For the more careful determination of the results of digestion of meat, fat, vegetables and starch a microscopic preparation is essential. The test-meal (A. Schmidt), appears necessary only in exceptional cases. To determine the integrity of gastric function, it may be necessary, particularly in those conditions which do not yield to therapy, to give a test breakfast.

In the treatment of dyspepsias of gastric origin, careful attention should be given to the avoidance of recurrences and to the increase of the functional capacity of the stomach. It is well to begin with stomach washing accompanied for several days by very scant feeding, without resort, however, to actual hunger therapy. Gradually the diet is increased, making use of butter or cream, prepared flours, meats and vegetables passed through a fine sieve. Renewed anorexia necessitates further reduction of the diet. Frequent use of the stomach-tube and of lavage at times becomes necessary. With continued care the functional ability may be greatly increased and the diarrhœa disappears. In severe cases, however, a year or more may pass before complete recovery takes place. As to medication: pepsin and hydrochloric acid; *tr. rhei*, 20 c.c. (5 drams), with *tr. nux vomica* 2.0-5.0 c.c. (½-1 dram), 5 to 20 drops, before each meal may be useful.

The usual treatment of the ordinary forms of chronic dyspepsia is dietetic. The basis of the diet is flour soup. It is customary to give cocoa, oatmeal water, proprietary foods, cereals, boiled rice, with the addition of meat juices and protein powders; and, later on, to add a pap of measured

vegetables and finally meat soups. For younger children, Liebig's malt soup is to be recommended. In quite a number of cases, the intestine actually recovers so far that a transition to mixed diet is possible. It is clear, however, that this course is not proper in dyspepsia due to carbohydrate fermentation; but that, on the contrary, carbohydrates must be reduced. It is better than to give chiefly meats, white cheese, eggs, vegetable soup, well-mashed vegetables, spinach, lettuce and fruit. Carbohydrates are best given in the form of toast, white bread, and soups, with very fine flour in quantities adjusted to the reduced tolerance. Taka-diastase in tablet form is frequently useful. The diet should be increased according to the indications given by examination of the feces.

Such a method of treatment is advantageous in typical cases, and is to be advocated whenever the carbohydrate treatment is not satisfactory. When the stools are acid or contain starch, this is especially advisable. The results are usually certain. Milk is hardly ever satisfactory and it is better to discontinue it entirely or to reduce it to as small a quantity as possible. Care should be taken in the allowance of sugar or sugared foods; stewed fruits should be prepared without sugar. Potatoes are frequently injurious. At first, meat should be given in chopped form and vegetables should be passed through a fine sieve. Course vegetables frequently maintain an irritating fermentation because the contained starch, surrounded by cellulose, escapes digestion in the upper part of the intestine. In children in the second year, protein-milk may be useful.

Similar rules apply to the treatment of severe digestive insufficiency. Varied diets are generally of great value in these cases because the status of these children is, to a large extent, dependent upon psychic factors, and a monotony of diet increases the suffering. The variety of foods which these children will bear is surprising and nothing is more harmful to them than an unbalanced flour and milk diet. Further indications may be gained by examination of the stools and the stomach contents. Frequently it is necessary to reduce the fats; in cases of achylia, the meat; and often again the eggs. With evident fermentation, at the beginning of an acute aggravation, the best results are obtained by a temporary reduction of the carbohydrates and even of the other food-stuffs. Days of hunger, or longer periods of underfeeding are hazardous. Little assistance can be expected from the digestive ferments.

It is hardly necessary to add that in regulating the diet of the patient, not only the quality, but also the quantity of the food and of its individual food components must be given careful consideration. Failure of treatment is frequently due to too much or too little food. A great influence for recovery is also employed by psychic stimulation and change of climate.

In the treatment of mucous colitis, it is necessary to increase the carbohydrate element in the diet, reducing meat, eggs, cheese and the like, and giving fruit, coarse vegetables and other foods containing large amounts of indigestible residue. Gentle intestinal lavage and occasional treatment with oil are useful.

The recovery from all forms of chronic dyspepsia is hastened by the improvement of the general health and especially by proper climatic conditions. In the severer types, one occasionally sees great improvement at the seashore or in the country. A water cure (Karlsbad) may at times be useful. So far as medication is to be considered at all, the methods recommended in infective intestinal catarrh may be employed.

ACUTE INFECTIOUS DISEASES OF THE GASTRO-INTESTINAL TRACT

Aside from the actual disturbances of nutrition, we find many diseases in childhood and especially in infancy which must be looked upon as infections of the digestive tract caused by the invasion of pathogenic organisms. The infection is often carried by the food; the disease-producing organism may come from diseased cattle (streptococci from abscess in the udder, or colon bacilli from diarrhœa), or the pathogenic organisms of human disease may have been introduced in some manner during its journey to the consumer. Numerous cases are seen in which the infection is carried directly from other individuals. Observations of house epidemics and especially of the epidemic-like outbreak of gastro-enteritis in children's hospitals and in institutions for the care of infants give definite proof of this.

The most common etiologic factor of infectious intestinal catarrh is doubtless grippe. Gastro-intestinal forms of this malady in varying degrees of severity are at times epidemic in extent. In other cases, blame must be placed upon varieties of the colon group, upon streptococci, paratyphoid or dysentery bacilli, and occasionally upon the pneumococcus, the *B. pyocyaneus*, the *B. proteus*, etc.

The symptomatology of the diseases produced by these different organisms is usually so variable and so little characteristic of any causative group that it is hardly possible to classify cases etiologically. It seems more to the point to differentiate them clinically.

Catarrhal gastro-enteritis is to be distinguished by its mucopurulent and occasionally, slightly bloody diarrhœa, without indications of colitis. The attack occurs suddenly and is accompanied by a rise of temperature. According to the height of the fever, the severity of the diarrhœa and the effect upon the general health, we may distinguish mild from severe cases. The mild forms may resemble an obstinate dyspepsia and the severer may present a serious disease-picture with the appearance of cholera-like symptoms. With proper treatment, the disease usually does not last longer than one to three weeks.

Most of the cases may be traced to a grippal infection as shown by the coincidence of symptoms of respiratory disorder (coryza, pharyngitis, bronchitis, etc.). On this account they are frequently termed broncho-entero-catarrh. In young infants, more especially in the new-born, and but rarely in older children, one sees similar disease of septic origin in which the upper portion of the intestine presents a hemorrhagic, purulent, even ulcerative inflammation of the mucous membrane, caused by streptococci or

similar organisms. Such forms usually run a very severe course and frequently have a fatal termination.

In epidemics of Asiatic cholera, children, especially within the first ten years of life, are attacked in great numbers and the mortality up to the fifth year is very high. Of infants sick with cholera about eighty per cent. die; during the second five years, only about fifty per cent. are lost. The clinical picture differs little from that in the adult. Both in mild and more fully developed forms, the disease appears with the algid stage; and as in typhoid. The resemblance to ordinary diarrhœa with vomiting, and to alimentary intoxication, may be great, so that the diagnosis, excepting in epidemics is very difficult.

Typhoid-like gastro-enteritis, so-called gastric fever, is especially distinguished by high fever, while the bowel movements may be only slightly diarrhœal. Other symptoms, excepting a coated tongue and anorexia, may be absent, or the spleen may be enlarged, while the patient may be extremely languid and may suffer headaches and vomiting. At times icterus may occur. After a period of from eight days to three weeks, during which a remittent fever usually persists, recovery sets in by lysis. The resemblance to real typhoid is often great and only the continued absence of the Widal reaction and the negative bacterial findings, make differential diagnosis possible.

Dysentery-like enteritis (*Enterocolitis*).—The most important causative factors of infectious intestinal disease, with a predominance of colitic symptoms, are the *streptococcus*, derived probably from the inflamed udder of the cow (the *streptococcus enteritis* of Escherich) and a species of the colon group (*coli colitis*). There are occasional cases in which other organisms are found. This dysentery-like disease is rather common during the first year and has been called follicular enteritis. Its onset is acute, with moderate or high fever, general symptoms of a serious nature and severe mucous, bloody or purulent diarrhœa. The mucous quality of the evacuations and the tenesmus accompanying them, show that the seat of the trouble is in the large bowel. In favorable cases, the fever disappears in from one to five days and recovery begins. Other and numerous cases are seen which take a different course. At times, cholera-like conditions arise, or an attempt at recovery is incomplete and the mucous diarrhœa continues. Sometimes we see intermittent periods of aggravation with recurrence of all symptoms. Again, with the long continuance of muco-sanguineous evacuations, a severe cachexia may gradually develop, which may end fatally with the picture of extreme atrophy. In still other cases the local disease spreads with unusual rapidity and produces severe, necrotic inflammatory lesions which in some cases run their course with a high remitting fever.

The anatomic findings of the disease are those of a sero-hemorrhagic or seropurulent hemorrhagic inflammation of the lymph follicles of the large intestine. The follicles may become eroded and thus lead to the formation of small ulcers. In serious forms real dysenteric changes in the mucous membrane, of greater or less area, are seen.

DYSENTERY

Dysentery has recently appeared in epidemic form in various countries and, as such, has taken its toll among the children of all ages. It is interesting to note that the epidemic incidence of the disease according to statistics follows very closely the incidence of summer diarrhœa of other types. Its morbidity rises gradually in July and drops to an isolated case in the late autumn. For this reason, every increase of intestinal disorder during this period should arouse our suspicion.

Etiologically, it is customary to distinguish two types or forms (1) true dysentery, caused by the Shiga's bacillus and (2) pseudodysentery, caused by the Flexner and Y-bacilli. This etiologic differentiation does not signify that the pseudodysentery is less serious or of shorter duration. In spite of the lower toxicity developed by the Flexner and Y-types in animals, there is no great difference in the clinical picture of the two varieties. Possibly the primary toxic forms of the disease is more grave and the number of the relapses greater with the Shiga type of infection. The demonstration of the causative organism is possible in fresh specimens only and even then not in all of the cases. If the bacteriologic demonstration of the organism is impossible, the agglutination test with the appropriate organism will be of great use. As a rule, the agglutination with dilutions as high as 1:400 is obtained even after recovery and will give the differential diagnosis. In infants the agglutination may be delayed or fail entirely. The incubation period is short, usually from five to seven days. The onset, generally without prodromes or with a day or so of non-characteristic diarrhœa, is sudden.

It is convenient to distinguish two forms according to the course the disease takes. (1) A form in which the infectious symptoms of diarrhœa and fever predominate, and (2) the form in which the toxic symptoms are paramount. The extreme dehydration and tendency to collapse simulate cholera. This type is less frequent but much more serious than the first. Among the infectious type of cases, a large number of mild attacks are seen. In these the fever may last for only a few days, the consciousness is not affected and the diarrhœa and symptoms of intestinal irritation are slight. In more severe cases the fever persists, diarrhœa, tenesmus and pain is very distressing and continues for a long time. The severe cases are characterized by extreme intestinal manifestations. There is vomiting, anorexia and prostration. These cases are the transitional stages to the cholera-like forms, in which the vomiting and terrific purgation form the clinical picture. In these the temperature, mildly febrile for a short time, becomes subnormal in collapse and the patient presents all the signs of a general intoxication. The tendency to collapse and subnormal temperature distinguishes this form from those cases of the first group in which there is high fever, restlessness, delirium and even convulsions but which soon become convalescent.

Very mild cases are also encountered. In these the temperature is never very high and the bloody mucous stools persist for only a day or two.

Some of these, however, go on to a chronic stage in which the stools always contain some mucus and from time to time contain a little blood. On the basis of this persisting disturbance, acute relapses may occur at any time.

These chronic recurring cases are especially common in weak infants. Dysentery in infants has several other characteristics; the cases with acute cholera-like course are common, and a large part of them have gastric symptoms. On the other hand, the very mild cases, usually rather persistent and with colitis, may resemble the more common chronic dyspepsia so closely as to be completely overlooked and thus form a source of danger to other infants.

In the course of dysentery in children, slight rises of temperature (100° to 101°) may continue for several days. Goeppert finds recurrences in about fifteen per cent. of the cases. In true dysentery, these are said to be especially severe. Undoubtedly this second attack—as in scarlet fever—is a result of the action of the dysentery toxin. Complications and sequelæ, such as conjunctivitis and arthritis, are much less common than in adults. Edema, however, is very common especially in undernourished infants. After the dysentery is completely cured, there may still remain a certain sensitiveness of the digestive tract which will require careful regulation of the diet for months.

Pathologic changes do not differ from those in the adult. The prognosis varies according to the epidemic and age of the patient. The mortality among children of one to two years is twenty-five to thirty per cent. In older children it is lower.

A special danger of the infectious gastro-intestinal diseases lies in their tendency to complications. The most important of these are nephritis and pneumonia, pyemia, cystopyelitis, the more variable pyodermias, and a general septicemia arising from the mucosa of the diseased intestine. More important than all of these is the association of a secondary disturbance of nutrition with the infective disease. It may be readily understood that the

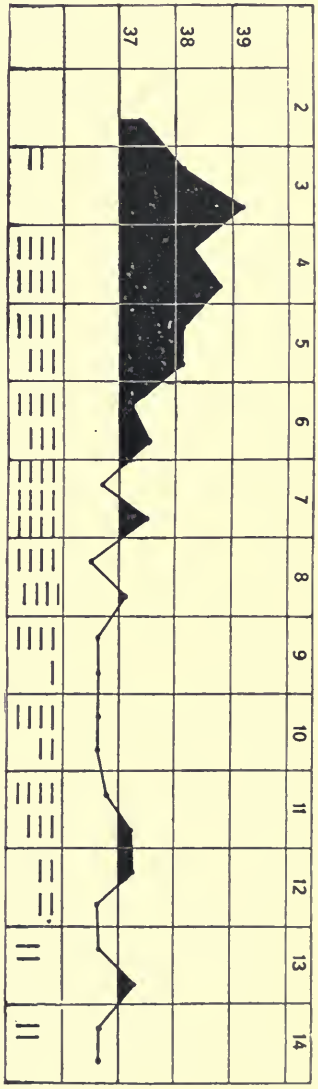


Fig. 93.—Clinical chart of moderate case of dysentery in child of twelve. Recovery. Attention is called to the slight rises of temperature after convalescence has set in. ⅂ = bloody watery stool, | = normal stool.

normal metabolic functions of the intestine do not take place in the seriously diseased organ and that acid fermentation, which causes dyspepsia, occurs easily. This secondary dyspepsia may go on to alimentary decomposition and alimentary intoxication, which will overshadow the original disease, and may carry the patient to the point of hazard. Inanition, due to the customary carbohydrate feeding, may add its menace. It may hardly be doubted that not only numerous cholera-like aggravations, but also a larger percentage of resultant atrophic conditions, are not due to infection, but to starvation and to other forms of secondary disturbance of nutrition. Very probably, a certain number of the severe ulcerative type, with its various complications, are due to the same cause. Underfeeding alone will weaken the total resistance of the body, reduce the general defense against bacteria and permit a local infection to spread unimpeded.

The diagnosis of intestinal infections in general offers no great difficulty. It is important, first of all, to differentiate them from alimentary intoxication and to establish definitely the importance, of the part played by a

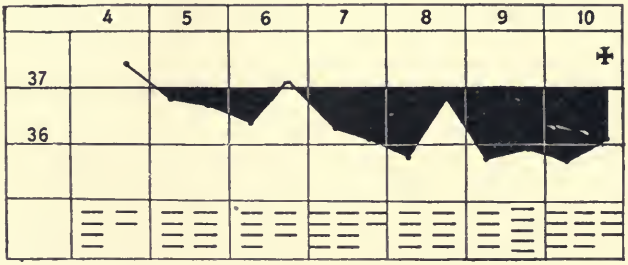


FIG. 94.—Chart of a case of severe dysentery in a child of four. Fatal termination. Subnormal temperature.

complicating dyspepsia or a toxic disturbance of nutrition. The results of a discontinuance of the food supply give a great deal of information. Experience shows that in the infant a diagnosis of the milder dyspepsia-like forms causes some difficulty. The obstinacy of the diarrhœa, in spite of the usual treatment for dyspepsia, should arouse suspicion. If fever does not disappear when the food is withdrawn, a final decision must be made in favor of infection.

The etiologic factors can be determined only by careful bacteriologic examination of the feces and of the blood together with agglutination tests. The prognosis of the various forms of gastro-enteritis in older children is generally favorable. In dysentery and cholera, it is of course, characteristic of those diseases. In younger children, the prognosis is dependent upon the ability of the physician to apply proper dietetic measures. If he is successful in avoiding a serious secondary disturbance of nutrition, which is possible to-day in a great number of cases, he will have a surprisingly large percentage of recoveries; if he fails, his statistics will be far from gratifying.

Treatment should begin by emptying the intestine [calomel 0.05-0.1 gm. (grs. 1-2), in three or four doses]. In the dysenteric forms, one-fourth to one-half of a teaspoonful of castor oil, every two hours, for twenty-four to

thirty-six hours, is recommended. The subsequent treatment is chiefly a question of diet. The general concensus of opinion among authorities is favorable to flour feeding; with the addition of Liebig's malt soup as signs of improvement appear, and with a gradual and careful return to a mixed diet. The procedure should be practically the same as that advised for dyspepsia. Usually the condition of the stools is made to govern the qualitative and quantitative increase of food. If this is followed out too carefully, the patient is usually underfed for rather long periods and, as has already been said, this is extremely hazardous. More recent experiences show that the increase of the food need not be very gradual. In fact, much better results are obtained, especially in children in the second and third years in whom the infectious symptoms predominate, if liberal amounts of food are given. The only precaution necessary is to see that the food is easily digested, and that it will not leave undigestible portions that may irritate the colon. Milk, sugar, flour, prepared cereals, finely chopped meat, egg, broth and fruit juices may be given in quantities large enough to prevent loss of weight. Coarse food should not be given until the stool has been normal for several weeks. In infants, too, the food requirement must be fulfilled as soon as possible. These younger children should be treated as prescribed for chronic dyspepsia and decomposition. Protein-milk or its various substitutes are of great value.

In the cases with the severe cholera-like course, on the contrary, the replacement of the water lost by the severe dehydration must be the first consideration. For this purpose some authors advise the use of diluted whey (1 : 1 oatmeal gruel) in gradually increasing amounts according to age. Better still is buttermilk, at first without carbohydrate additions. Subcutaneous or intraperitoneal injection of physiologic salt solution or proctoclysis may be necessary. The persistent vomiting is to be combated by the use of gastric lavage, or medicinally, by atropin, novocaine, etc. As soon as the case has improved sufficiently to permit the ingestion of food, the régime described for intoxication should be instituted in young infants. In older children, the food should be given in liberal amounts as soon as possible

In the way of medication, good results are obtained early in the disease with opium [0.001-0.02 gm. ($\frac{1}{60}$-$\frac{1}{3}$ gr.) according to age, 3-5 times daily]; later tannigen or tannalbin [up to 2.0 gms. (30 grs.) per day]; quinine tannate [0.1-0.3 gm. (2-5 grs.) three times daily]; lead acetate, [0.003-0.005 gm. ($\frac{1}{20}$-$\frac{1}{12}$ gr.) three times daily]; etc., may be used. In the later stages, those who still believe in irrigation may use lavage of the large intestine with a solution of aluminum acetate (1-500), albargin (1-1000), physiologic salt solution, or bismuth salicylate (1-8 per cent.) in gruel. To this should be added counterirritant measures and hydrotherapy to quiet abdominal pains and restlessness and to reduce high temperature.

The excreta should be disinfected in the customary way. It is well to use materials for diapers and bed-pads that can be burned immediately. Nurses and others in contact with the patient should be instructed as to the danger to which they themselves and the community are exposed in the

spread of infection; since adults may contract either dysentery or dysentery-like diarrhœa. In groups of cases, the source of the infection (food, water, germ-carriers) must be investigated.

INTESTINAL TUBERCULOSIS

Secondary tuberculous disease of the intestine is usually brought on by swallowing tubercle bacilli which have come from the lung. It is as frequent in children of every age as it is in later life. Primary intestinal tuberculosis in which the intestine is the original and the only seat of the disease is much less common. Great difference of opinion obtains among pathologists as to the frequency of the primary forms; nevertheless, the occurrence of primary pulmonary tuberculosis more than doubles the highest estimate of the intestinal form. The younger the child, the less frequent is the primary intestinal infection; in infancy only a few cases are recorded.

The food is, in many cases, the source of a primary infection of the bowel in others, it is a question of dirt infection. Whether animal tuberculosis

FIG. 95.—Rigidity of the abdominal muscles seen in tuberculous stenosis. Child of two years.

from the use of the meat, and especially from the use of the milk of tuberculous cattle, may infect the child is a question of special interest. In all probability, it is a minor matter as compared with the transmission of human tuberculosis.

Pathologic examination shows that intestinal tuberculosis begins with small tubercles, which break down and form ulcers with undermined edges. By the confluence of these, larger loss of substance occurs, around which new tubercles arise. The intestine is frequently covered with circular ulcers. In the immediate neighborhood, peritoneal adhesions are formed and infection extends to the mesenteric lymph nodes, (*q. v.*). Frequently these ulcers cicatrize and thus give rise to stenosis. Kinks of the intestine may be caused by peritoneal adhesions. The most important changes are seen in the small intestine and in the cæcum, while the colon is little, if at all affected.

Symptoms.—Early in the illness the child is fretful, tires readily, has irregular fever, and soon develops diarrhœa and abdominal pains. The

intestine is sensitive at points to pressure; the abdomen is usually little, if at all, distended. In the stools one may find mucus and, by microscopic examination, blood. The disease is of long duration, is marked, in severe cases, by extreme cachexia and hectic fever; and usually terminates fatally. Remissions may, however, occur and even a complete recovery is possible. Scar tissue, intestinal stenosis due to the contraction of scar tissue, compression of the lumen of the bowel caused by adhesions or kinks, may remain and demand special treatment (Fig. 95). Complications by way of the perforation of ulcers with accompanying acute peritonitis, bleeding from eroded intestinal vessels, tuberculous peritonitis, general miliary tuberculosis and tuberculous meningitis may be noted.

The diagnosis is not always easy. It turns upon the long continued fever, the obstinate diarrhœa, the synchronous presence of other symptoms of tuberculosis, and the demonstration of the bacillus by sedimentation methods. A positive von Pirquet reaction does not prove definitely that the symptoms are due to intestinal tuberculosis, for it may be produced by other latent foci.

In the treatment, one must strive to counteract the extreme cachexia, and to prevent, by suitable diet, the occurrence of secondary fermentation dyspepsia. For the rest, general measures (climatic treatment, etc.), which give any promise of results, may be adopted. Of medicinal remedies, the heavy metals are recommended. Bismuth salicylate or subgallate [0.5-1.0 gm. (7-15 grs.), several times daily], lead acetate, [0.003-0.005 gm. ($\frac{1}{20}$-$\frac{1}{10}$ gr.) for several days], ferric pyrophosphate, ($1\frac{1}{2}$ per cent. solution, a teaspoonful, twice a day), may be tried. Opium and the vegetable astringents may be used.

INTESTINAL POLYPOSIS

Symptoms.—In children, the passage of several drops or even of larger quantities of fresh blood from the anus, usually with the bowel movement, but more rarely alone, has been frequently observed. Barring the readily recognized bleeding, caused in the presence of fissures by the passage of a hard stool, and the very rare bleeding from hemorrhoids and malignant tumors, such blood comes from small benign tumors of the rectum. They are papillomatous in structure and occur in three varieties. The so-called rectal polypus, which has a pedicle and grows to about the size of a cherry, is one form. This may appear at times in the anus. The oozing of blood from one or more points of an apparently unchanged mucous membrane, which, upon closer examination, shows small wart-like hyperplasiæ, constitutes another type. In the third and more severe class of cases, a real intestinal polyposis exists; the entire lower portion of the intestine, or even the entire bowel being covered with innumerable and relatively large wart-like polypoid tumors.

For absolute diagnosis, digital examination is only occasionally satisfactory, because the tumors are small and very soft and, therefore cannot be recognized by touch. One should make use of a rectoscope of small calibre.

Single tumors are harmless. The true polyposis, on the other hand, may

cause a dangerous anemia because of the constant loss of blood. This may even occur when the loss of blood is very small. Major cases often take a course similar to that of chronic colitis, in the later stages of which severe cachexia and edema may appear in addition to the anemia. In these cases, the prognosis is very unfavorable.

The treatment consists in the removal of the polypi with the scissors or snare under a local anesthetic. Bleeding from the smaller tubercles may be controlled by the use of caustics or corrosives, trichloracetic acid, or hydrogen peroxide. In intestinal polyposis the repeated use of both methods may be successful. When a large portion of the colon is affected and to a high point, very little can be expected from treatment.

NERVOUS GASTRO-INTESTINAL DISEASES

Congenital Spastic Pyloric Stenosis

Hypertrophic Stenosis of the Pylorus.—The most noticeable symptom of this condition is violent, persistent vomiting, accompanied in severe cases by pain, spasmic deglutition and choking. While symptoms usually begin during the second or third week of life, they may develop earlier or later and may not occur before the third or fourth month. The vomitus does not contain bile and is usually extremely sour, giving a strong reaction for free hydrochloric acid. The patients usually show either no gain in weight, or a gradually progressive emaciation. They pass small amounts of urine and are obstinately constipated. The abdomen is retracted, but prominent at the epigastrium. Epigastric rigidity is observed and marked peristaltic waves, passing from left to right (Fig. 96), suggest the conclusion that the emptying of the organ is obstructed. As a matter of confirmation one may find, upon careful palpation, a small, movable, cylindrical mass at the right border of the rectus under the liver.

Pathologic Anatomy.—At autopsy, the tumor above described is found to be the thickened pylorus, surrounded by a hypertrophied musculature and the hypertrophied pars pylorica, firmly contracted to form a hard, almost cartilaginous, tumor-like mass, several centimetres long (Fig. 97). This contracture causes a stenosis of the stomach exit, which accounts for the clinical findings. Much greater pressure is necessary to force this contracture than is required in the normal systolic stomach at autopsy. Rare cases of "simple pylorospasm" are also seen. These present all the clinical symptoms of a hypertrophy, but show no pathology at autopsy.

A positive explanation of the anatomic findings has not been given. Most observers are of the opinion that it is a condition of primary pylorospasm with a secondary compensatory muscular hypertrophy. The actual cause of the disease is still under very active discussion. It is doubtless a neurosis which, according to the history, is dependent upon a hereditary nervous tendency which produces first pylorospasms and latterly hyperæsthesia, hyperkinesis, and probably, also, hypersecretion.

The disease is found especially in breast-fed infants and even in those

who have not suffered from any errors in the feeding technic. It has often been observed in successive children of the same family. In a certain percentage of cases, one sees ptotic conditions, not only of the stomach but of the remaining abdominal organs, and a general muscular atony is demonstrable.

The diagnosis is readily made because of the early appearance of the disorder and its typical symptoms. The simple nonhypertrophic pylorospasm is much more liable to lead to confusion. In rare cases a similar picture is produced by congenital stenosis, by the pressure of a ptotic liver upon the duodenum, and by compression from peritoneal adhesions, etc. The absence of bile from the vomited matter contraindicates stenosis in the lower part of the duodenum.

The course and termination of the disease are dependent upon its severity, which, in turn, is governed by the completeness of the stenosis and the persistence of vomiting. Mild cases may be recognized by the fact that the loss of weight is relatively gradual. The weight may be even stationary.

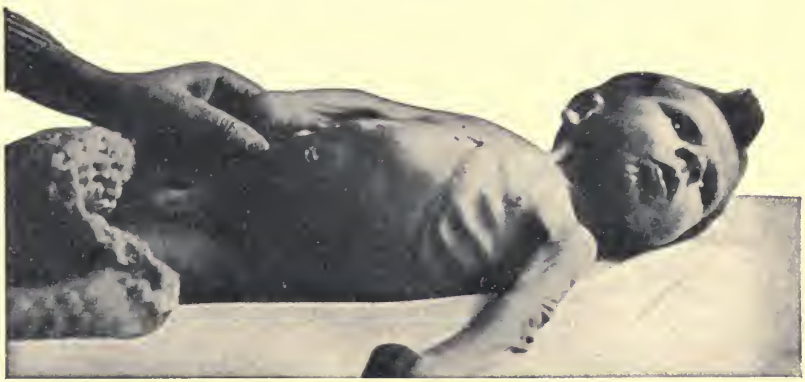


FIG. 96.—Gastric peristalsis in spastic plyoric stenosis. (University, Heidelberg, Prof. Feer.)

A bowel movement occurs at least every second day. In these conditions, one may be certain of recovery. After several weeks, at least after two or three months or more, the vomiting becomes less frequent and the bowel movements more numerous and, with the increasing possibility of adequate feeding, a rapid increase of weight takes place and complete recovery finally follows. Nor do these children show any particular tendency to gastric disorders in later life. In the more serious cases, the loss of weight is rapid from the beginning, the bowel movements occur at long intervals, and the large daily losses of weight do not cease even when the remission of the spasm permits of a temporary increase of the food supply. Usually these patients soon reach a peculiar state of apathy, eliminate sugar in the urine, upon taking even small quantities of food, and simply cannot be fed. The condition is nothing more than one of extreme inanition. At this stage, it is absolutely impossible to save the patient; excepting, per chance, by operation.

In the treatment of pylorospasm, the parents must be warned first against the customary attempt to quiet the stomach by the withdrawal of

food, or the substitution of tea or gruels. Starvation gives no results, and the risk of injuring the young and poorly resistant child by a period of hunger, and of converting a mild case into a serious one, is very great.

No method of feeding is known which is certain to relieve the vomiting. The best plan is to feed breast-milk, for with this the danger of a disturbance of nutrition complicating the inanition is, to say the least, reduced to a minimum.

Various methods of feeding are advised by various physicians. Heubner advocates regular three-hour feedings, permitting the child to ingest as much as it wishes without considering the vomiting. Ibrahim does not put the infant to the breast at once, but gives very small quantities of expressed breast-milk, cooled in ice, at frequent feedings; allowing at first, 10 c.c. every hour; then, with correspondingly increasing intermissions, 15,

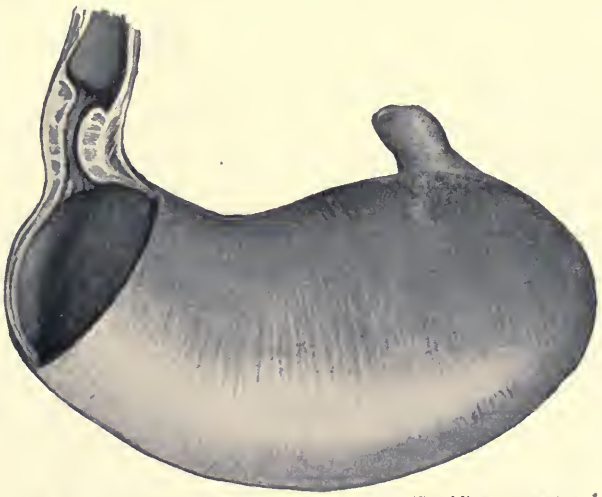


Fig. 97.—Stomach in spastic pyloric stenosis. (Semidiagrammatic.)

20, 25 c.c., and so on. With the ingestion of 300 c.c. the danger of death from starvation is over. Of methods of artificial feeding, the fat-free preparations (buttermilk, skim-milk), are entitled to first consideration, because of the experience with them in simple pylorospasm; but their successful use, if there be any, is by no means so well understood in this form of disease. Sometimes results are attained with concentrated food. There are no contraindications to this A tablespoonful of Karlsbad water before each meal is useful. Hot applications for periods of two hours, three times a day, may be directed. Medication is of very little use. Tincture of opium, $\frac{1}{20}$-$\frac{1}{10}$ drop to the dose, cocaine, novocaine, alypin ($\frac{1}{10}$ of gr. to the dose, shortly before feeding), anesthesin (3 per cent. in mucilage acacia; one teaspoonful, before feeding) and similar remedies may be tried. Stomach washing is recommended by some and opposed by others. To counteract the tendency to the undue loss of water, injections of physiologic saline solution may be used.

Enteroclysis may well be substituted for hypodermoclysis by means of a long tube or thin Nelaton catheter, held in place by adhesive plaster. The following solution may be used: sodium chloride 7.0, potassium chloride 0.1, calcium chloride 0.2, water 1000 grams. It is possible, with care, to give the enteroclysis twice a day for two hours each time, at the rate of 30 drops per minute; in this way the patient receives upwards of 400 c.c. (18 ounces), of fluid per day. In some cases this seems to have a directly favorable influence upon the vomiting. Used for so short a time, the tube will hardly cause decubitus.

As the stomach retains larger quantities, it is advisable to increase the food very gradually at first. Some children are so severely injured by the long inanition that, upon receiving large amounts of food too early, they become the victims of a severe disturbance of nutrition, which takes the form of an alimentary intoxication which might have been avoided by better care.

Recently, very good results have been obtained by passing a catheter into the duodenum, according to the method of Alfred Hess. The long Nelaton tube is carefully pushed through the stomach until it passes the pylorus. This can be recognized by the fact that, upon moving it gently to and fro, the resistance is greater than it is in the stomach. Food is slowly introduced through the catheter, the procedure being carried out several times daily. Frequently an increase in weight begins almost immediately and usually the condition so improves that in a short time progress may be made without the tube.

If duodenal catheterization is not successful and the child loses weight rapidly, surgical interference must be considered. The best and most rapid method seems to be longitudinal incision of the hypertrophic pylorus leaving the mucous membrane intact without suturing (Ramstedt). It is difficult at present to state the indications for operation definitely. This course seems advisable when the tissue loss approaches one-third of the previous body-weight. When rapid weight-loss is progressive it may be better to operate earlier, because with so severe an inanition a serious weakening of the organism is quickly threatened and a longer period of waiting may make the possibility of repair doubtful. Children who have passed into the comatose condition above described are lost under any circumstance.

HABITUAL AND UNCONTROLLABLE VOMITING OF INFANTS

Simple Pylorospasm

Many infants habitually vomit large or small quantities of food. If this is a means of getting rid of an excess, it can hardly be called a disease; it must be considered as such, however, when it continues in spite of carefully regulated or scant quantities of food. This vomiting may occur without any preceding gastric disorder. In many cases, the habit associates itself with dyspepsia and continues for a long time after recovery from that disease. As a rule, an accompanying mild ischochymia (gastric dilatation), is demonstrable.

In some cases, the vomiting becomes so severe that inanition results even to so serious a degree as in hypertrophic stenosis. The peristaltic waves, however, are not seen, there is no pyloric tumor and the constipation is not so obstinate. On the contrary, mucous watery stools are usual, in spite of the small quantities of nourishment that pass into the bowel.

This condition, like the hypertrophic form, is probably dependent upon an abnormal nervous irritability. Whether this is merely a hyperæsthesia of the mucous membrane, or whether pylorospasm is coexistent, is a moot question. Hypertrophy of the pyloric musculature is not found at autopsy.

The prognosis is favorable under suitable treatment; if errors are made the patient may die of starvation. Two or three months of time is necessary to complete the recovery.

Treatment.—Those cases which develop under artificial feeding are quite certain to be cured with breast-milk; but, as in the hypertrophic form, considerable time may be necessary before improvement is noticeable. Usually results are more rapidly obtained by giving a fat-free diet (skim-milk or buttermilk), with the necessary carbohydrate additions. Alkali waters (Karlsbad), are sometimes useful. Gastric lavage is not certainly helpful. Mild cases may sometimes be favorably influenced by the use of milk treated with a coagulating ferment, *e. g.*, pegnin, according to the method of V. Dungern. For young infants concentrated food, and with older children, a solid diet is often beneficial. In addition to remedies noted for the hypertrophic form, sodium citrate (2 per cent. solution, one tablespoonful, before each feeding), and protargol (0.2 per cent. solution, one teaspoonful, before feeding), are also recommended. Often the vomiting has disappeared under the use of fat-free food. Two or three months are usually necessary before an ordinary diet can be employed without recurrences of vomiting.

THE PERIODIC VOMITING OF OLDER CHILDREN

This term applies to repeated paroxysms of vomiting, rarely lasting over a few days or a week, accompanied, as a rule, by fever and other disturbances of digestion. Very frequently, indeed almost constantly, acetonemia, acetonuria and an acetone odor to the breath are noted. Milder forms closely resemble dyspepsia, with constipation and a tendency to vomiting. It is questionable whether cases have been observed in early infancy. In the second year, however, the condition has been known. Between the ages of four and eight years it occurs commonly; with puberty, the predisposition ceases.

Each attack begins with premonitory symptoms; such as change in disposition, gastric symptoms or diarrhœa; then, the vomiting begins suddenly and is repeated at short intervals. Nothing will stop it and the child is apparently in a seriously exhausted state. A strong acetone odor is noticeable in the urine and in the expired air: hence, the French phrase "*Vomissement incoercibles avec acetonœmie.*" Constipation is usual; icterus is sometimes present; temperature is usually slightly increased. After continuing unabated for a varying period, the attack passes suddenly; the

patient makes a rapid recovery, to suffer, after a week or a month or more, another attack. The exact nature of the condition is not understood. It rests, undoubtedly, upon a constitutionally nervous quality. This is shown by the frequent occurrence of cases among the well-to-do, by the usual presence of other neuropathic symptoms, and by the occasional beneficial effects of suggestive treatment. Some authors class the disease as a form of hysteria, but this does not add much to its etiology. It is probably a question of crisis in the metabolic functions, but the significance of the anomalies of metabolism which have been observed is not yet clear. Hecker believes it to be a disturbance of intermediate fat metabolism. The recent observation that attacks are often brought on by the withdrawal of carbohydrates from the food, tends in the same direction. At many points a relationship to migraine is indicated.

In the first attack, great care should be taken in diagnosis, for peritonitis, appendicitis, or meningeal disease may have a similar onset. A history of previous attacks is of value in diagnosis, but even this may be misleading, for brain diseases may cause recurrent attacks due to variations of intracranial pressure. The prognosis of the individual attack is good. Probably a few deaths have been wrongfully ascribed to this condition. In treatment, ordinary methods, as hot applications, small quantities of iced drinks, narcotics [chloroform water, cocaine, or better still, novocaine or alypin 3-5 milligrams ($\frac{1}{20}$-$\frac{1}{10}$ gr.) etc.], may be tried. Water should be supplied by rectum or subcutaneously. Suggestive methods may avail, a subcutaneous injection of sterile water possibly having such an effect. During intervals between the attacks the neuropathic tendency should be treated by hygienic, dietetic and pedagogic methods.

NERVOUS VOMITING

In many sensitive and constitutionally nervous children, an habitual vomiting, due to external influences, may appear from time to time; this does not create any constitutional disturbance but is most disagreeable. Excitement may be a cause, as in the well-known vomiting of school children, occurring on their way to school in the morning. In others, a peculiar sensitiveness of the pharyngeal reflex, responsive to certain sensations in the pharynx, has probably some relation. Again, the cause is sometimes purely psychic, being produced by an abnormal repugnance to certain foods. If the vomiting is repeated often enough, it probably becomes a reflex habit which may appear responsively to a feeling of dislike or even, in extreme cases, to pleasurable excitement. Very frequently children vomit at will, knowing that it will have an impressive effect upon spectators.

Common as this condition is, great care must be taken in diagnosis; all other possibilities, as gastric diseases and especially brain diseases, being considered. Many a case of brain tubercle has gone on for months under a diagnosis of harmless nervous vomiting, until other symptoms have indicated the actual condition. Disturbances of vision (strabismus, astigmatism), may now and then cause the vomiting, which will disappear after their correction. The general condition of the child should be considered

first. If it is not entirely well, otherwise, great care should be exercised. The treatment should be general and directed to the nervous cause. Skilful educational influence, energetic verbal suggestion, coupled with such occasional suggestive treatment as faradization, plaster applications, etc., may bring about recovery in a short time.

NERVOUS ANOREXIA

Loss of appetite, to which no other cause revealed in the pathologic findings of the digestive apparatus can be assigned, and reason for which must be sought in the neurotic field, is commonly called nervous anorexia. In only a part of such cases is a general neuropathic constitution an adequate cause.

These cases vary widely in character and a careful analysis of conditions is necessary. Frequently, the sufficiently well-fed appearance of children, who are brought to the physician because of loss of appetite, proves that no anomaly is present, but rather that the parents expect the children to eat too much. In other instances, children eat little because they are forced to drink large quantities of milk, which reduces the appetite for other food. The anorexia disappears so soon as milk is discontinued. A monotonous or an unbalanced dietary may have been given and its substitution by appetizing and varied meals, consisting of green vegetables, coarse bread, fruit, cabbage, etc., produces wonderful results. Such children may take too little exercise or not get enough fresh air. In true nervous anorexia, these causes do not obtain, but there appears to be an actual absence of appetite or of the sensation of hunger, frequently seen even in infancy, when only with great difficulty can enough food be given the infant to maintain growth. In older children, the amount of food taken may be small generally or the appetite may be capricious and certain foods only may be taken; while others, quite similar, are refused. With many of these cases, the general nutritive condition is so good that no noticeable deficiency in the caloric supply can exist. Only exceptional cases occur, in which so strong an aversion to all forms of food exists, that serious inanition results. Doubtless these are always based upon a *status hystericus*.

In a certain percentage of cases, the loss of appetite seems to be connected with a disturbance of mastication and resulting difficulty in eating solid food. At times it appears that certain foods actually produce nausea because they suggest some perverse association.

The probability of a nervous foundation for the condition is indicated by negative gastric findings. A degree of motor insufficiency may be present, or some hypoacidity with slight hypotonia, existing as part of a frequent general ptosis. With the anorexia, it is usually possible to demonstrate other nervous symptoms, *e. g.*, the facialis phenomenon, vasomotor irritability, ready exhaustion and psychopathic tendency, etc. Upon close inquiry into the family history, familial neuropathy and mental inefficiency can usually be established. Such a relation fails to explain, of course, the ultimate processes which actually cause the reduced appetite.

The treatment of those forms of anorexia that are caused by an im-

proper dietary offer no difficulties. A rational and balanced dietary, varied as much as possible, reduction of milk to a minimum, and regulation of the child's general hygiene bring good results. In the true nervous form, these measures are only preparatory to the real treatment, which lies in the exercise of a proper pedagogic influence. If the environment is such as to make it advisable, the most radical, but satisfactory method is to remove the child from its parents and to find for it a home with sensible people and intercourse with normal healthy children. Frequently, a quiet but energetic nurse or governess may accomplish much, if she has such full control that her orders are not countermanded by the parents. Treatment in sanatoria is not recommended, for pedagogic reasons, unless serious psychic disturbance is involved or a severe degree of inanition has resulted from the hysterical refusal of food. Only in the latter event are we justified in attempting forced feeding, entirely unnecessary in other cases and, as a rule, wholly without results.

We cannot expect by any method we employ to recover a normal appetite. We must be satisfied if the child obediently takes the necessary quantities of food without resistance or show of caprice. Gastric treatment, often presented by stomach specialists, is useless. Medicinally, we may use, besides pepsin and orexine tannate, strychnine [*tr. nux vomica*, grams 2-5.0 (15-30 minims), *tr. rhei vin*, grams 20.0 (5 drams); ten drops of this mixture in orange juice twice daily, a short time before meals]. In mild and even in somewhat severe cases spontaneous recovery may be expected at puberty.

OBSTRUCTION OF THE INTESTINAL CANAL

Congenital Intestinal Stenosis

Congenital intestinal occlusion has certain points of election; immediately above or immediately below the papilla of vater, at the duodenojejunal junction, or just above the cæcum. It occurs less frequently in other portions of the small intestine or the colon. The obstruction is caused by a membranous septum or by scar-tissue, or by the existence of long imperforate loops of the intestine. Extensive portions of the intestine may even be absent. Constriction by bands, diverticulæ or compression tumors may occur. At times, multiple obstructions are present, when other external and internal malformations are often observed. The formation of intestinal atresias is probably due to the temporary closure of the intestinal canal, which is normally well-formed and patent between the fifth and tenth week of fetal life, a closure which, in rare or exceptional cases, persists.

The diagnosis of atresia is made upon the occurrence of vomiting immediately after birth, upon the absence of stools containing fecal matter and other symptoms of ileus. Most of these cases die during the first or second week; only occasionally do they live longer. From the nature of the obstruction, it is clear that only in exceptional cases may results be expected from surgical interference.

The prognosis of congenital occlusions of the rectum or the anus is more favorable. Operations have proved successful in about one-third of these cases.

In rare instances, we have to deal, not with complete atresia, but with a reduction of the calibre of the intestine, the symptoms of which resemble those of chronic acquired ileus.

DILATATION AND HYPERTROPHY OF THE COLON

(Hirschsprung's Disease)

This term is applied to a disease that may be demonstrated clinically immediately after birth or, at least, in early infancy, and is characterized by the extreme dilatation and longitudinal extension of all or a part of the colon, with marked hypertrophy of its musculature. The ordinary anatomic findings resulting from intestinal obstruction are not present.

This anomaly may be due to various causes. Possibly a congenital megacolon, a primary congenital malformation comparable to congenital

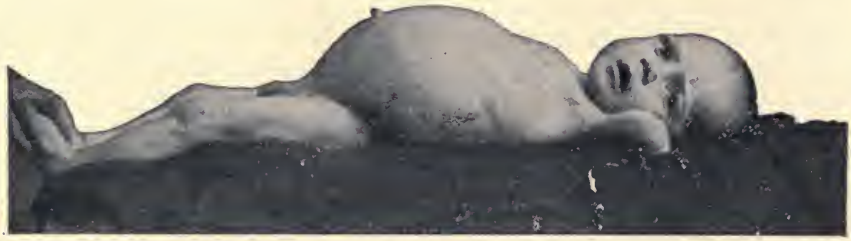


Fig. 98.—Eighteen-month-old boy with dilatation and hypertrophy of colon (Hirschsprung's disease). (Prof. Finklestein.)

dilatation of the esophagus, may exist. This, however, is a very rare condition. More commonly we find obstruction due to a valvular mechanism, which can be demonstrated only by a very careful topographic examination.

Normally in the infant the sigmoid flexure is relatively longer than in the adult. Occasionally it is so long that several loops are formed. It will be readily seen that scybalæ and a large volume of gas may gather in these loops, and cause them to become twisted. If this be long continued or frequently repeated, the loops gradually become dilated and later show compensatory hypertrophy.

In other cases the cause is to be found in the rectum or anus, provoked either by spasm of idiopathic or fissural origin, by a simple coprostasis which has been neglected, or by a paralysis of the lower section of the bowel, with consequent constipation.

Intestinal dilation due to congenital or acquired circular stenosis of the lower section of the colon is differentiated from Hirschsprung's disease by the existence of readily recognized obstruction.

The first indications of the disease, which may appear during the first few days of life, are distention of the abdomen and obstinate constipation (Fig. 98). For a time, these are the most noticeable symptoms. Usually

the circumference of the hugely distended loops of the colon, which contract forcibly from time to time, can be made out. The "dough phenomenon of Gersuny" in the fecal mass can frequently be demonstrated.

The anomaly and its results may be borne without much discomfort for a time, but serious symptoms soon appear. Attacks of ileus are characteristic, with colic, vomiting, and collapse; or, again, we may occasionally have foul-smelling diarrhœic stools containing blood or pus. Death results from peritonitis or exhaustion, incident to the attacks of ileus, and usually in early childhood. But few such children live to be more than ten years old.

Treatment consists in the removal of fecal masses and the prevention of their renewed formation. Enemata are most useful for this purpose, while cathartics are hazardous. Meteorism may be prevented by placing a drainage tube in the rectum, the tube being forced above the valve or kink. With continuous treatment satisfactory results may be obtained.

Fig. 99.—Hirschsprung's disease in an infant.

If this method is not successful, if the attacks of ileus become frequent or if ulcerative colitis occurs, surgical interference may be of assistance.

CONSTIPATION

The diagnosis of purely functional constipation may be made, even in children, only after a careful exclusion of all other possible conditions of disease, which might lead to the retention of feces, (*e. g.*, abdominal or pelvic tumors, paralysis, strictures; constitutional anomalies, such as myxedema, idiocy, etc.). Cases which are not traceable to any of these causes must still be considered from many and various points of view.

The constipation of breast-fed infants is due in some cases to insufficient quantities of food and is relieved by their increase. This is especially true of children who are being fed from a breast yielding but little milk, who are in a condition of actual underfeeding, and who are not gaining, or are even losing weight. Even with normally developing children, whose weight before and after feeding shows that they are receiving sufficient quantities, there may still be a relative insufficiency. In these cases, the milk is so completely absorbed in the upper part of the alimentary canal that there is

no material for fermentation, which causing an acid reaction stimulates peristalsis. These forms of constipation are best relieved by giving thick gruels made of coarse cereals or oatmeal. After the sixth month, the condition indicates the necessity of adding vegetables to the diet. Cathartics or enemata should be used only in cases with a long sigmoid flexure as described below.

The constipation of artifically-fed infants is usually the consequence of a faulty diet. In younger children, exclusive milk feeding plays the most important rôle and a reduction of milk with the addition to the diet of vegetable (flour) foods, or of large quantities of malt soup extract, bring about an improvement. In the second and third year of life too long continued absence of solid food is a large factor.

Constipation of older children is, in many cases, due to an excessive meat diet, which, as in the breast-fed child, gives too little material for fermentation in the colon. A reduction of meat and fish, eggs, cheese and milk, with an increase of vegetable foods, especially in the form of coarse breads, unstrained vegetables containing large amounts of cellulose, fruit, lettuce, etc., or with the feeding of large amounts of fat, will prove useful. As an aid in the production of free catharsis, lemonade, sweetened with large amounts of milk-sugar, and taken cold upon an empty stomach, or sour grape juice, may be recommended.

If results are not secured by these methods, we must consider whether the pressure of the abdominal musculature is properly applied. Very many children and especially young children do not know how to use, while some do not take the trouble to use, their abdominal muscles. It is possible that not infrequently there may be a disturbance of the complicated reflex mechanism of defecation, due to errors in the formation of habit, to fear of pain, etc. It is probable that such a condition exists when it is found, upon rectal examination, that fecal masses accumulates immediately behind the sphincter. Careful education alone can bring certain relief to these children. Many cases of obstinate constipation are cured in a short time by a change of the nurse, by strict prohibition of cathartics and enemata, or by correcting the conscious neglect of the act. Results are sometimes obtained by a change in the surroundings of the child. At first, it is often necessary to help the child by some anti-obstructive measures, such as the old fashioned sulphur treatment (sulphur, milk-sugar, āā ℥ss, once or twice a day), which prevents the formation of hard scybalæ.

In exceptional cases, the cause of difficult defecation is to be found in the existence of fissures, or in the recurrence of painful spasms of the anal-sphincter, upon the removal of which constipation disappears.

The diagnosis of essential obstipation, based upon peculiar anatomic and functional conditions, may be made when all the methods of treatment discussed above have failed. The long sigmoid flexure, already referred to, causing a condition resembling Hirschsprung's disease, comes in for consideration. Again the condition may be but a single manifestation of a general ptosis, expressed in the atony of the colon. It may be due to a relative insufficiency of the motor mechanisms of the bowel, as a result

of which variations of motility occur. In these cases only, are we justified in using massage, oil, and cathartics. Of the latter, the milder infusions, [frangula 15.0 grams (½ ounce), boiled with 250 c. c. (8 ounces) water, for fifteen minutes], or tamarind, aloin pills, and rhubarb are used. Treatment for the improvement of the general health and exercise of the abdominal muscles, etc., should be employed.

INTUSSUSCEPTION

While other forms of intestinal occlusion (volvulus, strangulation obstruction by adhesions or by compression), occur during childhood, they are relatively rare as compared with intussusception. About one-half of the cases are recorded in infancy, one-fourth in the first fourteen years of life, and the other fourth in older persons.

Intussusception is the involution of a portion of the intestine, the intussusceptum, into a lower part, the intussuscipiens. The mesenteric attachment and its vessels are dragged into the layers. There are four common forms, ileal, ileocæcal, ileocolic and colic; the second form, the involution of the ileum into the cæcum, is by far the most frequent.

The mechanism of intussusception is as follows: a portion of the intestine, in which there is very active peristalsis, forces its way into a portion in which the peristalsis is inhibited. This may occur physiologically. The condition becomes pathologic when the upper part is imprisoned and subsequent changes occur in it and its mesentery. It is not easy to determine either the cause of the reduced peristalsis in the lower segment, or of the failure of its physiologic return. In some cases, traumata, play a part; in others, it may be a question of local meteorism or of peculiarities in the mesenteric attachment. More appreciable causes are the presence of a polyp, of an epiploic appendage, or a foreign body attached to the entering point of intussusceptum. No satisfactory explanation has been found for the frequency of the condition in childhood. The suggestion of an excessive peristalsis is not very satisfying. In a certain number of rarer cases, the invaginated portion does not show any signs of injury for quite a long time. Usually, however, the obstruction of the mesenteric vessels produces venous congestion, edema, hemorrhage and, later, gangrene of the intussusceptum, with local or general peritonitis or general septic infection.

Extremely acute cases, ending fatally on the first or second day, are seen. An acute or subacute course, of from two to seven or even fourteen days duration, is more common. The chronic forms are rare.

Symptoms.—The symptoms of the acute type are as follows: Sudden onset of pain, frequently of a very severe nature; vomiting, first of gastric and, later, of intestinal contents; mucous and bloody diarrhœa, which often occurs with severe tenesmus, but which may be absent if the lesion is in the upper portion of the intestine. Sometimes the general condition is only slightly disturbed for a time, while, in others, serious shock occurs at the very beginning. It is usually the increased peristalsis and painful hardening of the involved segment which attracts attention to the obstruction; while meteorism may not at first be prominent. Occasionally, free passage of the

bowels is observed, but bowel movements and the exit of gas are usually suspended. In three-fourths of the cases the intussusception itself may be palpated as a U-shaped sausage-like tumor. Commonly it is found on the left side but in case of invagination of the small intestine it may be discovered in other parts of the abdomen. Extreme distention of the abdomen may make the examination difficult and, because of the pain, an anesthetic may be necessary. At times, the intussusceptum may be palpated in the rectum or may even protrude.

Course.—The younger the child, the more rapid the course of the disease. About one-half of the cases in infancy die during the first three days and four-fifths during the first week. The mortality of untreated cases is eighty per cent. The causes of death are shock, ileus, peritonitis, and sepsis of intestinal origin. Spontaneous recovery, by sloughing of the invagi-

FIG. 100.—Intestinal tumor showing through intestinal wall in intussusception.

nated portion of the intestine occurs in two per cent. of the cases in children in the first year of life, and in six per cent. between the second and fifth year. In later life it is more common. Even this event does not always escape severe symptoms. Many patients suffer from serious peritonitis or septic thrombosis during or after the sloughing, and girdle strictures may remain and subsequently produce ileus. After recovery, whether spontaneous or by interference, relapses are not infrequent.

The symptoms of the very rare chronic cases are: palpable tumor, pain and the signs of the partial obstruction of the bowel. The anatomic basis of this sequel is an involution resulting in comparatively mild changes in the intestine and in the mesenteric vessels.

Diagnosis.—With careful consideration of all the symptoms, a diagnosis is usually made readily. Difficulties may be encountered in its differentiation from hemorrhagic gastro-enteritis and from the severe intestinal hemorrhages which begin with the colic-like pains of purpura. In rare cases, seemingly typical symptoms of intussusception may be due to an unsus-

pected volvulus of the cæcum or sigmoid. If blood-stained stools are absent and the tumor lies at McBurney's point, appendicitis must be considered, a disease, however, which is much less frequent than intussusception during the first two years of life.

Treatment.—The non-surgical treatment of intussusception consists in a reduction by massage or by dilation with air or water. The first method is indicated in cases where the tumor is palpable. It must be done very gently as in the replacement of a hernia. The alternative methods are useful only when the lesion is in the lower segment of the bowel. Any of them must be employed early to give results. Hirschsprung reports over seventy per cent. of recoveries in cases treated within twenty-four hours and only thirty per cent. in those in which the condition had existed longer. An objection to these methods lies in the operator's inability to control the apparent results; the disappearance of the tumor may be due to a change in position. Moreover recurrences are common and there is grave danger of rupture. Under any circumstances, these methods, safely done only under an anesthetic, should be tried but once, and that upon the operating table; so that if absolutely certain restitution has not been obtained, a laparotomy may be performed immediately. If the replacement has been successful, the patient must be very carefully watched for a reappearance of the tumor or of the spastic intestine. The results of prompt surgical interference within the first twenty-four hours are very satisfactory.

PROLAPSE OF THE RECTUM

Symptoms.—Prolapse of the lower bowel segments resembles intussusception very closely. It is an evagination rather than an invagination. It may include only the mucous membrane or a greater or smaller portion of the rectum itself (Fig. 101).

Even in children otherwise well, anal prolapse may be caused by excessive dilatation of the sphincter with large scybalæ and under excessive pressure of the abdominal muscles. Rectal prolapse, on the contrary, is always caused by atony of the perineal muscles, as it is found in congenital ptosis or in spinal paralysis (as in spina bifida), or it may be acquired in the course of chronic disturbances of nutrition.

While in anal prolapse only the mucous membrane is extruded, in severe cases of rectal prolapse a conical mass, 10-14 cm. (4-6 inches) long, swollen and congested because of the compressed veins, may be found. The mass is hyperemic and bleeds readily; it is usually covered with pus and may be ulcerated. At its apex, the lumen of the bowel is visible. By the application of cold and by the use of gauze, the mass is readily replaced but usually reappears immediately or after a short time.

Treatment.—While the prolapse itself is not dangerous, it offers a port of entry for local and general septic infection and for this reason it should be removed as soon as possible. The bowels should be regulated so as to avoid constipation or to relieve existing diarrhœa. Further extrusion should be prevented if possible, for this purpose adhesive strips being applied so as to compress the buttocks, renewing these after each bowel movement.

As an alternative the cylindrical balloon pessaries, resting upon an external plate, may be tried. In milder cases, treatment with astringents, the mucous membrane being painted with a one per cent. solution of silver nitrate, or enemata or suppositories of tannin or aluminum acetate being employed, may give relief. These remedies have little or no effect upon severer cases. In these, surgical treatment should be resorted to; as, for instance, by imbedding a fine silver wire, according to the method of Thiersch, or, as recently recommended, implanting a ring of fascia from the thigh. The protrusion however is often forced through the ring. Lately the injection of two pillars of paraffin into the perirectal tissue, or their insertion by means of a trocar, has been suggested. The repeated injection of alcohol into the surrounding tissues for the purpose of shrinkage may also be

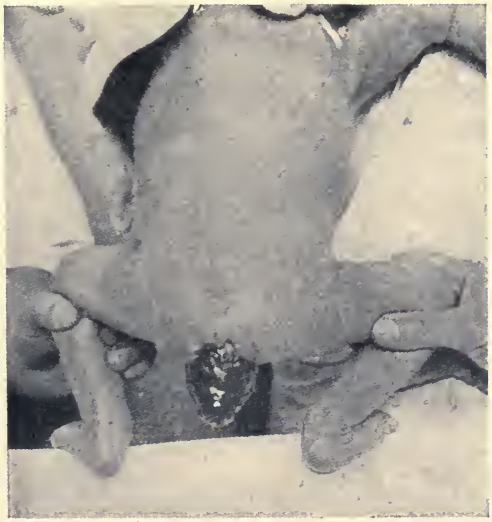


FIG. 101.—Prolapse of rectum in eighteen-month-old boy.

mentioned. All of these are doubtful methods, including that of amputation, and appear unnecessary, if it is possible to improve the general condition and to relieve the atony of the perineal musculature by proper diet. The case shown in Fig. 101 was treated in this manner and the prolapse completely disappeared within three months, the child gaining rapidly after recovery.

HERNIAS

Congenital inguinal hernia, or inguinal hernia appearing shortly after birth, is very common, especially in boys, because the inguinal canal has not closed or its closure is incomplete, the vaginal process of the peritoneum persisting for a long time. Accordingly, the sac does not lie beside the testicle as in acquired hernia, but the intestine and testicle are enclosed in the same sac of peritoneum, unless the lower part of the vaginal process

is practically obliterated (funicular vaginal hernia). By the second year, the number of inguinal herniæ decreases. Typical acquired herniæ are comparatively rare. The sac containing the intestine may, in girls, include also the ovary. Strangulation is comparatively rare in children.

The greater percentage of these ruptures may be cured by bandaging. Instead of the usual truss, long strands of soft yarn passing around the thigh and pelvis and knotted over the ring, are practical in very small infants. Only after the first year, when the rupture, failing to heal, becomes larger, is a radical operation indicated.

Umbilical hernia, or hernia through the linea alba above the umbilicus, hardly ever becomes large. Strangulations are exceptional. Careful bandaging, begun early and applied continuously, usually results in complete closure during the first six months or, at least, reduces the opening so that the intestine may no longer pass. A flat button, not a round one, sewed to a bandage of webbing or held in place by a strip of adhesive, may be used. The best results, however, are obtained by the use of several strips of adhesive, overlapping like shingles, which hold the hernia back under two longitudinal folds of skin over the umbilicus. This should be renewed whenever it becomes loose.

Diaphragmatic Hernia.—The passage of the intestine into the thoracic cavity through congenital openings in the diaphragm, which may be very large, is rare. In exceptional cases, the hernia is so great that only the liver and stomach remain in the abdominal cavity. The hernia is usually found on the left side, because the liver forms a barrier on the right. The condition may be present at birth or may occur immediately after birth. The symptoms are those of severe asphyxia, occurring paroxysmally, and in many cases resulting in death soon after birth. Tympany and absence of respiratory sounds, are noted on the affected side of the thorax and cardiac dulness disappears or is pushed to the right. The retraction of the abdomen is characteristic. In some cases, the condition gives no premonitory symptoms but develops suddenly with evidences of internal strangulation. In the new-born, nothing is to be hoped for from treatment; in older children only very extensive operations, involving resection of ribs, replacement of the hernial content and closure of the opening, are successful.

ENTOZOA

Ascaris Lumbricoides (round worms, resembling the angleworm).—The female may be 30 to 40 cm. (12-16 inches) and the male 20-25 cm. (8-10 inches) in length. They may be found singly or in very large numbers, in the small intestine, where they deposit their eggs. The eggs, which are oval, of double contour and surrounded by a gelatinous, fringed, rough capsule are found in the stools. The infection occurs by means of food, or by contact with dirt which contains the eggs. The diagnosis is made when a worm is passed or by the presence of ova in the stools, demonstrated by the microscope. Symptoms, such as abdominal pain, nausea, irritation

and unhealthy appearance of the skin are cited, but are by no means certainly present. In rare instances, large ball-like masses of entangled worms may produce symptoms of ileus, or the worms may reach the stomach and be vomited. They have been known to reach the larynx and cause asphyxia. A purulent cholangitis due to the irritation of the ascaris in the bile-duct, has been demonstrated. In treatment, the anthelmintic in general use is santonin, given twice daily 0.25 gm. (1/6-1/12 gr.), for two days, in conjunction with a cathartic (calomel or castor oil). Larger doses of santonin may produce poisoning.

The oxyuris vermicularis (pin-worms or thread worms), also inhabit the small intestine, and resemble a fine white thread in color and appearance. The male is 3-4 cm. (1-1½ inches) long and the female, 8-12 cm. (3-5 inches). The sexually mature female passes into the large intestine or out of the body and deposits oval eggs. The infection is carried by dirt adhering to the fingers or by food. From the feces, the eggs are again carried to the mouth by the fingers and with the food into the intestine, which is the cause of the obstinacy of the infection. Itching around the anus is the most important symptom. This may become very severe whenever a new group of worms passes into the colon. The diagnosis is established by the finding of worms in the stools. When worms are suspected, inspection of the anus, the passage of a catheter into the bowel, or an enema will produce the worms more readily than an examination of the stools. The ova are found more readily by microscopic examination of the material scraped from the skin surrounding the anus than in the feces.

The worms are removed by the use of cathartics and enemata, the best results being obtained with a five days' treatment. The first day, with a light diet, a cathartic is given in the afternoon, followed in an hour by a soap enema; the second day, no food is given and santonin is administered two or three times at two hour intervals, with again a cathartic in the afternoon; on the third to the fifth day, a warm daily bath and soap enemata, twice daily, are given. During this time and afterwards, reinfection must be prevented by cleanliness of the hands, finger-nails and anus. The stools should be disinfected. Naphthalin treatment 0.3-0.4 grams (6 grs.), three or four times daily, for two days, repeated in two weeks, is recommended. Fats must be avoided during treatment. The other members of the family should be examined and, if necessary, treated. Usually, there are several persons infected in the same family, which makes the results of treatment of only one member doubtful.

Tenia saginata is the most common of tape-worms. The treatment of the infection is the same as in adults, with fluid extract of aspidium 0.5 c.c. (5 minims) for each year of age, up to 4.0 c.c. (1 dram) with equal parts of powdered senna leaves and powdered tamarind bark. Older children may be given the remedy in gelatin capsules. Rest in bed is advantageous in all treatment of tape-worm and it is well to rest the intestine after complete removal of the parasite.

DISEASES OF THE LIVER

Catarrhal Jaundice

The most common disease of the liver in childhood, catarrhal icterus, usually occurs in epidemic groups and in children over two years of age. It is hardly ever seen in infants. This gives some indication of the etiology, of which nothing further is known. The disease usually has an acute onset, with slight or high fever and occasionally gastro-intestinal symptoms. The jaundice appears after the second or third day with coincident changes in the urine and white acholic stools. The liver and spleen may be swollen and itching of the skin may be noted. In older children, a reduction of the pulse-rate is demonstrable. The tongue is coated and the appetite poor. The course is, as a rule, short. The fever disappears after several days and within one to two weeks complete recovery is made. Only exceptionally, and for some unknown reason, does the icterus continue. Rare cases with cholemic symptoms and acute atrophy of the liver have been observed.

Treatment.—The diet forms the most important part of the treatment. Considering the absence of bile, it is customary to give food as free from fat as possible; that is, a diet consisting chiefly of carbohydrates (flour soup, rice, gruels, etc.). Skim-milk or buttermilk, vegetables, fruit stewed or raw, and finely divided, may be added. Protein is preferably given in the form of skim-milk or cheese, rather than as meat and fish. The usual constipation should be overcome by mild cathartics (rhubarb, or Rochelle salts). Mild laxative teas and aperient waters may be tried. If the case is of long duration, high enemata of Karlsbad water may be advised. Small doses of calomel, combined with podophyllin [calomel 0.0025-0.005 gram ($\frac{1}{20}$-$\frac{1}{10}$ gr.); podophyllin 0.005-0.01 gram ($\frac{1}{10}$-$\frac{1}{5}$ gr.) two or three times a day] is also very useful. To increase the appetite, various bitter tonics may be given [*tr. rhei*; 20.0 grams (4 drams) with *tr. nucis vomicæ*, 5.0 grams (1 dram) ten to twenty drops, of this mixture two or three times daily].

Other Forms of Acute Jaundice

Other, and rarer forms of acute icterus occur in childhood. Weil's disease, which is probably due to infection with a member of the proteus group, and occurs with severe general symptoms, high fever, tendency to hemorrhage, and nephritis, is one of these. Acute yellow atrophy, liver abscess with sepsis, appendicitis, gastro-enteritis and hepatitis, developing in the course of general septic disease, are complicated with jaundice. Gall-stone disease is probably extremely rare.

Cirrhosis of the Liver

Syphilitic Cirrhosis.—Cirrhosis of the liver is most common in infants during the first months of life. It may be congenital and, in this event, is doubtless always caused by syphilis. We distinguish three main forms. The most common is the diffuse portal cirrhosis, which arises from a general portal infiltration, and is chiefly characterized by marked swelling and

hardening, with or without slight icterus, and enlargement of the spleen. This goes on to granulation and shrinkage of the liver tissue, with ascites.

The knotty, gummatous form is also seen; and finally, the icteric cirrhosis, probably due to a gummatous cholangitis, and leading to marked enlargement and icterus. All of these forms occur also in later life, and although actually rare, are still relatively more frequent than any other types of cirrhosis.

Of these other forms, occurring even in two and three-year-old children, alcoholic cirrhosis has been seen: also, cirrhosis, with tuberculous peritonitis and rare cases of the hypertrophic icteric cirrhosis of Hanot, of uncertain origin, have been reported. Congestive cirrhosis with enlargement of the liver and spleen, with ascites, clinically distinguished from other forms by a marked general cyanosis, is somewhat more frequent. The cause of the congestive feature, as of its cyanotic result, is often, but not always, of a pericardial or tuberculous nature. This relationship to the changes seen in the liver is not yet clearly understood.

In addition to simple cirrhosis of the liver due to pericarditis, the type of lardaceous liver, characterized by hard cartilage-like deposits in the connective tissue, may, at times, develop under similar circumstances.

Cirrhosis of the liver rarely occurs in connection with general infectious diseases (*e. g.*, measles or scarlet fever). In tropical climates, an icteric cirrhosis, in connection with malaria and a biliary cirrhosis of unknown etiology (the so-called infantile liver), have been observed.

Tumors of the Liver

Among various tumors of the liver, the very malignant, partially diffused, infiltrating, and semi-tuberous carcinoma and sarcoma, must be mentioned. They are frequently secondary to the kidney or adrenal affection. Of the rare primary forms, the greater part may be traced to misplaced germ cells. Operation is impossible. In diagnosis they must be differentiated from gummata.

Cystic tumors of the liver are congenital or, at least, may be traced to a congenital matrix. They appear either as solitary tumors or, more commonly, as multiple cysts in the liver and may reach a great size. Finally the echinococcus cyst has been observed in childhood.

Congenital Obstruction of the Bile Ducts

Congenital obstruction of the large bile passages is a rare disease. It has its origin in a malformation, consequent upon an extensive or total separation of the germ centre of the liver from that of the intestine, as a result of which the bile passages are either rudimentary and imperforate or are entirely absent. As a result of obstruction to the flow of bile, a biliary cirrhosis is formed by distention of the bile passages and hypertrophy of the connective tissue. The malformation may exist in varying degree. Its most serious form is the complete absence of all the bile passages; the slightest and the most important for practical purposes is a simple occlusion of the opening of the bile-duct into the intestine. The disease is in no way related to syphilis.

Symptoms.—Children with this malformation are born with icterus, or become icteric after a very few days. The discoloration gradually reaches its highest possible degree. In the meantime, the liver and spleen become large and hard. The urinary findings correspond to the discoloration of the skin. (The stools are acholic from birth.)

Course.—The disease ends fatally during the ninth or tenth month, at the very latest (commonly before the fourth month), often with cholemic symptoms and those of a hemorrhagic diathesis. The possibility of forming a connection between the biliary system and the bowel obtains only in cases of atresia of the papilla, which, of course, cannot be diagnosed, and are rare as compared with the incurable forms.

In older children, serious and long sustained cases of icterus, due to diseases of the bile passages, such as congenital stenosis and cystic development, in particular, occur.

DISEASES OF THE PERITONEUM

Purulent Peritonitis

Purulent Peritonitis of the New-born.—The greatest prevalence of peritoneal disease is found in the new-born, and corresponds with the large percentage of other septic infections common to this age. Infections extending from the umbilical vessels are most common. Next in frequency are the metastatic infections and, finally, infections arising in the thoracic organs. Peritonitis in infancy from other causes, and especially from perforation, is rare. The diagnosis is very difficult at this age because the characteristic symptoms are not very definite and meteorism, vomiting and sudden loss of weight, so commonly depend upon other causes than peritonitis. The recognition of the disease is of much less importance in infants than in older children, because the age of the patient and the nature of the disorder forbid operative interference.

Peritonitis in Older Children Following Appendicitis

Appendicitis is extremely rare in the first year of life and is observed only exceptionally during the second year. From this time on, however, it rapidly becomes more frequent, together with secondary peritonitis incident to it. To one acquainted with the disease in adults, its manifestations in children do not present any peculiarities. The diagnosis is made difficult, however, by the uncertainty of the subjective symptoms, and by the frequency of other intestinal disturbances, which tend to lead the diagnostician in other directions. In the differential diagnosis we must consider, aside from acute intestinal catarrh and gastric conditions with painful colic, the following facts. In children, with separation of the recti and other signs of ptosis, attacks of severe abdominal pain occasionally occur and are relievable by properly applied bandages or adhesive straps. In their onset, diseases of the respiratory passages, and especially pneumonia, run an initial course attended by pain, which is frequently localized

by the child in the lower abdomen. Similarly, we sometimes find in influenza and other general infections, areas of hyperæsthesia and *zones of head* in this region. In childhood, an habitual torsion of the movable cæcum is common and is characterized by tumor, pain, and occasional diarrhœa of blood-stained stools. In chronic fermentative dyspepsia, also (*q. v.*), severe pains and local tympany are observed.

Other Forms of Purulent Peritonitis in Older Children

Those forms of peritonitis which do not arise from the appendix are comparatively unimportant in older children, when we consider that those cases which may be regarded as purely peritonitic give hope of remedy from surgical interference. In this group, are included peritonitis from perforation of a typhoid, tuberculous, duodenal or ventricular ulcer; peritonitis due to transmigration in enteritis and ileus; and peritonitis following the strangulation of a hernia. The majority of cases observed belong, however, to a group arising from pulmonary and pleuritic infections, or to the type of metastatic peritonites, in so far as these appear as primary localizations of an infection, or even as an apparently primary disease.

Peritonitis due to infection with pneumococcus occurs as a phase of a multiple purulent inflammation of the serous membranes (Heubner's disease), or polyserositis, polyorrhomenitis, affecting the pleura, meninges and joints, which is seen quite frequently, even in infants. In older children it is of greater individual importance and is, therefore, a direct object of diagnosis and therapy. The pneumococcus peritonitis of older children, chiefly affecting girls, although no definite infection from the genitals can be traced, may be regarded as a migratory peritonitis derived from the intestine or from the pleura. Probably most cases might show a hematogenous origin from some primary focus, as, for instance, an angina.

The disease begins suddenly with high fever, vomiting, diarrhœa and severe abdominal pains. After several days the condition improves but the diarrhœa continues, and gradually signs develop in the abdomen which point to the presence of an exudate. Distention, dulness and possible fluctuation are observed. The dulness is usually not that of a general exudation, but rather resembles that of a large walled-off abscess filled with thin pus, containing pneumococci. Left alone, it gradually extrudes through the umbilicus and finally ruptures it. Alternatives of rupture into the genitals, the colon or the urinary bladder may occur. A diffuse, purulent peritonitis with movable exudate is more rare than the encapsulated form.

The diagnosis is difficult. In the beginning, appendicitis is usually suspected; differential signs, *viz.*, severe diarrhœa, localization of pain, and absence of muscular rigidity, on the one hand, are hardly constant, and, on the other, scarcely serve as a sufficient basis for differentiation. In its distinction from typhoid, the severe pain and violent vomiting at the outset and the absence of leucopenia are valuable. In the later stages, the appearance of an exudate is distinctive. With the large quantity of exudate eventually present, tuberculous peritonitis might be suspected, but the

acute course and a bacteriologic examination of the pus, obtained by exploratory puncture, should rule this out.

Streptococcus peritonitis similarly caused, and with symptoms similar to those described above, excepting that the tendency to encapsulation is wanting, and that its course is much more acute and more malignant. In bacterial diagnosis, not only the pus, but the venous blood obtained by puncture may be used.

Gonococcus Peritonitis.—In girls showing a gonorrheal discharge circumscribed pelvic peritoneal inflammations are occasionally seen. These occur with symptoms similar to those met in adults. Exceptionally the inflammatory process continues for weeks with high fever and extends over the whole abdomen. This is distinguished from other forms of peritonitis by the relatively slight effect it has upon the general condition. A provisional diagnosis is based upon the presence of vulvo-vaginitis and upon the demonstration, by rectal examination, of a major genital disease. It should be remembered, however, that a child with vaginal discharge may have a coincidental non-gonorrhœal peritonitis.

The disease is relatively benign. Even the diffuse form is in most cases self-limited. Cases with fatal termination, however, are known.

The treatment of purulent peritonitis in childhood, and especially its operative treatment, is founded upon the rules applicable to adults. In general, the earliest possible opening of the abdomen is indicated. Only in gonorrhœal peritonitis may spontaneous recovery be expected. In the pneumococcus peritonitis it is of advantage to operate after the abscess has been encapsulated. This advice, however, which can hardly be followed in cases with severe symptoms and of uncertain etiology.

TUBERCULOUS DISEASES OF THE PERITONEUM

Tuberculosis of the Mesenteric and Retroperitoneal Lymph Nodes

As tuberculous infection of any locality produces tuberculosis of the regional lymph nodes, so those of the mesentery and of the retroperitoneal spaces may become diseased from a tuberculous lesion in the intestinal mucosa, which may be completely healed while the secondary process in the nodes continues and spreads. The nodes enlarge and caseate, and extensive confluent lymph tumors are formed by adhesive inflammation. The mesentery and omentum may become knotted and adherent and knob-like granulation tumors, resembling a string of beads, may develop in the intestinal serosa and in the mesentery. Many of these forms are produced by bacilli of the bovine type. It is then possible to palpate multiple, cylindrical and nodular masses of marked resistance in the abdomen. The abdomen is distended (Fig. 102), and usually the spleen is greatly enlarged. The process is insidious beginning with indefinite pains and later giving high and often hectic fever. Nutrition is seriously affected and cachectic edema is common. These general manifestations may continue for a long time before findings on palpation are possible. The prognosis is unfavorable; but recovery occasionally takes place even in severe cases.

The Adhesive Form of Tuberculous Peritonitis.—This greatly resembles the form just described. It has its origin in a tuberculous infection of the peritoneum, which gradually develops from crops of miliary tubercles to large masses of diffuse, scattered, caseous, granular masses, binding loops of intestine together by adhesions. Finally, the intestinal loops become adherent to each other and to the parietal peritoneum in an inextricable mass. The caseous masses between the intestinal loops may break down and rupture into the bowel, into the pelvic organs, or even externally. The abdominal cavity may become infected with pus organisms, when a septic and ichorous infection and general intoxication are added to the tuberculosis.

Symptoms.—Clinically, the disease begins with languor and indefinite symptoms, such as slight abdominal pains and occasional vomiting. With these there may be a slight temperature. The abdomen gradually becomes irregularly distended and is hard to palpate. Percussion and palpation lead to the suspicion of tumors. There is no free ascites. As the disease

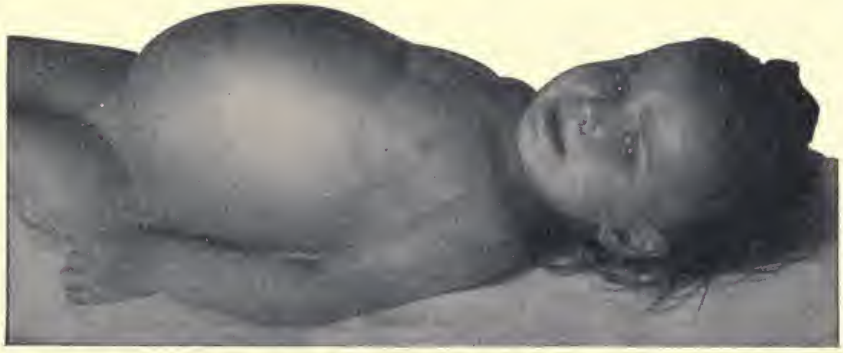


Fig. 102.—Tuberculous peritonitis. (Gisela Children's Hospital, Munich, Prof. Ibrahim.)

goes on the fever continues; and diarrhœa and anorexia considerably reduce the weight. Occasionally symptoms of partial intestinal obstruction occur. The situation becomes hopeless if the softening tubercles become infected or rupture. The prognosis is the same as in abdominal tuberculous adenitis.

The Exudative Form of Tuberculous Peritonitis.—This condition results from a crop of miliary or sub-miliary tubercles, which do not caseate but remain in the stage of granulation and degenerate. The tubercles are accompanied by a serous exudate, of which several litres may be formed. This type, which has a relatively good prognosis, begins insidiously, with slight temperature and mild indications of abdominal disturbance. Later, the abdomen increases in size and the presence of a movable exudate becomes definite. The patient's general health is but slightly affected. Hard, light-colored stools are usual, but diarrhœa may be present.

A large percentage of these cases are amenable to treatment, even though they go on for months and recurrences or exacerbations occur.

The diagnosis of tuberculous diseases of the peritoneum is dependent upon the history and the demonstration of other accompanying symptoms of tuberculosis (lichen scrofulosum, tuberculides, lymph nodes, etc.).

Even if these are not present, tuberculosis is probable, because other peritoneal diseases are more rare in children. The abdominal tumors should not be mistaken for masses of fecal matter. Rectal examination may, at times, reveal enlarged nodes high in the abdominal cavity and small nodular irregularities in the pouch of Douglas. In the presence of large quantities of free exudate, diseases of the liver, heart lesions, and pneumococcus peritonitis must be excluded. In these cases, a bacteriologic and cytologic examination of the fluid obtainable by puncture is necessary. A lymphocytosis is strongly indicative of tuberculosis. Bacteria can be demonstrated only in the centrifuged material or by injection into laboratory animals. Pseudo-ascites, in the course of severe chronic digestive insufficiency, may be recognized by close observation of the digestion, by the variation in its quantity, by the difference in the areas of dulness, which do not resemble those of a free general exudate, and by examination of the fluid obtained by puncture.

In the treatment of all forms of tuberculosis of the peritoneum, the hygienic and climatic factors, indicated in all forms of tuberculosis, should receive first consideration. To these are to be added methods which will increase local absorptions; *i. e.*, heat in all forms (hot applications, electric pads and local electric light baths), and action of the sun's rays upon the abdomen. The exposure to the sun should last only for a few minutes at first and the time should be gradually lengthened. In mountain resorts and at the seaside, these sun-baths may produce surprising cures; while in advanced cases they may cause serious recurrences and even fail entirely. Soft soap treatments and salt may be useful (see scrofula). In many cases the resorption of the exudate seems to be hastened by a salt-free diet. A generous mixed diet should be allowed, but actual overfeeding must be avoided. Diarrhœa should be treated according to the methods described for intestinal catarrh. Medicinally, the treatment is that recommended for scrofula and general tuberculosis. Iodine is especially useful.

The value of surgical treatment has probably been overestimated. Its best results are seen in forms associated with ascites, in which fairly good results may also be obtained by the methods of internal medicine. Nor does operation promise much in other forms. It is of chief use in children for whom a physical, dietetic, and climatic method cannot be applied, because of surrounding influences. Opening of the abdomen and removal of fluid is sufficient; sometimes, simple puncture is effectual.

FETAL PERITONITIS

At autopsies on the still-born or on children who have died immediately after birth, the residue of a fetal, aseptic peritonitis is found in the form of adhesions and bands of connective tissue. Such children, may live and show symptoms of intestinal obstruction from birth. Fetal peritonitis may be the result of a malformation of the intestinal tract or the genital apparatus; of the passage of the fetal intestinal contents through a perforation into the abdominal cavity; or it may be due to the placental trans-

mission of substances producing inflammation. Syphilis may be considered as a causative factor in a number of cases.

Syphilitic peritonitis, with pseudomembranous exudation is no uncommon symptom of the first irruption of the disease in the infant. In exceptional cases, it produces typical symptoms of peritonitis, vomiting, meteorism, and ileus due to scar-formation and kinking.

TUMORS

Among benign tumors of the peritoneum, simple cysts, echinococcus, fetal inclusions and dermoid cysts, may be mentioned; among the malignant, medullary carcinoma and sarcoma. The symptoms of the latter resemble the tumor-forming and exudative tuberculous peritonitis.

IV.

DISEASES OF THE RESPIRATORY ORGANS

BY

C. FRH. VON PIRQUET,

Vienna.

REVISED AND EDITED BY

WALTER H. O. HOFFMANN, M. D.,

Associate Attending Physician Children's Memorial Hospital, Chicago.

DISEASES OF THE NOSE

THE child's nose is not only absolutely, but also relatively, smaller than that of the adult. Its development does not keep pace with that of the cranium, but rather with that of the extremities; and in consequence it is coincidently and characteristically involved in those disturbances of development which may be distinctly demonstrated in the long bones. This delay in development results in a prolonged arrest of growth, or in the perpetuation of a phase resembling the embryonic snub-nose.

The nose remains particularly small in achondroplasia or true dwarfism. A deep saddle lies under the prominent forehead, and beneath this is only a slight nasal prominence. Similarly in myxedema, the nose is affected, together with the rest of the body, but to a less extent than in rickets.

The small nose in congenital lues probably cannot be charged to a delay in the bony growth, but is rather a local process of a chronic inflammation of the nasal mucous membrane and the bony framework of the nose.

In the healthy child, the nose is the exclusive passageway for the respired air. Mouth breathing is always a sign that the nose is not normally patent. Small children do not open the mouth even in very forced respiration. When the auxiliary respiratory muscles of the thorax are called into play, the movements of the *alæ nasi* become visible. When this is associated with an expiratory grunt, it is an almost certain diagnostic sign of pneumonia.

RHINITIS; CORYZA

The nares act as a filter for the respired air. Their moist mucous membrane, covered by ciliated epithelium, serves very effectively for removing particles of dust and soot and other gross physical impurities from the atmosphere. Most of the micro-organisms which appear in the nares are destroyed by the normally protective substances present in the blood circulating through the highly vascular mucosa.

This physiologic filter is not able to resist all invaders, and considering the large volume of air which requires disinfection by the nose, it is not at all strange that the frequency of nasal infection is great. It is therefore easy to see that diseases of this region are usually the first to affect children.

Small primary foci of infection are probably formed in the deeper parts

of the mucosa from which they spread to neighboring areas, whence the entire body is brought into participation in the disease. Since, however, inspection of the entire inner surface of the nose is not possible, the exact mechanism of such infection is not known.

Usually the only visible result of the infection is the secretion from the mucous membrane of an exudate which contains variable proportions of serum, mucous, blood and pus cells. Even the swelling of the mucous membrane is not always directly visible, but is rather inferred from the mouth breathing. The general disturbance which ensues is of varying character and depends upon the particular etiologic factor concerned in the nasal infection.

Variola, in which the primary focus is commonly in the nasal mucosa, may be considered typical of the infectious diseases which find their port of entry in the nose. About fourteen days after the infection, the exanthem appears, associated with severe general symptoms, and is followed by the ultimate formation of pustules. These develop not only upon the skin but also throughout the respiratory tract and particularly in the nose.

The method of infection in measles, a matter of much greater importance, may be theoretically assumed to be after the same fashion. Supposedly, a primary focus always precedes the development of the disease. The first evident disturbance marks the second stage, which is a catarrhal affection of the respiratory mucous membrane and the conjunctiva. The child coughs, sneezes and has a viscid secretion from the eyes. At this stage a diagnosis can not always be made and must necessarily await the appearance of the eruption of the mucous membrane of the mouth. If Koplik's spots are discovered, a diagnosis of the coryza as a prodromal symptom of measles may be made often a day or two before the eruption upon the skin appears, permitting the physician to take precautionary measures at once.

Nasal diphtheria is not a matter of secondary symptoms but rather localization of the micro-organism in the nasal mucosa, with a resulting inflammatory exudate, which in infections of greater intensity leads to the formation of a false membrane. The diphtheria bacilli either invade the nose first or migrate to it from the tonsils. In the latter event the recognition is easy since the characteristic exudate can be seen on the tonsils or on the posterior pharyngeal wall. In cases of isolated nasal infection the condition is not easily recognized unless the membrane is located low in the nares or is expelled by sneezing.

These conditions do not always obtain, and since latent nasal diphtheria is common, it should be a rule to examine every sanguino-purulent discharge from the nose for diphtheria bacilli. Equally, a nasal affection combined with hoarseness should arouse a suspicion of diphtheria. If a membrane, or if diphtheria bacilli are found, an injection of antitoxin should be given at once. In cases in which a fairly well-formed suspicion of diphtheria exists, it is well to give the serum without waiting for a report of the bacteriologic examination. A similar sanguino-purulent nasal discharge occurs in some cases of scarlatina.

The diagnosis in such an event must be made from the exanthem upon the skin and upon the mucous membrane of the oral cavity. Isolated disease of the nose as a result of scarlet fever has not been demonstrated.

The rhinitis of scarlet fever is of great importance in the matter of prognosis, because the cases with severe nasal inflammation are usually serious, and because the local infection is apt to extend to the Eustachian tubes and the middle ear.

A characteristic, chronic affection of the nose is seen in congenital syphilis. A congestion of the nares and excoriation of the surrounding tissues suggests a scrofulous form of tuberculosis as a question of first consideration.

In the new-born a gonococcic infection of the nasal mucosa, coincidently with the characteristic conjunctival blennorrhœa, is observed.

Herewith the types of coryza in which a definite infectious agent can be demonstrated are practically exhausted.

Coryza also occurs in epidemic meningitis and in acute poliomyelitis.

Meningitis probably starts as a primary infection in the nose, and this leads to a metastatic infection of the meninges. However, since the nasal catarrh has no definite characteristics, its specific quality is actually recognized only after the appearance of general symptoms, unless the occurrence of cases of meningitis in the family suggests the examination of the nasal secretion of all its members for the organism of Weichselbaum. The simple microscopic demonstration of an intracellular diplococcus has no significance since other similar bacteria are frequently found in the nose.

Acute poliomyelitis also commonly begins with influenzal symptoms but in this disease again, with our present knowledge, diagnosis is impossible until the typical paralysis appears. When the disease is present in the household, the coincident occurrence of coryza in other members of the family may lead one to suspect it to be a rudimentary form of poliomyelitis (Wickman).

The existence of a group of rather obscure influenzal diseases has to be recognized. In extensive epidemics in which coryza ushers in catarrhal symptoms of the mucous membranes and severe constitutional disturbances and in which the influenza bacillus of Pfeiffer is the cause, an etiologic diagnosis is possibly permissible. In the sporadic cases, however, which are seen almost daily, it is scarcely ever possible to determine the causative organism with any degree of certainty. Probably a large group of microorganisms, including the micrococcus catarrhalis, the pneumococcus, etc., behave in a very similar manner. Since therapeutically reliance is placed upon symptomatic measures, a differential diagnosis has no practical value, and the severe coryzas, accompanied by fever and other general symptoms may be designated as la grippe or influenza without implying that the condition is necessarily caused by the influenza bacillus itself.

In addition to these infectious causes of coryza, there are other influences which in especially predisposed individuals produce acute affections of the nasal mucosa. A typical example is tuberculin coryza. When an individual who is especially sensitive to tuberculin inhales this agent or the

tubercle bacilli in pulverized form, he is subject to coryza which may be considered analogous to the conjunctival tuberculin reaction. Similarly, there are persons who react by an attack of coryza to the inhalation of horse dandruff. An important clinical picture showing this tendency in one form is hay fever.

In some people the inhalation of air laden with the pollen of certain plants produces severe irritation of the respiratory tract and especially of the nasal mucous membrane. Those susceptible suffer with a persistent swelling and hypersecretion of the mucous membranes when these plants are in bloom, an event which occurs chiefly in the early summer and less generally in the fall. That the coryza is excited by the pollen is proved by the fact that in the predisposed individual the manifestations of hay-fever can be induced, even in winter, by the instillation of a suspension of the pollen, while a normal person does not react. It is quite certain that a large proportion of acute cases of coryza in the adult may be charged to hypersensibility to certain foreign substances. In early childhood, however, such hypersensibility probably merits less consideration. Hay fever hardly ever appears before the close of childhood.

In every day life, coryza due to exposure to cold plays an important rôle. Cold in itself cannot be regarded as the cause, since polar explorers remain free from catarrh in spite of the most severe exposure. (Nansen, Shackleton.) Similarly, in the open air department of the hospital, no catarrhal affections could be traced to exposure to cold, although the children slept out of doors, at zero temperature. It must be presumed, however, that the invading organisms more readily find a foothold when in consequence of the cooling of the body, the secretion of the protective fluids do not occur normally.

The pathologic anatomy of a mild coryza shows only a swelling and congestion of the nasal septum and of the lower turbinates. In severe cases, the deeper parts of the mucous membrane are also involved in a mucopurulent inflammation. From here the process may spread to the Eustachian tubes and the middle ear.

The Clinical Symptoms of Acute Rhinitis

The first sign of acute coryza is usually frequent sneezing. Then in a few hours there is a rise of temperature, accompanied by a watery secretion from the nasal mucous membrane. During the next few days the secretion becomes mucous and clouded with pus cells. Finally it becomes more and more purulent and tenacious. At the same time, the swelling of the mucous membrane makes nasal breathing difficult. This becomes most annoying, especially in infancy. The infant when nursing from the breast takes a very firm hold of the nipple and while sucking breathes quietly through the nose. Whenever, then, the nose is partially obstructed, breathing is difficult, and if the obstruction is complete, the nursing must be interrupted repeatedly in order that the child may breathe through the mouth. Then the child loses its patience and its desire to nurse. Occasionally the condition may cause attacks of choking on account of which, in the early

months of life, coryza may lead to alarming symptoms. At other times the respiratory rate is greatly increased and the sleep consequently disturbed because the infant attempts to breathe through the mouth and nose alternately.

As a result of the general infection, fever, and anorexia, the child becomes debilitated, loses weight, and may even die of a complicating bronchitis or broncho-pneumonia.

Until the third or fourth year, a severe coryza must be looked upon as a serious disorder, because of its possible complications, while in later childhood it is usually as readily overcome as it is in adult life.

Acute inflammation of the pharyngeal tonsil (the lymphoid tissue of the nasopharynx) does not necessarily lead to severe symptoms. In children with exudative diatheses, such an inflammation is common during infancy. It is apt to cause fever, malaise, disturbances of respiration but not always a nasal discharge. Very frequently a reddening of the posterior pharyngeal wall is the only finding that will explain some irregularities of temperature in infancy.

An indication of acute inflammation of the nasopharynx is given by the enlargement of the lymph nodes of the posterior cervical triangles, while a swelling of the nodes in the anterior or sublingual triangles is indicative of an inflammatory process in the tonsils.

The therapy of acute rhinitis consists essentially in combating the general disease. If there is a mucopurulent or sanguineous discharge, an examination for diphtheria bacilli should be made. If the suspicion is in the slightest degree justified, antitoxin should be used. The nasal mucous membrane is not very accessible to treatment. The injection of antiseptic fluids is contraindicated on account of the liability to forcing fluid and infected mucous into the middle ear.

An inert or mildly antiseptic ointment containing boric acid or zinc oxide may be used to prevent the excoriation of the skin by the secreted pus. If crusts or dried discharge are formed in the nares, small wooden applicators, such as a match stick or toothpick, mounted with cotton and covered with ointment may be placed in the nostrils for a few minutes, but of course only while the child is under observation. Instead, several drops of warm olive oil or almond oil may be instilled carefully into the nose several times a day. Possibly 1 per cent. of menthol may be added to these. The introduction of pledgets of absorbent cotton saturated with 1:3000 solution of epinephrin causes a temporary reduction of the swelling. If nursing becomes very difficult, the infant must be fed from a spoon. If this cannot be done successfully, it may be necessary to feed the child by gavage. The general therapy, if fever obtains, consists in inducing perspiration by means of hot packs, warm drinks and acetyl salicylic acid (0.1-0.25 gm. at a dose), or quinine (0.05-0.25 gm. to a dose). Very young children should be kept in bed, older ones in the house.

Recently, calcium lactate has been highly recommended (Januschke). One gram a day may be given to infants, while older children may receive five grams a day. This treatment is continued for three or four days.

		Grams.
℞	Calcii lactatis	5.0
	Syrupi rubi idæi	10.0
	Aquae q. s. ad	100.0

The room temperature should be about 18° C. (65° F.) in winter, and the air should be kept as moist as possible. The odor of oil of turpentine or of oil of pine, allowed to evaporate near the bed, is very pleasant.

Chronic Coryza

Subacute chronic inflammations of the nasal mucosa are common in childhood. They result either from acute coryza or as in the case of luetic or scrofulous snuffles, they develop without any acute stage, and with rather an insidious onset. In the chronic form, the centre of the local condition is to be found almost invariably in an enlargement of the lymph organs of the nasopharynx. The mucosa of the nares also shows a chronic swelling and secretes a purulent or mucous discharge.

A peculiar form of chronic coryza is ozæna, in which the submucosa atrophies. The air passage is not obstructed, and there is a small quantity of a tenacious discharge forming crusts over the entire mucous membrane which stagnates the secretion beneath.

Putrefactive bacteria (Perez), undestroyed because of the lack of normal secretion, cause a decomposition of the tissues accompanied by a characteristic and intensely foul odor. The disease occurs occasionally in older children, who in almost all instances present signs of tuberculosis. The therapy of ozæna must be directed chiefly to the improvement of the general health. Locally the instillation of liquid petrolatum containing 1 per cent. of salicylic acid, or the tamponade of the lower nostrils with cotton pledgets saturated with petrolatum has been recommended.

The prognosis of chronic coryza depends largely upon whether it is or is not the expression of a chronic infectious disease. Infants should be carefully examined for syphilis. If the history is negative, a Wassermann test should be made. If the diagnosis is positive, specific treatment should be instituted. To cases occurring later in childhood, the tuberculin test should be applied. If this test is strongly positive, the suspicion that the coryza is of a scrofulous nature is justified. The proper treatment should then be instituted. If the tuberculin test is negative, a prognosis of rapid recovery under suitable management may be given with some degree of certainty.

Children of sufficient age should be made to wash out the nose several times a day with physiologic salt solution or with luke-warm water containing a small quantity of borax. The patient may be given a small portion of boric acid ointment (3 per cent.) with instructions to apply it to the nostrils, one nostril being closed while the ointment is snuffed into the other. The ointment melts at body temperature and spreads over the mucous membrane softens the crusts, and stimulates the flow of secretion. The other nostril is then similarly treated. After ten or fifteen minutes the child must blow its nose thoroughly.

For smaller children, instillations of sweet oil, cotton tampons saturated with boric acid ointment, or insufflations of antiseptic powders may be used.

The best remedy for the last purpose is sodium biborate with 10 per cent. of sodium sozoiodolate and 1 per cent. of menthol. A marked hyperplasia of the mucous membrane must be treated by painting with a 2 per cent. solution of silver nitrate.

Adenoid Vegetations

In association with simple or scrofulous chronic rhinitis, or without any affection of the nasal mucosa, one finds in children very frequently an enlargement of the lymphoid tissues of the posterior pharynx. In children of the lymphatic type the pharyngeal tonsil, together with faucial tonsils and other glandular organs, is often subject to enormous hypertrophy, so that it fills the entire space intended to serve as a passageway for air. On this account the child is forced to breathe through its mouth and in consequence acquires after a time a characteristic facial expression. His speech acquires a peculiar resonance, because the sounds that are normally spoken through the nose (M, N) or those that require a complete closure of the soft palate (P, T, K) are enunciated indistinctly. This leads sometimes to the developing of various types of lisping or stammering. Stuttering is sometimes caused by the difficulty in breathing. Children who snore for want of a free passage of air have bad dreams, nightmares, and pavor nocturnus. Nocturnal enuresis often complicates such cases.

Beside the effect of adenoid vegetations upon the speech and the respiration, another unpleasant result is their occlusion of the Eustachian tube, which in its turn causes a retention of secretion and the development of chronic catarrh of the tubes and the middle ear. The hearing is subsequently affected, and the child is retarded in his school progress.

When adenoid vegetations are very large, they have a further effect upon the nasal skeleton. The development of the bony structure of the nose is delayed, as a consequence of which the eyes protrude, giving an appearance resembling Basedow's disease.

Adenoids may occur even in infancy, but they are most commonly seen between the fourth and the tenth year. Without treatment they may persist for years, but they tend to disappear toward the end of childhood. In many cases enlarged cervical and sublingual lymph nodes occur coincidently and probably result from adenoid vegetations. They usually disappear after the adenoids have been removed.

The diagnosis is readily made upon the evidence of the mouth breathing, the nasal speech, and the history of snoring and deafness. Many physicians are inclined in every case of obstruction of the nose to diagnose the presence of adenoids or polyps. Frequently the obstruction is due to a narrowness of the nasal passage, which is acutely exaggerated by intumescence of the turbinates. The diagnosis is confirmed by directly palpating the vegetations.

For this purpose the child is placed in front of the physician, but facing away from him. He is then told to open his mouth, the head being held from behind with the left hand pressing the cheeks between the teeth, so that the child cannot bite without hurting himself. His hands must be

held by an attendant. The index finger of the right hand is then rapidly introduced into the open mouth, passing back through the fauces and exploring the posterior pharyngeal wall.

Therapy.—If the vegetations are very large and cause much annoyance, they should be removed surgically. Anesthesia is not absolutely necessary. After the operation, the patient should be kept away from other children for several days on account of the danger of infection of the wound surface. If the symptoms produced by the growth are slight, or if coryza or angina is present, the treatment should be expectant. Topical treatment of the adenoid has no effect. The causative scrofulous condition should receive attention.

Foreign Bodies In The Nose

Small children often force small objects such as stones, beans, or seeds into the nostrils. Occasionally a foreign body is forced through the nasopharynx into the nose and becomes lodged in the anterior nares during vomiting. Such bodies may cause local inflammation which creates a foul-smelling, bloody discharge. In other cases these objects become encrusted and are termed rhinoliths.

If the child is observed in the act of forcing the foreign body into its nose, the treatment is usually simple. If the body has become lodged in the nostril, it can often be pressed out, or it may be expelled if the nose is blown while the other nostril is held. In very young children, the Politzer bag may be applied to the open side, a procedure, however, which requires great care.

If the diagnosis cannot be made from the history, a foul-smelling discharge from one nostril continuing for a long period is a quite definite indication of a foreign body. In acute cases diphtheria must be borne in mind. In these cases of impaction the removal of the object is not always easy, since the foreign body may swell, or it may be imbedded in the granulation tissue. Attempts may be made to blow the substance out with air or to wash it out with warm water after reducing the swelling of the mucosa by cocainizing it. If the body lies deep, it is sometimes easier to push it through the posterior nares to the throat. Sometimes it is possible to pull the foreign body out with a fine forceps or with a hooked probe.

Similar symptoms may be produced by polypi, which are very rare in childhood. They must be removed surgically.

Epistaxis

Nose-bleed in children, not occurring as a symptom of hemophilia, hemorrhagic diathesis, leucemia, pertussis, typhoid fever, or a heart lesion, is usually due to injury or is the result of picking the nose. The hemorrhage comes from the anterior portion of the septum. It is readily stopped when it is not a symptom of one of the above named diseases. The child is made to snuff up cold water from the hand. If this does not succeed, the lower nose may be tamponed with a strip of gauze. This gauze may be saturated with hydrogen peroxide. This medication frequently produces a

rapid cessation of the nose-bleed. After the bleeding has stopped, determine whether a chronic inflammation of the nostrils has caused irritation and invited injury. If this is the case, a 1 per cent. ointment of ammoniated mercury may be applied to the nostrils, or the hemorrhagic area on the septum may be touched with a 2 to 5 per cent. solution of silver nitrate.

In early infancy epistaxis suggests some serious condition and should raise the question of sepsis, scurvy, diphtheria or syphilis.

DISEASES OF THE EUSTACHIAN TUBES AND THE MIDDLE EAR

One of the most frequent complications of the diseases of the respiratory organs in childhood is the extension of the inflammatory process to the mucous membrane of the middle ear. Its frequency is due in part to the structural peculiarities of the child's auditory apparatus, and for the remainder, to the general predisposition to infection of the mucous membranes of the young. In the new-born the organ of hearing is not, like that of sight, completely formed, but undergoes its final development in post-natal life.

The chief cause for the extension of the inflammation from the pharynx is that the cartilaginous portion of the Eustachian tube is much shorter than in later life, while at the same time its lumen is decidedly greater. The pharyngeal opening of the tube is very much lower. In the new-born it lies at about the level of the hard palate and in the adult about one centimetre higher. The papillary fold covering the opening is formed toward the end of the first year. At birth the depression is small and lies directly above and behind the opening into the ear. In the fetus the tympanum is filled with a pad of dense embryonal connective tissue, which usually disappears before birth.

The tympanic membrane in the new-born forms a part of the external surface of the cranium. It is obliquely placed and directed downward, whereas in later life it is almost vertical. The membrane itself is usually thicker and more opaque than in older children, but it soon acquires its distinctive delicacy and transparency.

The external auditory canal is short and consists of a soft cleft-like channel without bony structure and cartilaginous only at its outer extremity.

An examination of the tympanic membrane of the new-born must be done with a speculum. A very short and thin-walled one is necessary on account of the shortness of the canal. The examination must be carried out cautiously. Difficulty lies in the fact that the membrane is very obliquely placed. Again, the canal is usually filled with vernix caseosa. To remove this, the canal may be washed out with tepid water to which hydrogen peroxide has been added. For this purpose a fine soft tube is fastened to the tip of a syringe. The most important point of orientation is the short handle of the malleus. The normal membrane of the new-born is gray, but when the child cries, it becomes pink, a change which must not be mistaken for evidence of inflammation. After the third month, inspection is an easier matter and in still later childhood, the picture resembles

that of the adult more and more. An attempt should always be made first to inspect the drum without the speculum. The child will remain quieter when he is not frightened by the insertion of the instrument, and since hairs are absent from the meatus, the membrane can usually be seen quite well in part if not as a whole. In order to make the inspection without the speculum, it is necessary to stretch the canal by lifting the external ear upward and outward with the second and third fingers, while the tragus is pushed forward with the thumb. If the handle of the malleus is seen, the gray color of the membrane distinguished, and the normal reflex appears, the examination is completed. Only when a pathologic condition exists is the speculum employed in order to get a more accurate diagnosis. If it is not merely a question of finding out whether there is a discharge, it is not necessary to use a speculum but instead to introduce peroxide of hydrogen.

The child is laid on the other ear, and three or four drops of 3 per cent. hydrogen peroxide solution are dropped in the external ear while the auditory canal is kept closed. After the solution has had time to get warm, it is permitted to flow slowly into the meatus. The formation of a large amount of foam indicates the presence of pus in the canal; a slight foaming may come from a minute amount of pus or from cerumen present.

Otitis Media Catarrhalis Neonatorum

At autopsies of the new-born or of very young infants, more or less purulent matter is found in the tympanum in a surprisingly large number of cases (40-90 per cent.). Children who die before or during birth always have amniotic fluid or meconium in the middle ear, which is probably pumped into the ears by premature efforts at respiration. If they live for a short time, a collection of leucocytes without bacterial contamination is formed, which may be considered in the nature of a pus formation around a foreign body (Aschoff). A similar affection seems to occur very frequently in the healthy new-born. Gompers found by a systematic examination of infants at birth that in nearly fifty per cent. of them a swelling or bulging or at least a reddening of the tympanic membrane appeared, which continued until the sixth to the eighth day and then subsided to a normal state.

The exudate disappears either by resorption, or it flows off through the tube. It never causes rupture of the membrane. No clinical manifestations have been demonstrated. Therapeutic and prophylactic measures are superfluous.

Acute Otitis Media

The middle ear may be invaded by the most varied infections. The micro-organisms probably reach it, as already suggested, in the majority of instances by way of the Eustachian tube. This is true of the otitis of scarlet fever, of diphtheria, and of the various influenzal diseases. In measles, the infection may be spread through the blood channels, appearing as an exanthem of the middle ear. This route of infection is surely true of certain of the tuberculous lesions.

In small children, the transmission of infection seems to be favored by

repeated vomiting, as well as by the large calibre and shortness of the Eustachian tube (Goeppert). Etiologically, the pneumococcus is the organism most commonly found in the exudate of otitis media. According to Preysing, it is responsible for 92 per cent. of all cases. The streptococcus comes next in frequency and various other bacteria follow. It cannot be said that the bacteriologic findings suggest any distinctive course or any differential prognosis, although in general the pneumococcus seems to be more benign than the streptococcic infection.

The symptoms of acute otitis vary greatly. It may be said that the younger the child, the more frequently is otitis overlooked, because in small children it causes no symptoms at all or but very indistinct ones. In some cases there is no pain at all. With the occurrence of general restlessness, lassitude, sensitiveness, anorexia, and fever, without any other focal symptoms, disease of the middle ear must always be considered.

If the child rubs his head on the pillow and is unable to find a comfortable position in which to go to sleep, and particularly if he puts his hand repeatedly to the head, it may be inferred that the ear is painful.

Pressure over the tragus usually reveals distinct tenderness. The infant prefers to lie on the affected ear if the condition is unilateral, and often prefers to nurse from the opposite breast (Pins). If the pain is severe, the child's appetite completely disappears, and he shows a tendency to vomit and will cry for hours. Convulsions, coma and meningeal symptoms may arise. Continual movements of mastication have been observed.

Other children may localize the pain very definitely, but even with them otitis may be entirely painless, and its existence may be discovered only after the rupture of the membrane. The rupture is caused by the pressure of the exudate and probably occurs more easily and less painfully with the thin delicate membrane; so that the younger the child, the less the discomfort.

The temperature varies within wide limits. It may run over 40° C. (104° F.) for several days, and again its rise may be hardly noticeable. The regional lymph nodes do not become enlarged in all cases. Those situated behind the ear, back of the sternomastoid muscle, and in subacute processes those in front of the tragus are affected.

The duration of an acute attack of otitis is rather variable. It may reach its maximum within twenty-four hours, while in other cases it may go on for several days before rupture takes place. When the rupture occurs, the pain usually disappears. The discharge is at first sanguino-serous, and later becomes mucopurulent, then purulent, and finally mucoid. It may produce an eczema of the external auditory canal and of the auricle. The discharge continues for a variable period. It may disappear after a few days, or it may become a chronic otitis. Perforation, however, is not the only method of termination of an acute otitis. In the majority of cases there is no rupture of the membrane, but spontaneous resorption of the exudate.

The diagnosis of acute otitis is based upon the local tenderness and the discharge from the ear. When rupture has not taken place, the diagnosis depends entirely upon the examination of the ear-drum. If the middle

ear is filled with a catarrhal secretion, the membrane, if thin, shows a change from its normal pearl gray tint to a yellowish-brown color. It is possible, if the cavity is not completely filled, to distinguish a dividing line between the fluid and the air. As soon as the membrane itself becomes inflamed, it takes on a bright color, appearing first around the handle of the malleus and later becoming distinct around the periphery of the membrane. This reddening may be very intense and is sometimes accompanied by small ecchymoses. The landmarks disappear. In very weakly children and especially in atrophic infants, the redness may be lacking, and then the diagnosis must depend upon the dulness and bulging.

Perforation generally takes place in the lower posterior quadrant and because of its small size is not easily recognized. In acute otitis the rupture hardly ever becomes larger than about one millimeter in diameter. It may be possible at times to see a small droplet of pus forming over the opening. The upper half of the tympanic membrane may also rupture, an event especially common in infancy.

The prognosis of acute otitis depends in some degree upon the nature of the disease process and again upon the general condition of the child. The otitis of scarlet fever is particularly to be dreaded because it may lead to the destruction of the auditory ossicles and the infection of the internal ear and of the mastoid. This tendency is seen, however, only in certain epidemics. Generally speaking, the otitis of scarlet fever cannot be distinguished from other forms.

In diphtheria a perforating otitis is rare, but autopsies show that a diphtheritic membrane may extend to the Eustachian tubes and to the middle ear. This type of infection does not cause serious destruction. Measles, during the catarrhal stage, sometimes leads to an otitis of a temporary character. The middle ear inflammation, however, which constitutes one of the secondary infections resulting from measles, must be considered a much more serious matter. This type has a tendency to become chronic.

Inflammation of the middle ear is most frequent as a complication of influenza and la grippe. This form lasts usually from four to eight days, and even though perforation occurs, recovery takes place within eight or ten days. The rupture of the drum commonly heals very rapidly and generally without a scar. The hearing is not particularly affected by suppuration of short duration.

An exception to this rule is to be noted in weak and especially scrofulous children. In these cases perforations show no tendency to heal, and the result is frequently a chronic otitis.

From the standpoint of prognosis, the occurrence of complications is of the utmost importance. The commonest are mastoiditis, subperiosteal abscesses, necroses of the temporal bone, and the extension of the inflammation to the meninges. Prompt attention should be directed to any inflammatory swelling or tenderness behind the ear. If these do not subside within two or three days, surgical procedures must be instituted. A sign of inflammatory invasion of the parts surrounding the middle ear is the rather frequent paresis of the facial nerve. It usually disappears after the suppuration has

subsided. Intensive meningeal symptoms are always cause for alarm. Simple vomiting is not necessarily an important symptom.

There is no rational prophylaxis against otitis. In young children the danger of the infection of the middle ear from within is present in every attack of coryza.

The therapy consists in promoting the action of the skin and of the intestinal tract with a view to modifying the general symptoms. A warm pack, a sweat-bath, hot drinks and sodium salicylate may be given. If the child has not had a free movement of the bowels, active catharsis sometimes has an abortive influence upon middle ear disease. If the patient suffers very severe pain and sleeplessness, small doses of phenacetin (0.05-0.2 gm.) or of veronal in doses of 0.02-0.2 gm. may be given in milk. Occasionally quinine gives good results.

If the tympanic membrane is distinctly reddened and bulging, paracentesis should be performed. The puncture is made with a long knife in the lower posterior quadrant. It is not necessary to anesthetize the membrane although a piece of gauze saturated in a 10 per cent. solution of cocaine may be applied. If the paracentesis is done at the proper time, a bloody or purulent discharge should immediately appear. The cut should be from two to three millimeters long. After the puncture has been made, a strip of iodoform or sterile gauze may be placed loosely in the canal and the dressing held in place by a hood, which will make it easy to change the gauze.

Paracentesis should be done only when there is a definite bulging and severe pain, but not in those numerous cases in which there is an abnormality of the membrane without clinical symptoms, or pain without definite drum abnormalities. Pediatricians, indeed, have almost entirely abandoned this practice in infants, since results are no better with the operation than without it. Even though spontaneous rupture occurs, the time required for healing is no longer than that required after operation, and in many cases the membrane never ruptures.

It is best, therefore, to attempt to control the pain with hot applications. At the same time, warm oil with 1 per cent. of menthol, or glycerin with 5 per cent. of phenol may be run into the ear, or pledgets of cotton saturated with one or the other of these agents may be inserted into the canal.

If perforation has occurred, the ear should be cleaned frequently by irrigation with hot sterile water, after which it must be carefully dried with pledgets of cotton. Insufflation of an antiseptic powder such as boric acid may be tried. If the canal is irritated by the discharge, a 1 per cent. ammoniated mercuric ointment may be applied.

Chronic Otitis Media

A number of cases beginning as acute otitis do not recover, but continue to suppurate for months and even years. The cause of the chronicity lies either in the etiology of the primary inflammation or in the general poor condition which prevents a tendency to healing. For this reason chronic otitis is very frequently seen in children who have signs of tuberculosis or syphilis.

The therapy of chronic otitis consists first in the treatment of the basic condition. If the discharge is tenacious and foul-smelling, the ear may be irrigated with a 1 per cent. solution of hydrogen peroxide, or with boric acid solution, or with a weak solution of potassium permanganate. The simultaneous affections of the nose and nasopharynx (adenoids) should receive attention. Careful examination should be made to see whether the discharge is kept up by the presence of polypi, granulations, or chronic disease of the mastoid.

Foreign Bodies in the External Auditory Canal

Unilateral earache, without coryza, arouses suspicion of a foreign body, which in children is frequently found in the auditory canal. Fruit seeds, flies, etc., becoming impacted in the cerumen, form a hard mass. It is better to soften this mass slowly by repeated irrigation with lukewarm water than to try immediate instrumental extraction.

CONGENITAL STRIDOR

Audible respiration occurs in children from various causes. In obstruction of the nasal passages (coryza) or of the nasopharynx (adenoids), the respiration becomes snuffling, usually associated with mouth-breathing and snoring while asleep. Swelling in the tonsillar region (angina, diphtheria, scarlatina, tonsilitis, retropharyngeal abscess and chronic hyperplasia of the tonsils), causes a snoring respiration while awake, associated with difficulty in swallowing. Noisy respiration is also caused by pathology farther down in the respiratory tract. A mainly expiratory, rattling sound is heard in bronchitis. Bronchial asthma and intumescent bronchial gland tuberculosis cause expiratory wheezing. Mainly inspiratory is the stridor in enlargement of the thymus, the thyroid, and in mediastinal abscess (Rach). The expiratory grunt of pneumonia, the in- and expiratory stridor with hoarseness in diphtheria and pseudocroup, and without hoarseness in edema of the larynx, foreign bodies and stenosis, originate within the larynx itself.

An inspiratory crow with free expiration occurs in pertussis, laryngospasm, and is a congenital affair. In pertussis the crowing occurs only during the coughing attack. Laryngismus stridulus, like the other symptoms of tetany and rickets, never occurs before the third month of life, and then only in attacks and never during sleep. When the history reveals that the inspiratory difficulty has been present since birth and persists during sleep, the case is one of congenital stridor. These children have a mediocre or weak constitution with negative findings in the lungs and other organs. Retraction of the suprasternal notch and epigastrium are seen only exceptionally if the inspiration is especially labored. This noise is caused either by a congenital anomaly of the larynx, *i. e.*, a narrowing of the entrance of the larynx due to a narrowing of the epiglottis and too close approximation of the aryepiglottic folds, or by an insufficient tissue turgor of the larynx (Heubner).

The prognosis of this disease is generally good, although occasionally

deaths from suffocation have occurred. With the growth of the larynx, the noise usually disappears in the first or at the latest the second year of life.

No therapy is known. Occasionally placing the child on the side, or stomach, causes a slight lessening of the stridor.

ACUTE LARYNGITIS (Pseudocroup)

The larynx of children with congenital stridor represents in a way an exaggeration of the normal infantile type of larynx in which the entrance is relatively small. The narrower the entrance, the more easily can a serious stenosis result from a swelling of the mucous membrane. For that reason, there are very often evidences of suffocation in young children with diphtheria (true croup), but even in a common swelling from a simple catarrh, little children can get attacks of suffocation. In the non-diphtheritic laryngitis, a mild and a serious, or a superficial and a deep-seated form can be differentiated. The mild form or simple catarrh consists of a reddening and secretion of the entire mucous membrane of the larynx and leads to hoarseness, barking cough, and a slight fever. The causes are the same as in acute coryza, but hoarseness can also come from breathing overheated or dusty air, as well as from overtaxing the larynx by continuous crying. The illness usually lasts only a few days. It is in itself benign. The danger consists only in a possibility of an increase in the swelling of the mucous membrane, in a transition to the second stage of laryngitis, and in a secondary complication of the lungs. Treatment is unnecessary when there is no fever. Keep the child indoors during cold weather, and possibly order inhalations. With fever, as in acute coryza, give hot tea with small doses of salicylates or quinine, water or oil compress on the chest, as well as turpentine inhalations.

The severe form of superficial laryngitis is known as pseudocroup. Anatomically it is characterized by an intensive swelling of the larynx in addition to redness and secretion, and this leads to severe symptoms of stenosis without formation of a membrane.

The swelling attacks principally the mucous folds which lie at the lower surface of the vocal cords. It occurs especially when the child has been changed from a vertical to a horizontal position, beginning a few hours before going to bed. On laryngoscopic examination thick, parallel, decidedly red swellings can be seen between the vocal cords.

The etiology of pseudocroup is, generally speaking, the same as that of acute coryza, with a special predisposition for the larynx. Just as some adults have with every coryza a completely obstructed nose from the swelling of the turbinates, while others suffer from increased secretion, so many children are inclined to a swelling of the mucous membrane of the larynx, while others from the same cause have a simple hoarseness. Especially susceptible are the children with "adenoids" and strong fat children with exudative diathesis, who are inclined to superficial catarrh.

The attack of pseudocroup runs as follows; the child has been somewhat indisposed for one or two days, with running nose, loss of appetite, some coughing and fever, but so like a common cold that the parents do not

think of calling for medical advice. In the middle of the night, the child awakens with all the signs of suffocating. The expiration is labored, the cough spasmodic and barking, the voice hoarse but not toneless, and inspiration is extremely difficult. There are marked retractions of the supraclavicular regions. When the first anxiety is over, the child breathes somewhat easier, and after several hours the whole acute attack may be over, while the hoarseness remains for several days. On the other hand, the symptoms may increase in severity and in rare instances lead to suffocation.

For the diagnosis and therapy it is of utmost importance in every case of severe dyspnœa, to make certain at once whether or not it is diphtheria. The examination with the laryngoscope is very difficult in small children, and is not completely diagnostic, because the diphtheritic membrane is not always plainly visible. Most of the time one must be satisfied with an inspection of the throat. Every membrane on the tonsils or pharyngeal wall points to a diphtheritic affection of the larynx. Even in an apparently simple angina or bloody discharge from the nose, laryngeal obstruction should be regarded as diphtheritic, and without consideration for the bacteriological findings, antitoxin should be given. The giving of antitoxin can be disregarded only when there is no suspicion of diphtheria.

A sudden appearance of the stenosis, especially in the night, with barking cough, speaks for pseudocroup; diphtheria has an insidious onset. In pseudocroup there is seldom a complete loss of voice, and in spite of the hoarseness, loud tones can be made. Complete loss of voice speaks, therefore for diphtheria. It is important to know that the initial catarrh of measles can cause pseudocroup. The symptoms disappear with the appearance of the eruption which is followed by a reduction in the swelling of the mucous membrane, and therefore has a good prognosis. It is a different matter if the hoarseness and difficulty in breathing begin after the appearance of the eruption; then it is often a secondary diphtheritic infection and is associated with great danger.

The therapy of pseudocroup is to promote abundant diaphoresis; warm tea, lemonade, mineral water with milk are given internally; packs with warm water or warm oil are used externally. Older children can advantageously use steam inhalations. (Bronchitis kettle see Fig. 103.) Small children usually turn their heads away from the steam and the bed must be transformed into a tent by covering it with sheets and the steam introduced from the foot or side. Care should be taken that the infant does not come too near the steam pipe. Burns occur frequently from lack of care. The old physicians used emetics in large doses (antimony tartrate, 0.05 gm. with pulvus ipecacuanhæ 1.0 gr.). In spite of numerous recommendations, this drastic cure has not been widely favored.

Gratifying results have been obtained from hot tea or lemonade with salicylates (sodium salicylate, acetosalicylic acid, etc.), in small doses; or with liquor ammonii anisati 1.0 in water 100.0 with 10.0 syrup, one teaspoonful every two hours, possibly with the addition of codein phosphate 0.02 gms.

In severe dyspnœa, intubation is indicated. For pseudocroup it is

much to be preferred to tracheotomy. A tracheotomy which takes at least fourteen days to heal, leaves a permanent scar, and many times a lasting injury to the voice, is entirely disproportionate to the illness which is over in a few hours. Intubation is very easy in these cases as there is no membrane, and it has no harmful results. After twenty-four hours, the tube should be removed if it has not already been coughed out. It is very seldom necessary to perform an intubation a second time. For the further treatment, the directions given in the chapter on Diphtheria can be applied.

Laryngitis Phlegmonosa.—This deep-seated form of laryngitis is a high grade inflammatory infiltration of the larynx and the upper part of the trachea and extends to the perichondrium, becoming very painful.

Laryngitis phlegmonosa follows measles or scarlet fever. Without these, it is probably due to the same infectious agents as the ordinary coryza and bronchitis, but is very rare. and probably occurs only in children of 2-3 years, following pneumonia.

Laryngitis phlegmonosa begins with a cough, hoarseness, and fever. These symptoms increase after several days, and in addition an inspiratory and expiratory stridor occurs, which does not lead to acute severe symptoms of suffocation, but lasts for days or weeks. With the laryngoscope the epiglottis appears red and thickened, also the ligamenta aryepiglottica. Differential diagnosis from diphtheria is made by the absence of diphtheria bacilli; from pseudocroup by the slow onset and the long duration of the condition; and from both, by the great tenderness of the larynx and trachea, which is found in the deep-seated form.

The treatment consists, in addition to the general measures as in pseudocroup and possible intubation or tracheotomy, in local blood letting at the larynx. Heubner recommends that two to four leeches be laid in the vicinity of the larynx and that the patient be allowed to bleed thoroughly.

FOREIGN BODIES IN THE BRONCHIAL TUBES

The aspiration of beans, seeds, coins, buttons, etc., occurs not infrequently in children. With larger objects it is easy to determine, on account of the acute onset, that the dyspnœa is due to aspiration of a foreign body. With smaller objects, the history is sometimes lacking. The diagnosis of a foreign body lying free in the trachea or in one of the large bronchi may be made by the flapping sound in respiration. If it obstructs a bronchial branch, there is early an absence of the respiratory murmur over one lung or a part of one lung with normal percussion findings; later there is usually infiltration. With metallic foreign bodies an X-ray plate is useful in determining the position. It is best to use the laryngo- or bronchoscope (Killian); with the latter it is usually possible to remove the foreign body. If these methods can not be employed, and it is suspected that the foreign body is in the upper part of the trachea, removal by tracheotomy may be attempted.

On the whole, prognosis is not favorable without treatment by a specialist. Secondary broncho-pneumonia, especially in little children, often causes death.

PAPILLOMA OF THE LARYNX

Granulomata of the larynx may be observed after too long continued intubation, due to decubitus. In the middle of childhood, small nodules may develop on the vocal cords without trauma. They are usually attributed to overexertion of the vocal cords from crying or singing (singer's nodes) and are probably to be regarded as scrofulous affections of the mucous membrane of the larynx. They cause a prolonged hoarseness and usually disappear spontaneoulsy at the end of childhood.

The most common true tumor is the papilloma. It is seated usually on the true vocal cords, produces a husky or hoarse voice, and if extensive, severe dyspnœa. The diagnosis can be made only be means of a laryngoscopic examination. With small tumors, one may wait for several months for spontaneous healing. In most cases the removal by the endo-laryngeal way is indicated, but this unfortunately does not prevent recurrences.

THE ACUTE TRACHEOBRONCHITIS OF OLDER CHILDREN

One of the most common illnesses of school children is the catarrhal affection of the trachea and the large bronchi, while the small bronchi, which in infants are so often attacked, remain unaffected. There is a swelling, redness, and extensive secretion of mucous, which beside mild general symptoms causes cough. The fever is high for one or two days, then gradually subsides, but the cough remains longer. In the beginning it is hoarse and barking; later with an increase in secretion, it becomes looser. Inclination to cough is usually more pronounced in the morning after arising and in the evening after retiring, and lasts until several thick masses of mucous have been coughed up. The sleep may be disturbed by paroxysms of coughing lasting several hours, which exhaust and frighten the child. Auscultation and percussion usually gives negative findings, except for large râles over the trachea. A differential diagnosis must be made from whooping-cough and tuberculosis. If the tuberculin reaction is negative the prognosis is good; with a positive tuberculin reaction, it is less so.

The treatment consists of moist compresses on the thorax, put on at night with a dry linen cover and taken off the next morning. As long as the fever lasts, the children should be kept in bed and after the fever is gone, if the weather is cold, they should be kept indoors for a week. For the cough an expectorant should be given (codein sulphate 0.1, ammonii chloridi 3.0, syrup tolu 50.0, aquæ ad 100; in teaspoonful doses).

BRONCHITIS

The etiology of catarrhal affections of the middle sized bronchi is that of rhinitis. Here it must be mentioned again that children with exudative diathesis have an especial inclination to recurrent and prolonged bronchitis, without ever being able to find a plausible for the cause of this anomaly. Similar to these attacks of bronchitis of the exudative child are those of the rickitic child. These children often have for a long time a loud rattling sound in the chest, which may be heard at a distance and be felt by the

palpating hand. This originates in the large bronchi and has the peculiarity of not being associated with an inclination to cough.

Acute bronchitis is not only closely related to acute rhinitis but easily passes into bronchiolitis and broncho-pneumonia. Most of the time the entire respiratory system is affected, but medical nomenclature designates only the most affected part. It is presupposed that in severe affections, as in broncho-pneumonia, the rest of the mucous membrane is also involved, but interest centres only in that part which has the greatest importance in prognosis. In a similar way, in gastro-enteritis of children, the entire tract is affected, and only rarely an affection of an isolated part can be diagnosed.

The symptoms of acute bronchitis in children are first of all cough and fever; the sputum which in adults plays an important rôle in the diagnosis, is not expectorated by children, but swallowed.

Only toward the close of childhood does the expectoration of sputum occur spontaneously; in smaller children this very seldom happens and then only when there is much secretion and prolonged illness. Children from four to five years who cough up sputum almost always suffer from tuberculosis or bronchiectasis or at least have had a prolonged attack of whooping cough. If one wishes to examine the sputum, one must take a small wad of cotton, press it with a tweezer against the back wall of the throat until a cough results, or one obtains the swallowed sputum by aspiration of the empty stomach in the morning.

In acute bronchitis a conclusion as to the type of secretion can be drawn from the character of the cough. In the early stages as long as there is a little secretion, the cough is harsh and barking. Later with the increase of secretion, it becomes looser. The fever may be of varying heights; depending on the infectious agent, the temperature varies from a little over 37°C. (98.6°F.) up to 40°C. (103°F.) or more and may be very irregular at different hours of the day. The circulatory system usually shows nothing striking with the exception of an acceleration of the pulse, which corresponds to the height of the fever. The gastro-intestinal tract in small children is often sympathetically affected. In the beginning, as in every illness with temperature there is slight vomiting; the tongue is coated; there is loss of appetite. Bad stools and pain in the epigastrium are symptoms which are to be explained by the action of the infective agent on the mucous membrane of the intestines. Older children have headache, lassitude, and sleeplessness caused by coughing.

In the physical examination, auscultation gives coarse, loud, but not sonorous râles. The vesicular breath sounds are sharp, the expiration prolonged. Percussion shows no peculiarities. Holding the breath for some time or persistent loud crying during the examination are favorable signs. In severe affections like bronchiolitis or pneumonia, it is not possible to hold the breath long. Uncomplicated bronchitis with temperature subsides usually in one to two weeks.

In general the prognosis of simple bronchitis is good as long as it remains as a catarrh of the large bronchi; with higher temperature and labored respiration, the prognosis must be guarded, as it can always end in broncho-

pneumonia. This is especially true in infants; in older children the danger of broncho-pneumonia is not so great, but in every localized bronchitis, the possibility of tuberculosis must be considered. In every beginning bronchitis one must also think of measles and pertussis. In measles the conjunctivitis soon appears, and after a few days the diagnostic Koplik's spots; with pertussis on the other hand, the diagnosis must be deferred eight to fourteen days until the typical whoop appears, because so far there is no other way of establishing the diagnosis. Severe cough, out of propor-

Fig. 103—Croup kettle.

tion to the negative or slight findings on auscultation, and lack of fever arouse suspicion of a beginning pertussis.

As a prophylaxis against bronchitis, the children should be kept away from adults who have coryza.

An infection producing only nasal and throat symptoms in adults, when transmitted to children, especially infants, may cause an extensive reaction of much greater severity and longer duration. People should be warned against kissing children, or coughing or sneezing near them.

As a general prophylaxis, gradual hardening of the child is desirable in order that it may be able to withstand changes of temperature which in delicate children favor the development of the infection. In summer the children should be outdoors as much as possible, and at night the window should be left open. Cold sponges are advisable in strong children only and then with water at room temperature. This should be done either in the morning or in the evening before going to bed and should be followed by a warm rub.

The therapy of acute bronchitis is similar to that of acute coryza: Rest in bed and sweats, as well as wet packs which should be warm or cool depending on whether or not the child has fever. In bronchitis with temperature, chest compresses with water at room temperature, and renew every one to two hours, are useful.

Such a compress is most conveniently applied as follows: A Turkish towel, or a diaper, long enough to encircle the chest, is folded so that its width will cover from the axilla to the umbilicus. This is wrung out of water of the desired temperature and the child laid in the centre on its back. The ends are brought forward under the arms and crossed in front of the chest, passed over the opposite shoulder and pinned to the part of the towel covering the back. The whole is then covered with a dry woolen cloth or with oiled silk to prevent evaporation. It is convenient to have two covers so that they may be dried.

Oil may be used for hot compresses. For this purpose a large flannel saturated with hot oil and wrung out as dry as possible is applied in the same manner as the moist compress described. The advantage of the oil compress is that it does not dry out and hence holds the heat better. It is, therefore, best adapted for use in those cases in which a compress is to be left in place all night.

For high fever warm sponge-baths or tepid (35°C. or 90°F.) tub baths may be used. Body packs with water at room temperature in which the patient is left for ten to fifteen minutes will serve to reduce high temperatures. In using these hydrotherapeutic measures great care must be exercised to prevent chilling. Cyanosis is a distinct signal for their prompt interruption.

The air of the sick-room should not be too warm and should be kept damp. It is of great importance, especially with older children, to humidify the air by means of a steam kettle. The bronchitis kettle equipped with an alcohol lamp can be placed beside the bed and is very practical for that purpose. In summer the open-air treatment is excellent; in winter it is to be used with care. It is very good for the children to lie outdoors on the veranda on sunny days even in cold weather; they must, however, be well wrapped and have hot water bottles around them. The medical examination, meals, and changing of diapers should take place in a warm room.

For the medicinal treatment of acute bronchitis, expectorants are commonly used, but without much result. For extensive râles, ten drops of syrup of ipecac, or two to ten drops of liquor ammonii anisati in sugar water three times a day, or finally, creosote carbonate, 0.5-1 drop in milk.

For few râles and severe inclination to cough, give children from six years up, morphin (two to five times one milligram) internally. Codein sulphate can be given to children over two years of age, giving one centigram (⅙ gr.) per day in divided doses.

If there is a disturbance of the digestion, it is better not to use internal medication. Especial attention must be given to the diet which during the time of the fever should be liquid or soft. Later gradual return to the

usual diet is permissible. During the fever, liquids are to be given freely (lemonade, weak tea or milk with mineral water).

In case of chronic bronchitis, if a change of air (a sojourn at the sea or in the mountains) is not possible, inhalations of hot air (Schmidt) or of salt steam (Mayerhofer) may be used.

This latter method rests on the following: Chemically pure sodium chloride, molten at 1000 degrees Centigrade, gives off a salt vapor and a dense fog of minutest sodium chloride crystals. This sodium chloride fog is exceedingly mobile and hard to condense, so much so that, for example, it can be blown like the smoke of a cigar, through water, or even through a silver nitrate solution without being completely destroyed, although a part is dissolved or reacts with the silver nitrate. Contrary to the usual inhalations of moist steam, the sodium chloride fog penetrates into the finest alveolæ of the lungs, and there produces the strongest possible sodium chloride irritation.

During such an inhalation, for example, the character of the râles is so changed that after ten minutes instead of dry râles, moist ones can be heard (Mayerhofer). The necessary apparatus can easily be constructed by any chemist.

ASTHMATIC BRONCHITIS

In the discussion of the etiology of acute coryza, hay fever, in which the rhinitis is caused by pollen, has been mentioned. A similar condition, the cause of which is still unknown, develops in the bronchi. This disorder occurs chiefly in neuropathic children with an exudative diathesis, and especially if they have or have had a chronic eczema. The past history of these children shows that they have had bronchitis frequently, even during the first year, and that they have had eczema of the scalp and cheeks or lichen strophulus.

With every recurring attack of bronchitis in such children, more and more definite signs of labored respiration without obstruction in the larynx gradually become noticeable. This respiratory embarrassment resembles the asthma of adults. At the age of six to ten years these children, otherwise strong and well-nourished or even over-nourished, regularly at the beginning of each winter, develop a slight febrile cough. If a first attack has disappeared and the child goes out during cold weather or is exposed to high winds, the cough reappears and may continue for weeks. With short intervals of relief, this may go on during the whole winter. Expiration becomes more and more difficult. The child raises himself from the bed, and the breathing is wheezing and labored. The convulsive cough brings up only a little tenacious sputum. Physical examination shows a well-formed and very deep thorax persistently in the phase of inspiration and with small respiratory excursions. Percussion gives increased resonance over the entire lung, and auscultation, many coarse, piping râles. Eosinophilic cells are always found in the sputum. Charcot Leyden crystals and Curschmann's spirals are rarely discovered in children.

The prognosis is favorable. The attacks usually disappear in the spring, but tend to reappear at the slightest exposure during the next winter.

In making a differential diagnosis, tuberculosis is to be considered first of all, particularly in small children. In infancy expiratory asthma is due in many cases to tuberculous swelling of the bronchial glands. In this event the prognosis is, of course, grave.

With negative tuberculin reaction, and in older and stronger children, a diagnosis of asthma is justified. If permanent recovery is to be secured, the neuropathic factor must be taken into account.

With older children the best results are to be obtained by a change of climate. In the matter of choice it makes little difference whether the patient be sent to the sea-shore, to the mountains, or merely into the country. The main thing is to convince him that he can breathe more freely in another atmosphere, so that he may lose the fear of the attack and the expectation of the cough. If a climatic change is not possible or if it fails, breathing exercises may be instituted. Several times a day the thorax should be compressed in expiration for a few minutes.

Kuhn's mask is usually borne very well by children. The apparatus lessens the ease of inspiration, and this draws the blood to the lungs.

Hydrotherapeutic procedures by way of the cold sponge or douche are at times successful.

As a further means of relief, a complete change of diet serves as effectively as in a chronic eczema. Children accustomed to taking large quantities of milk should discontinue it in favor of a mixed diet. A salt-free or a fat-free diet may be tried also. Expectorants or sedatives are required during the acute attacks. Codein (0.005-0.02 gm.) or morphin in one milligram doses (gr. $\frac{1}{60}$) for each year of age may be used. If necessary an enema, containing 0.3 gm. of chloral hydrate in 30 gms. of mucilage of acacia may be given.

Sodium iodide is recommended for continuous treatment. It may be given in the following formula:

	Gms.	
℞ Sodii iodid.....	1.....	(gr. xx)
Syrupi simplicis.....	10.....	(ʒ ii)
Aquæ destillatæ ad.....	100.....	(ʒ iv)

M. Sig.—Two teaspoonfuls each day.

Larger doses may be given in the proportion of one gram to each year of the child's age, in 100 c.c. of water, to be given in doses of one tablespoonful, in milk, after the noon and evening meal.

Asthmatic conditions in children are frequently the expression of their sensitization to foreign proteins. The younger the child the more often food proteins are at fault. (Walker and others.) Of these, according to the skin tests, eggs and cereals are the most frequent offenders.

This sensitization may be due to inhalation through the respiratory tract, ingestion into the gastro-intestinal tract, which may be permeable for undigested foods (Schloss and Worthen), absorption from the skin or conjunctivæ, and infection anywhere in the body, particularly the so-called foci of infection, as tonsils, teeth, etc.

In the differential diagnosis of asthmatic conditions the skin test plays an important rôle. Not only food proteins, but also animal emanations and bacterial proteins must be considered.

In the treatment of the attack the subcutaneous injection of 0.2-0.3 c.c. of adrenalin chloride solution (1-1000) repeated if necessary gives the best results.

The dietary treatment requires omitting the food at fault, and diminishing non-specific protein to the minimun.

The desensitization of the patient by the feeding of minimal, gradually increasing, amounts of the food at fault seems to be followed by success in some cases.

The removal of the foci of infection is essential.

The value of vaccines is, to say the least, doubtful.

CAPILLARY BRONCHITIS

A purulent catarrhal inflammation of the smallest bronchioles is a form of bronchitis common in infancy and hardly ever seen in later years. Most of these cases occur between six and eighteen months of age. This is also the period of florid rickets. As an actual fact, this form of bronchitis is so frequently seen in severely rickitic children that causal relationship must be suspected. Whether this relationship depends upon the mechanical conditions of the soft rickitic thorax, which prevents ventilation of the bronchioles or whether the mucous membrane of the bronchioles is in itself injured by rickets, cannot be determined. The etiology of capillary bronchitis is no more specific than that of coryza. Frequently the disease develops during pertussis or measles, and still more frequently with influenza. Most often it is seen as a secondary infection by the influenza bacillus, following measles; and it is supposable that a bronchial catarrh may spread from the larger to the smaller bronchi by a secondary infection.

The clinical difference between this disease and broncho-pneumonia is is not always a very distinct one, but pathologically the differentiation is very clear. At autopsy, the lung is found to contain air throughout; it is a light red in color and without compression of its lobules. If pressure is made upon a cut surface, innumerable droplets of pus exude from the smallest bronchioles. The mucous membrane of these minute air passages is bright red, swollen, and covered with mucopus.

A microscopic examination shows the vessels filled with blood clots and the bronchioles plugged with mucous, polynuclear leucocytes and epithelium. The alveoli are almost entirely clear and even abnormally expanded. The walls of the bronchi and the interstitial connective tissue show small cell infiltration.

These classical findings, however, occur only in those cases that die very shortly after the onset of the disease. After the closure of the bronchioles has existed for a longer period, secondary changes appear. Foci of lobular infiltration are found here and there, resulting from the aspiration of pus into the alveoli or from the extension to them of the inflammatory process. Atelectases and partial emphysema develop. The distention of

certain parts of the lung may be explained by the fact that the air is pumped in with inspiration but its expression through the narrowed tubes is imperfect. In these regions, gradual absorption of the air takes place, and these areas become dark brown and atelectatic. The atelectases occur chiefly in the posterior dependent parts, and the emphysema in the anterior portions of the lung. The pleura is negative and other organs show only congestion.

The clinical picture develops very rapidly. The disease is usuall sequent to an old bronchial catarrh. The first symptoms are high fever and labored respiration, followed by a very characteristic condition; an acute pallor, especially noticeable in children who previously had good color. Later, a bluish tinge, due to cyanosis, mingles with the pallor, eventually resolving itself into a peculiar grayish hue.

The respirations become very frequent and labored. The thorax is held spasmodically in the inspiratory phase, but only a small amount of air enters. Consequently, the normal bulging of the epigastrium is replaced by retraction

In all febrile diseases, excepting tuberculous meningitis, the respiration is of increased frequency, but the rate is never so markedly increased as in capillary bronchitis. In this disease it may be increased to one hundred or more per minute and out of proportion to the pulse-rate. While under normal conditions the ratio of heart-beats to one respiration is three or four to one, in capillary bronchitis the ratio is two to one, or even less. The respirations are apt to be accompanied by movements of the alæ nasi. The expiration is convulsive, and whistling râles may be heard at a distance. The thorax of rickitic children will change its form with each respiration, especially along the rosary and in the flanks. At the outset of the disease the child is still strong enough to overcome the reduced aëration by forced efforts, but later the strength fails, and the respiration becomes more and more frequent and superficial. The cough, which at first resembles attacks of pertussis, becomes gradually weaker.

The physical signs are in the beginning comparatively slight. Percussion gives a clear, deep note and only occasional fine bubbling râles can be heard on auscultation. In spite of this, tactile fremitus may be reduced on account of the plugging of numerous bronchi with secretion. Later the râles become general and an indefinite dulness is found on percussion over the back and especially along the spine, indicating atelectasis or beginning pneumonic foci. The former disappears on deep breathing if the patient is turned upon the opposite side from that under observation. Anteriorly, the cardiac dulness is usually covered by parts of the distended lungs.

As a result of the deficient aëration of the blood and the lowering of the heart's force, cyanosis becomes more and more marked, and the extremities become pallid and cold. The face, too, grows strikingly pale.

As in all acute diseases of childhood, the nervous system gives symptoms of associated disturbance. Initial vomiting occurs, also extreme restlessness,

and very frequently general convulsions of an epileptiform nature. These disturbances later give way to a general apathy.

The prognosis of capillary bronchitis is always very grave. Severe rickets and general convulsions must be considered a bad omen. Over 50 per cent. of clearly defined cases die within a few days. If the patient survives the first week without the development of lobular pneumonia, there is some hope of recovery.

The diagnosis is based mainly on the dyspnœa, without definite physical findings. The disease is not always distinguishable from an early broncho-pneumonia or from a lobar pneumonia. Later in its course auscultation will determine whether there is consolidation of lung tissue. In children having signs of tuberculosis or scrofula, miliary tuberculosis must be considered. A positive differentiation is difficult because the tuberculin reaction often fails in miliary tuberculosis. The Roentgen picture may enable one to make the distinction.

The dyspnœa may be so severe as to suggest an obstruction of the larynx incident to a diphtheritic or false croup. If the tonsils, nose, and nasopharynx are clear, diphtheria will hardly be present. If a constriction due to false croup is suspected, intubation may be tried. If this does not give relief, the trouble lies farther down in the small bronchi.

The therapy is, at first, that of bronchitis in general in the way of sweats, warm packs, expectorants, or emetics (see p. 367). When the dyspnœa becomes intense and general pallor appears, a mustard bath is indicated. This has its disadvantage, however, in the fact that the irritating volatile oil of mustard affects not only the skin but the air passages as well. For this reason Heubner advocates the mustard pack. This is prepared as follows: A cupful of ground mustard is mixed to a smooth paste with a little cold water and this paste is stirred into a quart or more of boiling water in an open basin. A towel or sheet large enough to completely enfold the patient is wrung out of this mustard water and spread upon a woolen blanket and quickly wrapped around the patient. Because of the irritation due to inhalation of the mustard vapors it is well to hold a wet cloth over the patient's nose and mouth. The pack is then surrounded by hot water bottes and left in place for fifteen minutes to one-half hour. If the child reacts it will soon become restless and begin to cry. It has been found advantageous to give oxygen inhalations or place the patient near an open window or even stick the child's head out of an open window during these deep respirations.

A satisfactory reaction is achieved when the skin becomes bright red wherever the pack touched. As soon as this desired result is obtained the pack is removed, the patient sponged to remove all particles of mustard from the skin, and then wrapped in a further tepid pack with a clean cloth wrung out of plain water. This latter is left in place for one to two hours, when it is removed, the skin dried and gently rubbed with olive oil or cocoa butter. The temperature often rises during this procedure and after it is completed the patient should be put in bed and not disturbed for several hours.

The failure of the mustard pack to redden the skin is a bad prognostic sign. A distinct improvement is often seen in a few hours after an intense reaction of the skin. The mustard pack may be given once a day. In mild cases, in which this treatment would be too severe, since there is always some danger of collapse, a hot bath with douches will suffice.

Heart stimulants should also be employed. Digitalis, or one of its derivatives, may be used; or in cases of long duration, tincture of strophanthus; or in acute relapses, injections of camphor or caffein may be tried. (See cardiac diseases, broken compensation.)

In very severe cases, venesection may be employed. This is quite a difficult procedure in small, fat children. The attempt should first be made to withdraw blood by puncture. If this is not successful, the saphenous vein must be exposed by dissection, and from 30 to 50 c.c. (1-2 ounces) of blood withdrawn from it.

Inhalations of oxygen are very helpful in some cases. When the more acute, threatening symptoms have disappeared, the treatment is the same as that of acute bronchitis.

Broncho-tetany.—Some cases of asthma and capillary bronchitis, occurring at the age when florid rickets develop, may be features of the disease-picture of broncho-tetany recently described by Lederer. In spasmophilic children who had died with the signs of acute suffocation or even of supposed broncho-pneumonia, he has found no evidences of pneumonia autopsy, but of edema or atelectasis instead. He suggests that tetanic contractions of the bronchioles may have caused the symptoms in question. Early indications of dyspnœa in rickitic and spasmophilic patients should be treated, therefore, with calcium salts in the form of calcium lactate (1.0-5.0 gms.) each day. (Rietchel, Curschmann.)

BRONCHO-PNEUMONIA

In most instances an inflammation does not confine itself to the small bronchi but invades the alveoli. At first the air-cells are filled with mucous, but later an infiltration of the interstitial tissue occurs. The numerous small foci of infiltration soon become more or less confluent. Pathologically whitish-yellow miliary areas are found at the very outset. On section these discharge a purulent fluid. Later, reddish-brown indurations, varying in size from that of a pea to a small nut, are observed. If the process continues wedge-shaped foci appear which may eventually involve the entire lobe. The borders of the lungs are emphysematous, and atelectases are formed where portions of the bronchi are obstructed by the secretion or compressed by pneumonic foci in the neighboring tissue. According to the extent of the area affected, two types of broncho-pneumonia are to be distinguished, the disseminate and the pseudo-lobar forms.

Bacteriologically, numerous organisms are productive of the disease. Among the more common are the diplococcus pneumoniæ, the diplococcus catarrhalis and the influenza bacillus. Again, pneumonias occur in young infants and especially in atrophic nurslings, in whom no micro-organism is found. These forms are afebrile and simply hypostatic.

Aspiration pneumonia has a much more serious course. This condition may arise during the first few days of life as a result of the aspiration of amniotic fluid. It also occurs, as in adults, following severe exhaustion, as for instance in typhoid fever. Aspiration pneumonia is frequently a result of diphtheritic paralysis. Since the larynx is not efficiently closed on account of the paralysis of the epiglottis, and since effective coughing is impossible because of the incomplete closure of the vocal cords, fluids often enter the bronchi and cause pneumonia. This occurs more readily when there is coincident cardiac weakness.

Broncho-pneumonia is a very serious complication of measles. It is probable that all infectious organisms find a very fertile soil in the child ill with measles, since the antibodies of various bacteria, like those of the bacillus tuberculosis, are unable to maintain their normal activity during its acute stage. Pneumonia usually sets in after the disappearance of the rash and is first announced by the renewal of fever and the development of dyspnœa.

Pertussis, like measles, is not usually in itself a dangerous disease; its fatality depends upon a complicating pneumonia. In scarlet fever, on the other hand, pneumonia is extremely rare. When it does occur it has a rapidly fatal termination.

Rickets has no direct influence upon the occurrence of broncho-pneumonia, but it does tend to make the prognosis of coexisting pulmonary disease the more unfavorable. This is probably due to the mechanical difficulties it occasions in respiration.

The Clinical Picture.—When broncho-pneumonia is not imposed as a complication upon a preëxisting catarrh of the respiratory passages or upon the course of an infectious disease, its onset may be either sudden and attended by vomiting and fever, or it may be gradual. The vomiting soon stops, but anorexia follows. The fever is continuous as in lobar pneumonia, but usually recedes in the morning, and reaches 39°-40° C. (102°-104° F.) in the evening. The pulse is increased proportionately to the temperature, but the respiratory relationship is disturbed, showing an abnormally high frequency. Early, the respiration, and especially the expiratory movement, becomes labored. The alæ nasi and the auxiliary thoracic muscles are soon brought into action.

Physical examination shows, at first, only a few scattered râles, but as the disease progresses, these grow more numerous, louder, and more ringing. Expiration becomes bronchial in character, and finally distinct bronchial breathing and subcrepitant râles are heard.

The physical signs are most frequently found first at the lower borders of the lungs and between the scapulæ. It is not long before râles are heard all over the lower lobes. Here and there increased breath sounds, evidencing infiltration, may be heard in distinctly circumscribed areas. In the course of a day or two, one or both lobes are infiltrated, when bronchial breathing and ægophony are found. The anterior surfaces are, as a rule, clear, but râles are eventually heard over the entire lung. Percussion may be at times entirely negative, or only slight dulness and tympany are ap-

parent. Generally a relative dulness running along the vertebral column may be recognized. The Roentgen picture shows disseminated shadows representing the infiltrated areas.

The clinical picture of broncho-pneumonia is not constant. Cases are observed in which there is only a slight fever, with small foci of infection and accordingly little disturbance of general health. On the other hand, with quite similar findings, the patient may be completely prostrated, and death speedily ensues. Again there are cases which from their onset are attended by high fever, great restlessness, diarrhœa and early unconsciousness, with rapidly spreading infiltration of the lower lobes, and ending fatally in a very short time.

In consequence of the disturbed function, a small and very frequent pulse and cyanosis, especially at the finger tips, are observed. The urine is diminished in quantity and contains albumen. The intestinal tract may be affected with resulting diarrhœa, muco-sanguineous stools and meteorism, accompanied by a high position of the diaphragm.

The prognosis depends essentially upon the area of infiltration and is difficult to forecast. The age of the patient is an important matter. Broncho-pneumonia is especially common and most to be dreaded between six months and two years, as is shown in the following table:

FREQUENCY AND MORTALITY OF BRONCHO-PNEUMONIA DURING EARLY CHILDHOOD (Holt).

Age.	No. of Cases	or	Per cent.	Mortality Per cent.
During first year	224		53	66
During second year	142		33	55
During third year	46		11	33
During fourth year	10		2	16
During fifth year	4		1	

In the first year feeble children react to the disease very much as old people do. By simply lying on the back, they may acquire a hypostatic pneumonia which is localized in a border of infiltration on both sides of the vertebral column (the paravertebral pneumonia of Gregor).

In the differential diagnosis, broncho-pneumonia is to be distinguished from bronchitis by its auscultatory signs. Both lobar pneumonia and pulmonary tuberculosis are to be excluded. The latter, in its onset, sometimes resembles a diffuse bronchitis with pneumonic respiratory qualities.

Treatment.—Therapeutically, the most important item of treatment in pneumonia is to see to it that the child is carried about or at least permitted to sit up a great deal. This is equally important as a matter of prophylaxis if the child has bronchitis. Extraordinary results may be obtained by proper feeding and care. Nursing infants should be kept on breast feeding. It is because of these safeguards that complete recovery from broncho-pneumonia is often made in homes of poor if the mother can devote herself entirely to the care of the sick child, while pneumonia patients in the best equipped hospitals die if the nursing force is inadequate.

The treatment of broncho-pneumonia consists chiefly of hydrotherapeutic measures. In contradistinction to capillary bronchitis, in which the

mustard pack is used, the Priessnitz pack, with water at room temperature and frequently changed, or warm baths combined with cold douches, are used in this disease. The bath should be given at about 37° C. (98.6° F.) and the water for the cold douches at about 25° C. (77° F.). After the bath the child should be rubbed with a warmed bath towel and put back into the previously warmed bed. These baths may be repeated three times a day, if necessary, provided the patient does not show signs of exhaustion. To safeguard the occurrence of collapse, an analeptic may be given before the bath, in the form, for instance, of a few drops of brandy in tea or coffee.

In broncho-pneumonia, warm baths (35° C.) or even hot baths (up to 42° C.) are very beneficial. Cool baths in delicate and weak children lead to cyanosis, increase of the pulse-rate, cold extremities, and general prostration while hot baths, even in fever, have a beneficial and quieting influence. (Feer.)

The inhalation of oxygen gives very good results in some cases, especially if dyspnœa and cyanosis are present. The pulse improves, and the color becomes rosier if the child inhales the oxygen well. A difficulty in the use of this method however is the tendency to tire the child in the attempt at inhalation. An older child may be permitted to take the glass tip of the tube in his mouth. With younger children the gas is passed over the face from an inverted funnel, or a specially constructed mask.

Medicinal measures have a very slight influence upon the course of the disease. It is customary to give such mild expectorants as ipecacuanha, senega, liquor ammoniæ anisati, etc. Cardiac remedies are of more value in the event of threatened heart failure. Camphorated oil, caffein, and more especially digitalis may be effective. Digitalis is best given in the form of the infusion, in doses of 0.6 c.c. for a period of two days; or as digalen in doses of one drop for each year of age, three times a day. (See also the therapy of cardiac insufficiency.)

LOBAR PNEUMONIA

The acute inflammation of the lungs chiefly characterized by infiltration of a single lobe and by a typical temperature curve has received various names. It is known as fibrinous, lobar, croupous, massive, and pleuropneumonia. Not one of these terms fully covers the peculiar features of the disease. In America, lobar pneumonia is the designation most widely adopted, while in Germany, the term croupous pneumonia is more generally used.

Bacteriologically and pathologically, the course of the disease in children is identical with that in adults; and, therefore, it will not be necessary to discuss these details here. Clinically however, lobar pneumonia presents a number of peculiarities in children. Formerly the disease was supposed to be very rare in childhood; but this remains true only in the first few months. It is never observed before the third month, and after that period its frequency increases rapidly, reaching its maximum incidence between the second and the fifth years. The following table shows:

THE FREQUENCY OF LOBAR PNEUMONIA DURING THE VARIOUS PERIODS OF CHILDHOOD
(Holt)

Ages	Cases	Percentage
During 1st year	76	15
From 2nd to 6th year	309	62
From 7th to 11th year	104	21
From 12th to 14th year	11	2
Total	500	100

The frequency varies greatly from season to season. The disease is at a minimum early in the autumn and at its maximum in the spring. At times exposure to cold or some direct traumatic influence seems to stand in direct causal relation to the development of pneumonia which, as in coryza, may be charged to temporary reduction of the resistance of the body.

Lobar pneumonia is most frequently localized in the right upper lobe or in one of the lower lobes. In 950 cases in children under fourteen years of age, Holt found the distribution of the disease in the lungs as follows:

Seat of the Disease.	No.
Right lung,	
upper lobe only	176
middle lobe only	12
lower lobe only	168
more than one lobe	77
Total, right lung	433
Left lung,	
upper lobe only	93
lower lobe only	263
more than one lobe	38
Total, left lung	394
Both lungs,	
upper lobes	13
lower lobes	41
variably localized	69
Total, both lungs	123

In older children, the disease begins, as in adults, with a chill and with pain in the side. In younger children, the onset is not always so distinct, and the chill, in particular, is not often marked. On the other hand, those initial symptoms that usually usher in all severe affections in childhood, such as vomiting or convulsions, are common. Sudden vomiting without diarrhœa or a history of previous overfeeding is a warning signal that should find one prepared for almost anything. Scarlet fever, tuberculous meningitis, pleurisy or pneumonia may be announced in this manner. It requires a careful examination to determine from what special site this one symptom is provoked.

Another difference lies in the fact that while in the adult the pain is referred to the side, in the child it is replaced by abdominal pain. Young children localize pleural and all other thoracic pain in the abdomen. This is a point to be remembered, since the cause of all abdominal pains cannot be found there. Not infrequently appendectomies have been done in vain, when the pain which led to a diagnosis of appendicitis was really caused by pneumonia or pleurisy.

Aside from the abdominal pain, an expiratory grunt is typical of lobar pneumonia in small children. This symptom is even more prominent in this disease than it is in broncho-pneumonia. While this slight, non-stridulous sound is produced with every expiration, the inspirations are silent.

The sputum that older children sometimes expectorate is glairy, transparent, tenacious, and of a reddish-brown color. It is, at times, even mixed with blood. When placed in water it will occasionally show dichotomously branching casts of bronchi. As resolution occurs the sputum becomes yellowish.

The lung findings in childhood are not so distinct as in the adult. The dulness is not so pronounced. It is necessary to percuss very gently, rely-

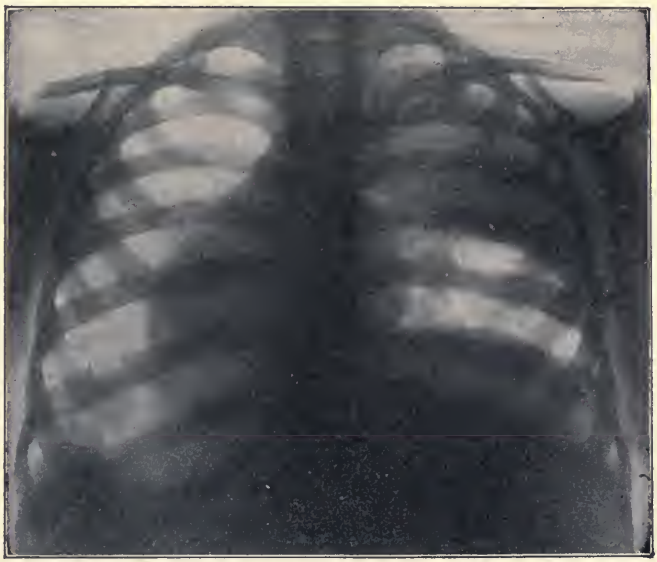


FIG. 104.—Pneumonia of upper right lobe. (Vienna Children's Hospital.)

ing more upon the tympanitic resonance than upon the shortening of the sound. The physical findings are usually not clearly apparent until two or three days after the clinical onset of the disease. Indeed there are cases in which the symptoms become manifest only after the fifth or sixth day and sometimes not until crisis occurs. These are particularly of the type in which the pneumonic infiltration is centrally located and only gradually approaches the pleura. The Roentgen examination renders wonderful service here. By its aid it has been possible to show that pneumonia begins, as a rule, at the hilus of the lung and spreads thence to the periphery.

Auscultation is rather more difficult than it is in the adult. The child will not breathe when bidden, or may even hold the breath. If the child cries, the respiration is more intense, but the inexperienced observer hears only the crying. The pediatrist has to learn to use these crying periods for auscultation. Over infiltrated areas the crying or sighing has an entirely

different sound than it has over the intact lung. The sounds seem as close to the ear as though the stethoscope had been placed over the trachea. During inspiration the metallic quality of the râles is of importance.

If the foci of infiltration are very small or are centrally located, the the changes may be distinguished only by the auscultation of vocal sounds over the various portions of the thoracic wall. Special attention should be given to bronchophony in the axilla where it usually occurs earliest.

Aside from the type of respiration and the high and continuous fever, the labial herpes, the red and slightly cyanotic cheeks, and the short painful cough are points which aid one in making a tentative diagnosis on sight. True herpes is not constant, nor does it appear early, but rather at the fastigium. Frequently there is a subicteric discoloration of the skin. At this stage the patient seems very low, and if it were not for the knowledge that the prognosis of lobar pneumonia is very favorable in childhood, the general appearance might suggest that death is actually imminent. Fortunately the appearance of the relieving crisis may be predicted with considerable certainty at this point. Free perspiration and restful sleep may suddenly take the place of the distressing symptoms, and within twelve to twenty-four hours the temperature may fall from 40° C. (104° F.) to the normal, or to subnormal level. Great weakness persists, but the appetite improves, and within a few days, the appearance of the patient changes completely.

The crisis generally occurs on the seventh day, but this is not by any means constant, as is shown by the following table:

Appearance of Crisis in 567 Cases of Lobar Pneumonia in Children. (Holt.)

Days of Illness.	Number of Cases.	Days of Illness.	Number of Cases.
2	3	11	18
3	22	12	7
4	43	13	8
5	88	14	1
6	83	15	1
7	132	18	3
8	73	21	1
9	55	26	1
10	22		

Cases are noted in which the crisis occurs very early, some, indeed, in which the disease is only of one day's duration. (Feer.)

The physical signs do not keep pace with the clinical findings at the period of crisis. The fine crackling râles that indicate resolution may be delayed for several days, precisely as the dulness and the bronchial breathing, in exceptional cases, may appear only after the crisis. The sudden drop of temperature is not always definite. Pseudocrisis is almost as common as crisis itself, a renewed rise of temperature being followed later by the true crisis. More rarely the termination, in the event of pseudocrisis, may be lysis. After several days of low temperature, a reappearance of the

fever may indicate the involvement of another lobe. This is the form of so-called pneumonia migrans.

In most cases, not only the parenchyma of the lung is involved, but the inflammatory process reaches the pleura. An exudate of varying extent is formed, which may be either serofibrinous or purulent. Pneumonia as the chief cause of empyema will be discussed later.

The pericardium is affected in a similar manner. Pericarditis of this type is a serious complication and usually fatal. The pericardial exudate is generally not very large and the friction rub is obscured by the pneumonic sounds in the neighboring lung tissue. On this account pericarditis is often discovered only at autopsy.

Complicating otitis media is quite frequent, but nephritis is exceptional.

Endocarditis is extremely uncommon, and the heart muscle, which so often fails in the adult, is unaffected in the child. Ninety-five per cent. of pneumonias in childhood survive because of the great reserve power of the heart, while over thirty per cent. in the adult die in consequence of its failure.

Beside the pleura and the pericardium, distant organs, among them the joints, the bone-marrow and the meninges, are at times affected by the diseases.

Meningitic symptoms may occur when the pneumococcus cannot be found in the cerebrospinal fluid—that is without actual metastasis of the infection. The central nervous system, in fact, takes a distinct share in the pneumonic symptom-complex. For example, aphasia and temporary hemiplegia are occasionally seen in pneumonia among older children. At an earlier age, convulsions are often observed, especially at the onset of the disease. It is important that the meningeal form of pneumonia be recognized. It occurs particularly between the ages of three and seven years. The symptoms begin with vomiting and headache. Rigidity of the muscles of the neck and transitory strabismus resemble very closely the conditions seen in epidemic meningitis. Coma or delirium with spastic extremities and hyperesthesia of the skin may complete the picture. If pneumonia is found in the lower lobe, the meningeal symptoms are usually regarded as evidence of an increased severity in the general manifestations of the disease and nothing more. The diagnostic difficulties are much greater when the pneumonia is so located that it is difficult to find, as for instance when it is located in an upper lobe. Meningeal symptoms have been particularly emphasized in apical pneumonias; but actually they seem to occur with equal frequency in pneumonias otherwise localized. (Schlesinger.) They have no serious significance and disappear with the crisis.

The differentiation of the disease from broncho-pneumonia is unimportant from a therapeutic standpoint, but it is undoubtedly of great value in prognosis to be able to predict crisis. Special attention should be paid to the character of the fever, the appearance of the herpes, and the localization of the disease. Infiltration of the lower lobe with coexisting bronchitis, speaks for broncho-pneumonia. It is a matter of experience that a great variety of the febrile diseases of childhood are mistaken for pneumonia. Among these, typhoid fever, meningitis, and pleuritis may be

especially mentioned. The high fever and the rapid respiration are usually responsible for these errors. Icterus with a high temperature is suggestive of pneumonia. In this disease the spleen is not always enlarged. Frequently enlargement is found only after crisis. In addition to the physical symptoms, the blood and the urine should also receive attention. Their examination is necessary in doubtful cases for the exclusion of other diseases. In lobar pneumonia the blood during the first few days shows a marked increase of leucocytes which may run to 40,000 per cubic millimeter. The urine is scant and therefore concentrated and contains an excess of uric acid. Further, a febrile albuminuria, a diazo-reaction, and a decrease or complete absence of chlorides are observed. The sodium chloride is probably retained in the lungs and the pleura.

Therapy.—In uncomplicated lobar pneumonia treatment is really superfluous. Good nursing is the only requirement. The food supply should be diminished commensurately with the loss of appetite, but the patient must be given plenty to drink in the form of lemonade, milk with mineral water, etc. The fever should be controlled by temperature packs, changed at half-hour intervals and continued for a few hours several times a day, or by frequent cool sponging or tepid baths. If the fever lasts over eight days, antipyretics may be given. If diarrhœa occurs, tannin preparations may be used; or for constipation, castor oil or glycerin suppositories. Indications of cardiac weakness, especially with cold extremities, may require digitalis, caffein, or camphor. (See Cardiac Insufficiency.) After crisis the temperature sometimes falls so rapidly that it may be necessary to use hot water bottles or other warming measures. Expectorants may be dispensed with, but they are harmless in small quantities. During convalescence suitable nourishment must be provided. (See tuberculosis.)

CHRONIC PNEUMONIA

Chronic infiltration of the lung tissue sometimes persists after bronchopneumonia complicating influenza, pertussis or measles. It usually involves but one lobe, but this often to a considerable extent. In such cases the parietal and visceral pleura are apt to become firmly adherent, although leaving, sometimes, small fluid-containing pockets. The affected lung retracts and an excessive growth of interstitial tissue follows. The branches of the bronchi running through the disease area are often enlarged and form cylindrical bronchiectases.

Upon physical examination, extreme dulness with a tympanitic note and with diminished or bronchial breathing is discovered. The side of the thorax involved appears smaller. On account of the extreme dulness one may be led to suspect pleurisy, but contrary to the expectation which the latter would suggest, exploratory puncture proves negative. The infiltration may be resorbed after several months. In general, the prognosis is good if the condition is not the result of a tuberculous infection. It is very important, therefore to exclude tuberculosis which may be done by means of the tuberculin reaction. There is no special therapy aside from general measures, good dietary, breathing exercises, and fresh air.

EMPHYSEMA

Acute partial emphysema is often found at autopsy in fatal cases of broncho-pneumonia. In the event of recovery this emphysema disappears rapidly and has no clinical significance. True emphysema, due to over-expansion of all the pulmonary alveoli, develops upon rare occasions in the wake of bronchial asthma and pertussis. Interstitial subcutaneous emphysema, in which the air is forced under the skin through the mediastinum, is occasionally seen after wounds of the lungs, especially after tracheotomy and sometimes after exploratory puncture. It may occur spontaneously from a tuberculous cavity, or from the rupture of alveoli in pertussis.

BRONCHIECTASIS

Aside from extremely rare cases of congenital bronchiectasis, dilatation of the bronchi may develop as a result of subacute pulmonary disease in children of over three years. The most frequent of these is whooping cough, then measles, and finally pneumonia from other causes, all of which produce a distention of the bronchi by increasing the intrathoracic pressure during the attacks of coughing. Again, bronchiectasis may arise after pleural diseases in which in consequence of retraction, an inspiratory traction acts upon the walls of the bronchi.

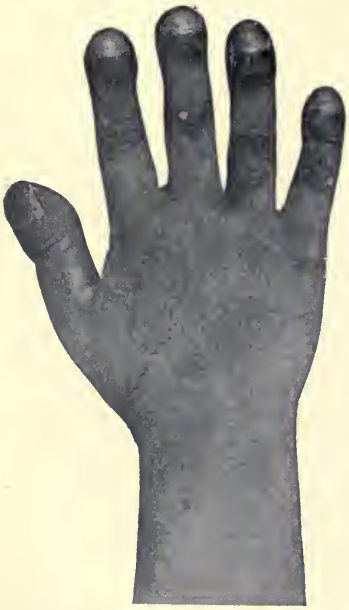


Fig. 105.—Drumstick fingers in case of bronchiectasis, ten-year-old girl. Watch glass finger-nails.

Pathologic Anatomy.—Cylindrical or sacculated dilatation of the bronchi are found usually in the lower lobe. They may be single, reaching the size of a hen's egg, or they may consist of multiple pea-sized distensions. The mucous membrane becomes atrophied and loses its ciliated epithelium, the secretion stagnates in these cavities and presents a fertile soil for the growth of various micro-organisms.

Clinically, the distressing cough persisting for hours is characteristic. It occurs chiefly in the mornings; it is loose and ends with the expectoration of large quantities of sputum, which often has a foul odor. This expectorated material sometimes shows a characteristic division into three layers. Locally, coarse râles are heard in a limited area and most commonly over the lower posterior border. Definite signs of cavity formation are rare. It is an important point, that before the paroxysm of coughing, there may be a tympanitic note or a slight dulness, which disappears after the expectoration. If the dilatation is near the surface, a cracked-pot resonance is heard, and it is possible to demonstrate a change in tone when the mouth is opened or closed.

The course of the disease is extremely chronic. Slight rises of temperature may occur for a long time without any general symptoms or even anorexia. The fever probably varies widely with the type of microorganism that has found entrance into the bronchiectasis. The condition is almost always accompanied by local bronchitis. Pleural adhesions, misplacement of the heart, and chronic circulatory disturbances may result, the latter leading sometimes to the formation of clubbed fingers (Fig. 105).

Diagnosis must depend, first of all, upon the exclusion of tuberculous cavities, and this may be done by means of the tuberculin reaction and by bacteriologic examination of the sputum.

The prognosis of large bronchiectases is not favorable. Recovery hardly ever takes place. Death usually results, sooner or later, due to some complication.

Therapeutically, the general treatment of bronchitis may be supplemented by massage of the thorax, breathing exercises, expiratory compression of the thorax, and inhalation of oil of pine and turpentine. In extreme cases, surgical interference is indicated.

Resulting pulmonary abscess and gangrene of the lung with thickening of the lung tissue and a gangrenous odor of the expired air, are extremely rare in childhood.

PLEURISY

The most important difference between the pleural exudations of childhood and those of later life consists in the relative frequency of pus. Empyemas are rare during the first half-year, but they occur quite often in the second half-year, and very commonly during the second year, after which their frequency decreases rapidly from year to year. It would seem that the child's pleura is especially predisposed to infection with the pneumococcus. In four-fifths of all cases of pleurisy in childhood, the pneumococcus is found in the exudate, while it is present in only one-fourth of the adult cases. This explains the frequency of metapneumonic empyema after lobar or broncho-pneumonia. As pneumonia, so also empyema occurs chiefly in winter and spring. Streptococcus infections of the pleura are less common in children than among adults, excepting in the new-born in whom they appear as metastatic localizations of a general sepsis. Pleurisy also occurs in cases of inflammation of neighboring organs, as for instance in appendiceal abscess. It may be associated with nephritis and sometimes develops in the course of rheumatic disease. In rheumatic and tuberculous cases, a serous exudate is found. Dry pleurisies are relatively uncommon in childhood.

In the septic pleurisy of the new-born, the disease runs concurrently with a general sepsis arising from the umbilicus, or from the phlegmon of the thorax or the mediastinum. The pus in the pleural cavity contains streptococci. It may be demonstrated by the dulness, but it is usually discovered only at autopsy. The prognosis of this form of the disease is of course bad. Surgical interference is hopeless.

Fibrino-purulent pleurisy in infancy usually represents merely a second-

ary finding at autopsy. Around the infiltrated lobes in pneumonia, villous exudates in thick masses are found. By careful examination this form may be recognized clinically, in some instances, by the friction sound. Finkelstein lays great stress upon the presence of an edema over the area of the skin of the thorax beneath which the exudate lies. This edema is so slight that it can be felt only upon careful examination.

In the infant a pleurisy may arise, without pneumonia, as an apparently primary infection of the pleural cavity or from a lymphangitis of the mediastinum. The pleuritic fibrinous exudates are very tough and show no tendency to softening. This primary lymphangitic form is peculiar, furthermore, in that it tends to spread first to the pericardium and then to the peritoneum and to the terminal joints, while the skin, the muscles, and the internal organs remain free of metastases. Heubner has described it as a clinical entity under the term multiple purulent serositis. The disease at first takes a course resembling pneumonia, with high fever and indications of dyspnœa. Distinct pulmonary symptoms, however, do not develop, but collections of pus in the pericardium and the joints become manifest. Death occurs during the first two weeks.

EMPYEMA

In most cases the onset of the disease cannot be definitely established. Especially in infants the collection of pus usually forms during the pneumonia and the symptoms gradually pass from one to the other type. In other cases, signs of pleurisy are found soon after a high febrile onset, but it may well be that these symptoms have been merely added to those of an ill-defined pneumonia. A high grade dyspnœa and pains which are referred again to the stomach region are characteristic of the involvement of the pleura if it develops rapidly. The patient prefers to lie upon the side and especially upon the affected side, because it permits freer breathing with the other lung. This also diminishes the respiratory movements of the affected side, and thereby the pain. Inspection reveals fulness of the affected side The thorax is fixed and does not participate in the respiratory movements. The intercostal spaces bulge, a fact which can be determined in thin children by palpation. Percussion gives absolute dulness. Care must be taken not to percuss too hard over the small thorax lest the tone transmitted from the unaffected lung decrease the dulness. The phenomenon which Rauchfuss and Hamburger have recently described, consisting in a strip of resonance over the affected lung, depends upon the fact that on deep percussion the tone of the one side is transmitted to the other by the thoracic wall. Similar results may be demonstrated upon the anterior surface of the thorax. However, the seemingly aberrant note over the border of the dull areas which can be made out upon gentle percussion, does not depend upon these physical causes, but rather upon changes within the mediastinum itself. For if the exudate is on the left, the heart is pushed to the right, its dulness extends far beyond the right sternal margin, and the apex beat and the heart sounds can be felt and heard to the right of their usual areas, while in

right-sided empyema the heart is not only forced to the left, but the liver is pushed downward.

Auscultation does not present the definite evidence of the condition in children that it does in the adult. It is important to remember that the breath sounds are not inaudible, but may be almost normal or even of exaggerated distinctness. Usually clear, but rather distant bronchial breathing is heard. At the upper border of the dulness, loud bronchial sounds and ægophony are heard. When the exudate recedes, a friction rub

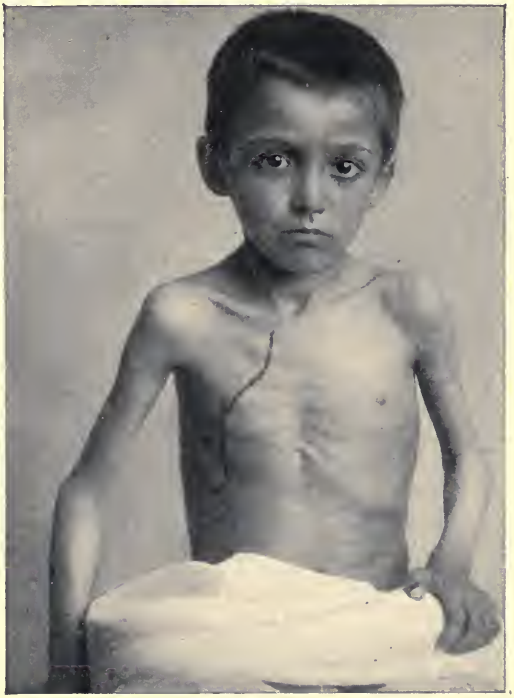


Fig. 106—Empyema of the left pleural cavity, distention of the left side of the thorax with loss of the intercostal spaces, heart and mediastina pushed to right.

or fine crackling râles become audible. The filling up of Traube's space cannot be considered a symptom in childhood.

The course of empyema depends upon the extent of the disease process. Small collections of pus are resorbed and leave a fibrinous layer upon the pleura. With larger collections spontaneous recovery does not take place. In time a rupture into the lung may occur, with expectoration of large quantities of pus without, however, emptying the abscess completely. In other cases an empyema necessitatis, with rupture through the thoracic wall, results. Death may occur at any stage of the empyema either from insufficiency of the lung, cardiac failure, cachexia, or finally from the spread of the infection to other body cavities and especially to the pericardium.

The prognosis of empyema depends, to a certain extent, upon the type of the infecting micro-organism. Comparatively speaking, the prognosis of pneumococcus empyema is favorable, while the streptococcic form may cause very high fever, typhoidal symptoms, peritonitis and sepsis. In the latter form the pleural sac refills rapidly after removal of its seropurulent exudate. Empyema due to putrefactive organisms, such as may appear after gangrene of the lung or typhoid pneumonia, is not very acute but is very obstinate. In this type, pyopneumothorax may be produced by gas formation. Tuberculous empyema may also lead on to the latter form, but this is a comparatively rare occurrence and its prognosis is unfavorable.

SEROFIBRINOUS PLEURISY

Serofibrinous pleurisy, like empyema, resulting from an acute infection, may have an insidious onset. The patient does not recover from the acute disease, has no appetite, becomes emaciated, and develops an irregular fever. Pain is felt only in cases of rapid onset and is excited by coughing. The signs of percussion and auscultation as well as misplacement of organs are the same as those in empyema. Probably a larger proportion of the serous exudates than is commonly supposed are due to tuberculosis. If the tuberculin reaction is positive, this etiology is always the more probable, even though tubercle bacilli cannot be demonstrated in the exudate either microscopically or by animal inoculation.

Rheumatic affections may be reckoned as second in importance as a cause of serous pleurisy. In older children serous exudates occur in association with acute articular rheumatism or as sequelæ of angina. In these cases, also, the exudates are free from bacteria. They are often bilateral. It is difficult, sometimes, to determine whether these are really transudates or exudates. They do not as a rule become very large.

The course of serous pleurisy is much more benign than that of empyema. Death results but rarely. The exudate usually disappears after several weeks or at the most within a few months; nevertheless, recovery is hardly ever complete, adhesions and thickening of the pleura usually remaining. These are found for the most part posteriorly and over the lower lobes, where, by an area of slight dulness and by restricted respiratory action, it is possible to demonstrate their existence for years. In other instances, retraction may ensue which draws the mediastinum and the heart toward the affected side and in children may result in scoliosis and serious malformation of the thorax.

Diagnosis.—The diagnosis of simple fibrinous pleurisy may be made by friction sounds. Relatively, this condition is of little importance. It is of large consequence, however, to be able to recognize the exudative pleurisies, because recovery and life itself may depend upon proper treatment. The thorax of every child with respiratory disease should be carefully percussed. Every marked degree of dulness should awaken the suspicion of pleurisy first of all.

If increased bronchial breathing and crackling râles are heard over the area of dulness, it is safe to conclude that there is consolidation, which sug-

gests either pneumonia or tuberculosis, depending on the course of the disease. If there is an absence of bronchial breathing, the suspicion of pleurisy is strengthened. Nevertheless, emphasis must again be placed upon the fact that diminished breath sounds are not invariably found over an exudate. Normal or even bronchial breathing may be heard over the pleurisies of children. The most important questions are: Is there an exudate? Is it serous or purulent? Exploratory puncture should be made in every case in which the slightest doubt remains. The diagnosis cannot always be made from the physical signs alone, and frequent surprises are met in cases in which infiltration has been definitely diagnosed,

Fig. 107—Exploratory puncture of the pleura.

but in which the needle brings forth fluid. It may happen, of course, that blood is aspirated if the needle is passed into the lung, or that the puncture gives no results at all when the point is imbedded in a thickened pleura. In case of only a small circumscribed area of dulness, it is unnecessary to explore, because little depends upon the result. Small pleuritic exudates, even pus, do not require operation: they are resorbed spontaneously.

Exploratory puncture is best done below the scapula at a point where thoracotomy may be performed later if necessary. Occasionally it may be found necessary to puncture anteriorly, then care must be taken not to injure the pericardium, liver, or diaphragm. It is best to mark the site of the intended puncture, determined by careful percussion, by pressing a finger ring firmly against the skin over the spot. The red mark of the ring remains visible for several minutes and cannot be washed off. No anes-

thetic is required for the operation. The wound may be closed by means of collodium and cotton, or with adhesive tape. The trocar should be large enough to permit the passage of thick purulent fluid. If the fluid is of a serous nature, no operative procedure is required, unless the exudate is on the left side and causes marked signs of compression of the heart. In this event a larger needle may be passed at the same point and the fluid drawn out with a syringe or a Dieulafoy aspirator.

If the exploratory puncture reveals purulent fluid, steps must be taken to remove the exudate as completely as possible. The most satisfactory measure for this purpose is thoracotomy with partial resection of the ribs. Puncture in the intercostal space with drainage is less satisfactory, since the drain may be compressed by the ribs which will obstruct the flow.

Irrigation of the cavity has little value; in fact, it seems to retard healing in some cases. Partial resection of the ribs is carried out under anesthesia. A longitudinal incision is made over the rib immediately above the puncture. The periosteum is pushed aside and a semicircular piece is taken out of the rib with rongeur forceps having curved jaws. A fine forceps carrying a suitable drain is then forced through the periosteum and the pleura into the cavity. The drain is fastened in place in the usual manner and closed at its outer end. If the skin incision is small, it is sometimes possible to make all the pus flow out through the drain, which is opened for the purpose once or twice a day. Usually pus flows along the outside of the drain into the dressings, necessitating frequent change.

Even after thoracotomy, the prognosis of empyema is not very good. About twenty per cent. of the cases die and during the first year of life this percentage is even higher. Nevertheless, the prognosis is better with than without operation, so that failure to perform thoracic puncture and to withdraw the pus must be considered an error in treatment.

TREATMENT OF SEROUS PLEURISY

In mild cases of serous pleurisy the treatment should be expectant.

The salicylates, digitalis (0.1-0.3 gm.), theobromine sodio-salicylate (2.0-5.0 gms.) for a period of two days, together with the local use of iodin ointment or the inunction of volatile oils, may be given. Codein may be used to quiet the cough. During the acute stage, moist applications, either hot or cold, as may be the better borne, may be used. The exudate is removed only when the signs of cardiac compression become marked. (See Fig. 106.)

Orthopedic procedures must be instituted early to overcome the deformity of the thorax. Creeping, gymnastics, swimming, etc., are advised, with general good hygiene.

V.

DISEASES OF THE HEART

BY

E. FEER,
Zurich.

REVISED AND EDITED BY

HENRIETTA ANNE CALHOUN, M. A., M. D.,
State University of Iowa.

Disturbances of cardiac rhythm are common in childhood. Tachycardia, more or less physiologic in young children, may reach a high degree in neuropathic patients, and is readily and temporarily caused by fever, excitement, etc. Even in older children of nervous temperament excitement or exertion may cause a marked increase in the pulse-rate. The apex beat may be more diffused while the pulse may become small and compressible without indicating by this transient disturbance, cardiac weakness or dilation. For the purposes of observation, mild exercise may be standardized as follows: (a) running up two flights of stairs twice; (b) slowly raising the body from a horizontal to a sitting posture ten times; (c) ten complete flexions of the knees while standing. If the pulse-rate which has been increased as a result of such mild exercise does not return to normal within three minutes, it is safe to conclude that there is a heart lesion.

The electrocardiograph may be used to differentiate benign from serious cases. The heart-beat originates in the upper part of the sino-auricular node and spreads in all directions over the auricle at a rate of about 1000 mm. per second. The left auricle contracts 0.013 second later than the right, and the ventricular contraction is 0.2 second later, the wave passing through the ventricle at a rate of 400 mm. per second. In the Purkinje fibres the rate is 2000 mm. per second. The contraction wave passes from the right auricle to the left auricle, through the auriculoventricular bundle to the ventricle. The interval between the right auricular and the ventricular contraction is the P-R time on the graph. Each contraction calls forth the full response of the heart muscle cells, and is followed by a period of rest. At the beginning of systole there is a short refractory period during which outside stimuli have no effect. The R wave on the graph is the ventricular contraction or the apex beat or the radial pulse time. At 72 beats per second the interval between the R wave is 0.6 second. Irregularities due to auricular and sinus variations in time are benign, while disturbances of the ventricular rate and rhythm, dissociation of auricle and ventricle, and auricular fibrillation are of serious import.

The electrocardiogram in a child shows some important variations from the adult type. In infants it is smaller, the S wave is unusually prominent gradually becoming smaller and reaching the adult type from the second to the twelfth month of extrauterine life. The Q wave is more frequently present in the new-born, and the P-R interval varies from 0.10 second in nurslings; 0.13 in early childhood; 0.14 at puberty; to 0.12-0.17 second in adults.

Paroxysmal tachycardia has been observed in older children. In several instances it has been hereditary. Complete recovery is possible.

Paroxysmal tachycardia of auricular origin and auricular flutter are very similar, both showing rapid coördinated auricular systoles, the contractions arising outside of the sino-auricular node. Both are reported in children but differentiation of the two is difficult because in children the auricle may exceed 200 beats per minute without any heart-block being present. The auricular rate in paroxysmal tachycardia is seldom more than 200-250 per minute, although 500 per minute have been counted on a polygraph record. (Hume.) Auricular fibrillation is a rare but very grave condition in childhood which is most frequently associated with cardiac decompensation and a history of rheumatic fever.

According to Hochsinger, compression of the vagus by enlarged bronchial lymph glands may result in permanent tachycardia.

Arhythmia is occasionally physiologic in young children, and may be demonstrated during sleep.

This is usually a type of respiratory arhythmia which becomes more marked from birth to puberty; children whose normal heart rate is rapid showing the more marked variation.

AVERAGE DIFFERENCE IN TIME BETWEEN THE LONGEST AND SHORTEST PULSE PERIODS AT VARYING AGES MEASURES IN $\frac{1}{5}\frac{1}{0}$ OF A SECOND. (Hecht.)

Age.	Time Interval.
Premature infants	2.5
New-born infants	3.4
Nurslings	2.8
Young children	6.25
Older children	8.4

In convalescence from infectious diseases, especially pneumonia and typhoid fever, arhythmia frequently continues for days or weeks. Usually this is not of much importance; but in diphtheria it is serious.[1] It frequently appears in the acute and chronic disturbances of infancy, especially in decomposition. Tuberculous meningitis at its onset, is frequently a cause of arhythmia. In older children it is often impossible to find any cause other than nervousness. When the pulse-rate is increased as a result of fever or exertion the arhythmia disappears. It is a frequent and quite harmless symptom which is by no means always attributable to myocarditis. It is usually dependent upon a lengthening of the diastole. Extra systole (Hirsch) often occurs with organic injury of the heart as in diphtheria, etc.

In children premature beats arising in the auricles are more common than those arising from ventricles, which is the reverse of the findings in adults. Extra systoles are more serious when they occur before ten years of age associated with other signs of cardiac disease.

The cardiac diseases of childhood cause arhthymia much less frequently than do those of adults, because chronic myocarditis is far less common in youth and because a severe degree of arteriosclerosis is practically unknown. Arhythmia is present occasionally in endocarditis and with the ordinary valvular lesions. Bradycardia is often associated with arhythmia. It is characterized by ineffective contractions which do not give a palpable pulse-wave, and is found in the same diseases as arhyth-

[1] Not infrequently this arhythmia in diphtheria is an atrioventricular rhythm recognized by a reduction in the a-c interval in the polygraph. (Wilson.)

mia, and especially with an infectious myocarditis incident to either diphtheria or scarlet fever. Occasionally bradycardia occurs during a recovery from appendicitis. In infancy slowing of the pulse is less common than in later years, and is often absent even in icterus and tuberculous meningitis.

The author has seen in a four months' old infant a pulse-rate of sixty to seventy, uninfluenced even by a temperature of 40° C. (104°F.). This condition was supposedly due to a myopericarditis. The case terminated in sudden death. In such cases heart-block must be taken into consideration.

So far only auriculoventricular and sino-auricular block have been reported in childhood. Most cases are due to acute inflammatory conditions, especially to acute rheumatic fever and diphtheria. Pneumonia, influenza and other infectious diseases are less frequent factors. Heart-block in acute infectious diseases may occur without any demonstrable structural changes in the auricular ventricular bundle. Congenital malformations may also be associated with heart-block, but septal defects do not involve the a-v bundle. Digitalis in large doses may cause heart-block. Syphilis infrequently or never produces heart-block in children. (Wilson.)

In the electrocardiogram the distance between the P wave (auricular systole) and the QRS group (ventricular systole) is lengthened with an increase in the a-c interval in the phlebogram, in heart-block.

HEART MURMURS

There are many cardiac conditions that involve changes in the heart sounds, as determined by auscultation, in which percussion findings are not necessarily changed. In infancy, the first sound of the heart is often dulled and even impure. The loud sounds characteristic of childhood are noted only at a later age.

Heart murmurs are extremely frequent. During the first two or three years they commonly indicate congenital lesions, because acquired changes are quite rare at this age and accidental murmurs are heard in exceptional cases alone.

The murmurs which arise in so-called functional insufficiency, as the mitral and tricuspid murmurs in uncompensated heart lesions, or those which follow the nephritis of scarlet fever, due to dilatation, are closely related to the murmurs of true valvular disease.

All other murmurs may be classified as accidental. These are more common at school age than during any other period, and they greatly exceed in frequency the murmurs due to organic valvular lesions. According to some authors these murmurs are found in fifty per cent. of all school children. Lüthje found slight systolic murmurs in three-fourths of children of school age. He attributes them to a relatively large pulmonary artery with a small ostium.

According to Hochsinger, accidental heart murmurs hardly ever occur during the first three years. But, in common with Thiemich, v. Starck, and others, the writer has often been able to demonstrate distinct murmurs in children of one or two years of age, in whom autopsy showed no structural cause and this, too, in cases in which the discovery of the murmur antedated the period immediately preceding death. It cannot be denied, however, that accidental sounds are comparatively rare during the first three or four years.

Accidental murmurs are characterized by their systolic synchrony, by their soft, sighing quality, and by their optimum recognition at the left border of the heart, that is in the region of the pulmonary, and only occasionally in the area of the mitral. The cardiac dulness is normal, the pulmonic second sound is not accentuated. The cause of these accidental murmurs has not been determined. Many authorities, among them Hochsinger, Potain, Herman Mueller and recently Schlieps, with whom the writer agrees, suppose that most of these sounds are cardiopulmonic —that is, that they are caused by the changes which the lingula of the lung undergoes during systole. The explanations, however, of the phenomenon differ essentially. The fact that these sounds are not at all constant speaks for their extracardiac and pulmonic origin. Furthermore, that they appear or actually become more distinct during excitement, in rapid breathing, or when deep inspirations are taken, and that they disappear upon forced expiration, or when the breath is held in expiration, or upon changes of position, are equally suggestive facts. They are heard more frequently when the patient is standing than when he is lying down. The systolic sound is never completely obscured by the murmur which begins in the middle phase of the systole.

A minor number of accidental murmurs seem to be due to a transient insufficiency of the valvular papillary muscles of the mitral, without lesion of the valve itself and without cardiac dilatation, as in the group of relative insufficiencies. Many of the murmurs occurring in the febrile diseases such as those of acute myocarditis come under this head. The sounds temporarily disappear with an increase of nervous excitation or of muscular force. Schlieps calls these murmurs, to the existence of which the writer has called attention in earlier editions, atonic heart murmurs. They occur principally in large, flaccid, neuropathic girls and in cachectic convalescents from typhoid and scarlet fever. With this type the systolic sound is entirely lost. The murmur often disappears when the abdominal aorta or the femoral artery is compressed (Schlieps).

In still other cases, accidental murmurs may be dependent upon an increased rate of flow or upon a diminished density of the blood. Sahli maintains that these causes frequently obtain in adults. Murmurs which occur in fever under increased cardiac activity, in the anemias of late childhood, and particularly in those cases in which a venous hum may be detected, probably belong in this group.

From this discussion the reader will recognize the difficulty of determining the cause of a systolic murmur in certain cases; especially since in the mitral insufficiencies of childhood the accentuation of the pulmonic second sound and the dilatation of the right heart may not appear for a long period.

In the differentiation of questionable cases these especially distinctive points may be applied, but they do not always enable us to reach a definite conclusion. Persistent observation gives one the best results and proves how frequently in older children the diagnosis of organic valvular lesions is made upon insufficient data.

A split pulmonic second sound is often heard in healthy children when they are under the influence of excitement, or when they are crying or straining, the congestion of the lung causing an earlier closure of the pulmonary than of the aortic semilunar valve. Accentuation of the pulmonic second sound may be observed under the same circumstances, but has no significance.

Venous murmurs are often heard in older children. Apart from the venous hum which may often be heard in anemic patients upon auscultation, with careful avoidance of pressure, over the veins in the neck, there are weak, long-drawn out or even continuous murmurs, traceable to the large veins, which can be heard oftentimes on both sides of the sternum. On the right of the sternum alone, a particularly loud murmur, with its maximal audibility below the aortic area, may be discovered. This murmur again is long-drawn out, almost continuous, often of very striking quality, and loudest in systole. It is possible that this sound, which is variable and most commonly heard in anemic patients, arises in the superior vena cava. Resembling this sound, demonstrated over the sternum, is a note which in tuberculous patients is produced by the pressure of enlarged bronchial glands. In the tuberculous and the non-tuberculous alike, such an intrathoracic note may be developed by bending the head backwards as far as possible. (Eustace Smith.)

When the diagnosis of an organic heart lesion has been made, there remains the difficulty of deciding whether the injury is congenital or acquired. In addition to the facts already recited the following points bear upon this differential question. A loud, rough, musical murmur, with normal area of dulness; or a murmur present in infancy, with enlarged area of dulness and a weak apex beat (Hochsinger); or a murmur, absent at the apex, but prominent in the pulmonary region—indicates, in general, a congenital lesion. A very loud murmur, distinctly audible over the entire heart, but without purring quality, lends probability to the diagnosis of an open septum. A purring systolic murmur, with its maximal intensity at the upper third of the sternum, unassociated with distinct cardiac hypertrophy, is indicative of an open ductus arteriosus (Botalli).

A large number of causes of disease which play an important rôle in the adult are rarely present or entirely lacking in the child. Thus arteriosclerosis, which in the adult often leads to lesions of the aortic valves and even to aneurism, never develops in early life; nor are its consequences, by way of chronic myocarditis and angina pectoris, ever seen at this period. Injuries to the heart from the excessive use of alcohol and tobacco, or incident to obesity, are extremely rare in childhood.

NERVOUS DISTURBANCES

Primary nervous disturbances of the heart are very much less common than in the adult. Cardio-neuroses, due to thyreotoxic causes (Basedow's Disease), occur only occasionally in late childhood. Children hardly ever complain of palpitation before the sixth to the eighth year, even when

heart action is very much increased. Occasionally, long-continued masturbation may lead to palpitation and increased rapidity of the pulse.

Spasmophilia is a frequent cause of sudden death in young children; and death in laryngospasm is caused by sudden stoppage of the heart and, according to Ibrahim, by tetany of the heart and not by asphyxia. The diminished resistance of the heart in cases of exudative diathesis is discussed under the head of infectious diseases. The etiology of sudden deaths in this condition, which may occur even without infection, is not well understood. Since the heart muscle is often entirely normal, it appears to be rather a question of failure of the nervous mechanism. The greater instability of the heart in these cases must be caused by the general diathesis.

The cardiac disturbances of neuropathic children have been little studied. They are generally characterized by great instability of the pulse-rate. Severe pain and sudden fright may cause a slow and irregular pulse. Sudden death has occurred from these causes.

CONGENITAL HEART LESIONS

The relative frequency of congenital heart lesions challenges great interest, for the relative frequency remains even after the exclusion of disturbances so severe as to cause death within a short time after birth.

Anomalies of growth are the causative influence of a large proportion of these cases, a relationship occasionally shown by other coexisting malformations (hare-lip, cleft palate, etc.). Congenital heart lesions are found too commonly in myxedema and mongolism to be regarded as a matter of mere chance. Intra-uterine infection of the heart and the great vessels is a more frequent cause of congenital lesions than has been hitherto believed. It is very often impossible to differentiate between these two causative factors even at autopsy, since inflammatory conditions are easily added to prenatal malformations.

Many congenital heart lesions are associated with irregularities or perversions of the normal heart growth, into the discussion of which we cannot enter. The customary division of the primary simple cylindrical tube, the truncus arteriosus of the early embryonic heart, into the aorta and the pulmonary artery, the subsequent development of the left ventricle and of the interventricular septum, is so complicated a process that it is frequently disturbed and gives rise to most of the abnormalities of development. Atresia or stenosis of one or the other of these channels, or even transpositions of them, occur, in consequence of the early abnormal relation of the aorta to the pulmonary artery. Very often various anomalies appear in combination and others, such as the non-closure of the ductus arteriosus, are merely compensatory.

These several combinations make the diagnosis of the individual case much more difficult than the recognition of acquired lesions. Many physicians never attempt a diagnosis, but are content with the application of the term "*morbus cæruleus.*"

Cyanosis is, of course, a very frequent and prominent symptom of congenital heart lesions and is often noticeable immediately after birth. It is

of some prognostic value, since, generally speaking, infants who show severe cyanosis from birth rarely live very long. Children who are only slightly cyanotic, or those that do not become cyanotic until their first or second year, have a much more favorable prognosis. The cause of the cyanosis is not quite clear. Certainly pulmonary congestion does not always occur, as evidenced by the fact that there is no edema. Nor does the relative thickness of the walls of the veins readily permit their distension. Frequently an admixture of venous with arterial blood is a factor in its causation. Finally, hyperglobulia, a later accompaniment of congenital heart lesions, runs parallel in a measure to the cyanosis (6–8 millions). This condition, with its significant increase of the hemoglobin and its enlargement of the erythrocytes, is in the nature of a compensatory arrangement, whereby the strained oxygen supply of the organism is reinforced.

There is an intimate relationship between the degree of cyanosis and the oxygen unsaturation of the blood; (*i. e.*, the difference between oxygen capacity and oxygen content per 100 c. c. of blood). With an oxygen unsaturation below 8 volumes per cent., there is no cyanosis; with 8-13 volumes per cent. there may or may not be cyanosis, while with an unsaturation of more than 13 volumes per cent. there is always cyanosis. When the hemoglobin falls below 35 per cent. cyanosis is not found. In hyperglobulia compensating for the deficient oxygen unsaturation, the color is reddish, an erythrosis which should be sharply differentiated from the bluish color of a true cyanosis. (Lundsgaard.)

In some cases, even at birth, the cyanosis is so distinct, affecting the entire body—the skin and mucous membranes—that it is observed by the laity. In other instances only the lips, the ears, the finger-tips and the toes are slightly blue, so that the condition may not be noticed in a hurried examination. In time, the drumstick fingers (see Fig. 105), develop sometimes in a very marked degree. Then, too, there are cases in which, while the patient is at rest, no sign of cyanosis can be recognized; but in which it appears distinctly when the patient cries, or strains, or holds his breath. In this connection it must be observed that even healthy new-born babes may become cyanotic when they cry very hard. Finally, there are cases in which cyanosis makes its appearance only after months or even years.

Moreover, it must be remembered that there are many congenital heart lesions which never show any degree of cyanosis, so that it is quite inappropriate to apply to them the term *morbus cæruleus*.

At the outset the symptoms are not always distinct. Cardiac changes to be determined by auscultation may be absent or easily overlooked at this early period. Generally speaking, heart murmurs give the most important indications. Acquired lesions or functional murmurs are very uncommon during the first three years of life, so that the demonstration of a murmur in infancy may be said to predicate a congenital lesion. If the murmur is unusually loud, the diagnosis may be established upon this evidence alone (see below). In most cyanotic cases, however, percussion reveals an enlarged area of cardiac dulness. Its extension is most marked in the right field because the majority of these lesions involve the right side of the heart and are in the nature of pulmonic stenoses. Other defects, and particularly those of the septum, occur without evident enlargement.

Respiration is always increased in frequency and sometimes to the extent of dyspnœa. Catarrhal symptoms, due to vascular congestion, are common and are often the precursors of broncho-pneumonia—the most common cause of death in infants with congenital heart lesions.

Edema is comparatively rare and occurs late if at all, a fact explained by the thickness and hence the resistance of the vessel walls. There is far less tendency in the organism of the young, than in the adult, to respond to cardiac insufficiency by the development of edema—a peculiarity noted, also, in the acquired heart lesions of young children.

A common coincident of congenital heart lesions is a general hypoplasia, a retardation of physical and mental development. The condition may be readily understood. Such results of severe lesions may be seen even in childhood. In the absence of any disturbance of nutrition, the failure of normal development in itself often leads the physician to suspect a congenital heart lesion, a suspicion often confirmed later, when no heart symptoms are immediately apparent. The tendency to subnormal temperatures and cold extremities, common in children with congenital heart lesions, is explained by the insufficient decarbonization of the blood. Such patients are often inclined to be peevish and irritable.

The prognosis of congenital cardiac lesions is extremely variable. Some cases are inevitably fatal immediately after birth, or within a few days; while others are met with which do not affect the general health at all. Death is usually due either to cardiac insufficiency, to intercurrent infectious disease, or to broncho-pneumonia. Tuberculosis is a generally recognized cause of death in pulmonic stenosis.

Treatment consists in prophylaxis against dangerous complicating diseases, rather than in active measures against the heart lesion itself. The best possible care, warm clothing, avoidance of exposure to cold, and plenty of fresh air, are the essentials to be secured. Especial care should be taken to avoid the contagion of measles and pertussis. If cardiac insufficiency and increasing cyanosis are threatening, the usual heart remedies, and especially digitalis, together with inhalations of oxygen, are indicated. The effect of these remedies is usually temporary. Their use should be delayed as long as possible in order not to whip the heart unnecessarily.

1. DEFECT OF THE INTERVENTRICULAR SEPTUM

Roger's Disease

Defect of the wall between the ventricles is of common occurrence. It is always due to developmental error and for this reason other structural anomalies, such as hare-lip, etc., are frequently associated with it.

The defect is nearly always situated in the upper membranous portion of the septum. If it is extensive it produces no audible murmur. In most instances, however, the all important symptom is a very loud, rough, systolic murmur, audible over the entire cardiac area, with its maximal intensity on the left of the sternum at the second or third intercostal space (Roger). It is transmitted also to the back, but not to the carotids. If the opening is not too small, the pulmonic second sound is accentuated,

because the stronger left ventricle forces blood into the right and thus increases the pressure on this side.

The heart may remain of normal size for many years. Eventually, moderate dilatation and hypertrophy of the right ventricle may develop. Usually there is no cyanosis. The general health and the physical strength are usually not impaired, so that the anomaly is generally discovered by accident and the affected individual may live to old age.

The diagnosis, during infancy, is often made quite definitely from the rough systolic murmur heard over the entire heart area, with its maximal point to the left of the sternum. There is no purring quality to the murmur and no increase in the cardiac dulness. The distinct or accentuated pulmonic second sound and the absence of cyanosis differentiate the condition from pulmonary stenosis.

The diagnosis becomes much more difficult when the same symptoms are initially discovered at the age of four to six years. At this period, acquired mitral insufficiency becomes more and more common and gives similar signs on auscultation and percussion. The maximal location of the point of intensity of the murmur at the apex, the lesser diffusion of the sound and its diminished roughness, indicate mitral insufficiency.

Very frequently the open septum is associated with congenital pulmonary stenosis or a patent ductus arteriosus, or with both of these anomalies at the same time; so that an exact diagnosis often meets with insuperable difficulties.

A permanently patent foramen ovale is a rather common finding. It may be present without causing any disturbances producing symptoms. Theoretically, an auricular diastolic, *i. e.*, ventricular systolic murmur, should appear, but in reality this is hardly ever observed.

2. PATENCY OF THE DUCTUS ARTERIOSUS (Botalli)

The closure of the ductus arteriosus, normally completed during the first month, may be delayed by disturbances in the pulmonary circuit, usually in the form of atelectasis, or by cardiac lesions. This failure, in association with other anomalies, is not uncommon.

When this patency occurs alone, the open ductus causes a systolic murmur increasing during the first year. Its maximal intensity is in the pulmonary area and is probably caused by the meeting of the currents from the pulmonary artery and the aorta (see Figure 108). The pulmonic second sound is always accentuated because the aortic pressure is superadded to it. The systolic murmur is transmitted to the carotids from the aorta. In older patients a palpable thrill may often be detected in the pulmonic area and in the jugulum or aortic arch.

When the condition has persisted for some time the pulmonary artery usually becomes dilated and gives a characteristic dulness, some finger's breadth in width, situated to the left of the sternum, which is shown as a shadow in the Roentgen picture. Later the right ventricle may be dilated also.

In uncomplicated cases the general health remains for a long time unim-

paired. Usually there is no cyanosis. Later a tendency to catarrhal affections shows itself, but the individual may live for some decades.

Diagnosis may be made from the above picture. Where a combination of lesions exists it is often quite impossible.

3. PULMONARY STENOSIS

This is the most common of congenital heart lesions. It has been estimated that three-fifths of the cases observed during the first year, and as

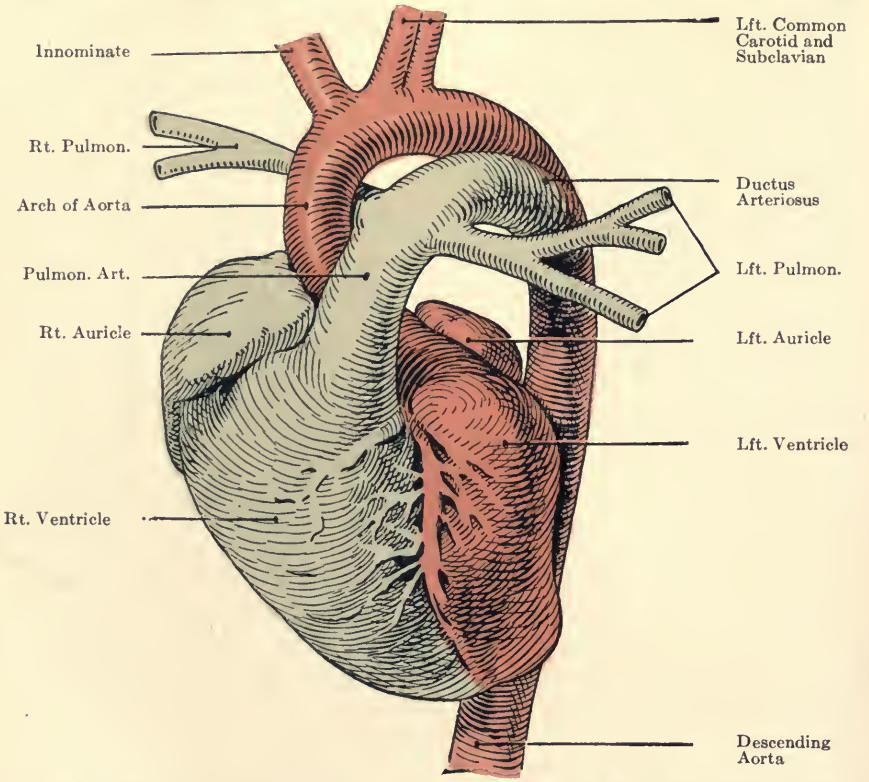


FIG. 108.—Heart of new-born infant. The ductus arteriosus (Botalli) is still open, and the different mixtures of blood in the arch of the aorta and descending aorta are shown (Kollmann).

many as four-fifths of those of later discovery, are of this type. Longer life is compatible with pulmonary stenosis than with any other congenital heart lesion. It is true that life is sustained by the compensatory influence of other lesions which take care of the blood-supply to the lungs. Thus in cases of patency of the ductus arteriosus, of so frequent occurrence, the lung receives its blood supply in part from the aorta; and in the event of an open septum the blood which cannot flow through the narrowed pulmonary orifice passes into the left ventricle and thence into the aorta. Complete atresia of the artery is rare. The stenosis may be situated at the

mouth of the vessel, or beyond it in the conus arteriosus, or outside of this in the pulmonary stem.

Symptoms.—A systolic murmur is heard in the pulmonic area and the pulmonic second sound is weakened or absent. The first sound is indistinct. If the stenosis is marked, the murmur may be wanting. If there is a coincident defect of the septum the blood flows from the right to the left ventricle and thence into the aorta, and thus the murmur may be transmitted to the aorta and the carotids. If the pulmonary stenosis is combined with patency of the ductus arteriosus the second pulmonic sound may be accentuated while the remaining sign of the open ductus, a purring murmur markedly transmitted to the carotids, is present. In the course of some years a considerable dilatation and hypertrophy of the right ventricle may develop.

Generally there is marked cyanosis, increasing whenever the child cries. The patient may become a very dark blue indeed. The cyanosis is often noticed immediately after birth. In time, an extreme degree of the drumstick fingers develops. The general health is disturbed by dyspnœa, by a tendency to pulmonary catarrh, to fainting spells, choking, and dizziness. Death often results from respiratory disease, associated with edema, with special liability to pulmonary tuberculosis.

4. AORTIC STENOSIS

Aortic stenosis is much less common than either of the three lesions already reviewed. The constriction occurs near the mouth of the vessel and gives symptoms resembling those of acquired aortic stenosis. If the construction is of very marked degree the patient seldom lives longer than a few weeks and during this period is simply kept alive by the flow of blood from the pulmonary artery through the patent ductus arteriosus.

Stenosis of the isthmus, in which a constriction occurs in the region of the opening of the ductus arteriosus into the aorta (isthmus aortæ, Fig. 108), is a matter of more important study, because it is compatible with long life. This stenosis is hardly ever extreme and is readily compensated by hypertrophy of the left ventricle. A systolic murmur is heard at the upper part of the sternum, in association with which, unlike pulmonary stenosis, there is no diminution of the pulmonic second sound. Later in life a characteristic collateral circulation is established. In its development the internal mammary artery, the intercostals and other vessels are markedly enlarged, in order to supply this region normally receiving its blood from arteries arising below the arch of the aorta. These enlarged vessels become palpable and visible. In comparison with the greatly congested carotids and the arteries of the upper extremities those of the lower extremities are poorly filled.

5. TRANSPOSITION OF THE GREAT VESSELS

This anomaly is rare. In this event the aorta arises from the right and the pulmonary artery from the left ventricle. Its chief symptoms are the absence of murmurs, a high grade cyanosis, and an accentuation of the

pulmonic second sound. Life is sustained only through the agency of associated defects of the interventricular septum. The author has seen a case of transposition, with defect of the septum, which reached the age of five years.

Among other congenital heart lesions, tricuspid stenosis should be mentioned. Mitral lesions in combination with other anomalies of the heart are not uncommon. Occasionally the heart is found in a mesial position. A congenital idiopathic hypertrophy of the heart, causing sudden death during the first or second year, and associated with enlargement of the thymus, has been described several times. The author has seen rare instances of idiopathic hypertrophy, symptomatically showing marked pallor, attacks of dyspnœa, a small and very rapid pulse, and terminating fatally, in which it was not possible to say whether the huge heart was congenital or acquired.

ACUTE ENDOCARDITIS

If acute endocarditis is of prenatal origin, its results are generally localized at the junction of the right ventricle and the pulmonary artery. It has been already said that congenital heart lesions depend, in part, upon inflammatory changes and that those which are definitely known to be due to anomalies of development, tend in postnatal life to inflammatory complications. In the latter event, the diagnosis, during life, cannot be definitely made.

If, following the ordinary custom, the inflammatory diseases of the heart are discussed as separate entities and under the terms of endocarditis, myocarditis, and pericarditis, it must not be forgotten that in actual experience these distinctive forms of disease are more rare than general carditis.

Etiology and Occurrence.—The acquired form of acute endocarditis is very rare before the fifth or sixth year. After this age it becomes more common and has its maximal frequency between the tenth and fifteenth year. In fact, three-fourths of all cases in youth may be traced to acute rheumatism, while in adults from one-third to two-thirds of the number are attributable to this infection. The infrequency of the disease before the fifth year is explained by the fact that rheumatism itself is uncommon before that time. But very few children who are affected by rheumatism and chorea, coincidently or successively, escape endocarditis entirely. Those who suffer from chorea alone apparently stand a better chance of avoiding the heart complication (Weill).

Next to rheumatism and chorea, scarlet fever is the most common cause of the endocarditis. This, contrary to the reports of Pospischill, seems to be quite definitely established. All the other contagious diseases are occasionally causative and, among them, diphtheria, angina, tuberculosis and erythema nodosum should be especially mentioned.

Attention should be called to the fact that rheumatism, in childhood, even though it be ever so mild and clinically not well defined, tends to endocarditis much more readily than it does in the adult. Accordingly, endocarditis is almost always secondary. A seemingly primary attack is

generally traceable to rheumatism or tuberculosis. Various micro-organisms must be considered in its etiology. The most common is the streptococcus. In some insidious cases the streptococcus viridans has been found (Schottmüller). Next in frequency are the staphylococci and the pneumococcus; then the typhoid and colon bacilli;[2] and occasionally the gonococcus. The organism responsible for rheumatism is still unknown.

The pathologic anatomy of endocarditis does not differ essentially from that in the adult. The changes in the valves are in the nature of fibrinous varicosities, leading either to constriction or ulceration. In the child, slight structural changes may be entirely repaired while the ulcerated form is very rare.

Symptoms.—If endocarditis does not develop during or following a recognized disease, it is often the first manifestation of acute rheumatism which has been ushered in often by an unobserved angina. If the initial causative disease does not obscure the characteristic picture of acute endocarditis, listlessness, pallor, nausea, and loss of appetite appear at its onset. Usually an irregular, often remittent fever develops, which does not range very high and may sometimes be so slight that it is discovered only upon repeated use of the thermometer. Slight subfebrile temperatures, continuing for weeks after rheumatism or scarlet fever, are not infrequently seen. When the patient is put to bed this temperature disappears, only to reappear when he is allowed to leave the bed. In this condition, all the signs of endocarditis, that is of a valvular lesion, very gradually develop.

In the child the mitral valve is by far the most commonly affected and initially its aortic flap. At the onset the first sound is diminished at the apex. Soon it becomes indistinct and is replaced later by a murmur. This is either of a blowing character, or of sighing, soft quality and sometimes is transmitted to the pulmonary and, less frequently, to the aortic area. The wonderful reserve power of the child's heart often makes it possible for the left heart alone to compensate the lesion for a long time, so that there is no congestion of the pulmonary artery and accentuation of the pulmonic second sound may not be noted for a long period.

The aortic valve is affected much less frequently, and secondarily to the inflammatory process in the mitral. This happens very commonly in endocarditis of long standing and with repeated attacks of fever. With this extension, a systolic murmur is at first heard in the aorta. A diastolic sound appears later when contractions have formed.

Tuley and Moore report a case of congenital endocarditis in a boy of 13 years of age, with a patent foramen ovale, where the pulmonary orifice was almost obliterated by pendulous vegetations and wart-like growths. Only three similar cases are recorded in 2400 medical admissions, with only .01 per cent. of 1050 cases of valvular involvement showing only the pulmonary valve involved. Very few are diagnosed during life.

Percussion may show normal cardiac dulness for a long time and even the Roentgen ray may not show any enlargement. Its absence is to be explained by the power of resistance of the child's heart, which shows a

[2] B. Influenza may be the causative agent especially during pandemics (Malloch and Rhea).

large measure of reserve strength and undergoes dilatation and hypertrophy at a much later period than in the adult.

The pulse is always rapid, but commonly shows none of the irregularity which usually indicates myocardial disease. In infancy the customary picture varies so markedly (Lempp–Finkelstein), that a diagnosis is hardly ever made—a failure the more likely because the disease is extremely rare at this age. At this early period the chief causative factor is usually sepsis, quite often arising from an unimportant rhinitis. The local symptoms often fail the diagnostician in these cases, since no murmur is audible throughout the course of the disease and the enlargement is observed only a little while before death.

Following the febrile onset, great pallor develops which may pass into cyanosis. From time to time, in fact, serious attacks of cyanosis appear. The respiration becomes more and more rapid and deep, without any evident cause in the lung itself. Clinically, the disease-picture resembles that of miliary tuberculosis very closely and the differentiation is difficult. Most cases occurring in infancy are fatal within a few weeks.

Its course is variable. Save in infancy, the disease terminates fatally only if it takes on an ulcerative form, or if it is complicated with pericarditis or myocarditis, and particularly with the former. Usually a chronic valvular lesion results. Differences of opinion still obtain as to the possibility of complete recovery with full restitution of the injured valves to their normal state. With other authors, the writer is fully convinced that he has often seen recovery from mitral endocarditis. In such an event, the murmur gradually disappears after some weeks. There is, then, no reason to suppose that muscular insufficiency always ensues in these cases. Of course, with recovery it is hard to prove that there has been a true endocarditis.

Ulcerative endocarditis is much less common in the child than in the adult. This is a really surprising fact, because sepsis is of so frequent occurrence in the early years of life. It is explained, perhaps, by the integrity and good nutrition of the endocardium. This form of the disease may seem sometimes to be of primary origin and again it may occur as a sequence of simple valvular lesions or in combination with septic disease of the mouth or bladder. Its clinical picture, strongly resembling typhoid or general sepsis, is sharply defined. The disease is ushered in by restlessness, chills, and irregular temperature, followed by delirium and other grave cerebral symptoms. The picture is not completed, however, until hemorrhagic and purulent emboli in the skin reveal its true septic character. With the exception of the greatly accelerated pulse and a slight cyanosis, the heart signs may be negative in its early or even later history. Occasionally, the presence of murmurs and of acute dilatation announces the correct diagnosis. Death results in from one to three weeks.

The diagnosis of acute endocarditis offers many difficulties. First of all, the numerous accidental and muscular murmurs that occur in febrile diseases must be excluded. If careful daily examination shows a gradually increasing and persisting murmur, constant in character, appearing in the

course of some other disease, the diagnosis of endocarditis may be accepted. The differentiation from pericardial sounds is made as it is in the adult.

The very small, delicate nodules, which are frequently found at the free edge of the venous valves in children during the first years of life, must not be mistaken for endocardial exudates. These Albini's nodules may be very numerous. Formerly, they were erroneously described as inflammatory processes in autopsies following deaths from diphtheria. In the new-born, discrete, dark violet hematomata, of pinhead size, are quite commonly found on the valves. They represent capillary ectasias and disappear as the valves become more vascular. They are of no significance whatever.

The prognosis is clear in the light of the facts already cited. Prophylaxis must be directed against angina, among other things, and care must be taken to see that even the mildest and seemingly unimportant cases of acute rheumatism receive a thorough course of salicylates.

Treatment.—In the matter of therapy it is well to begin with a course of salicylates in any case where rheumatic symptoms exist or appear (see Acute Rheumatism). As long as the heart is normal this therapy can certainly do no harm and may protect the heart. As soon as acute endocarditis is suspected, the patient must be kept in bed and treated in every way as though the disease were actually established. The child should be kept as quiet as possible and avoid all unnecessary motion. He should not be permitted to sit up even to take food. With fever and greatly accelerated heart action it is customary to apply an ice-bag. The author has never been convinced, however, of its usefulness, unless it be that, at times, it enables the child to be still for a longer period. With the infant, and particularly when there is no fever, it should certainly be omitted. Tepid baths carefully given are to be preferred. If there is great restlessness and a sense of fear, codein and, in older children, morphin, give required rest and beneficial sleep. Ordinary cases will usually do without cardiac stimulants, such as digitalis, etc., although they must sometimes be considered. In severe cases such stimulants as camphor and caffein cannot be avoided (see page 416).

The food should be fluid, or semifluid in character, consisting chiefly of milk, soups, thin flour puddings, fruit juices, mashed potato and soft stewed apples. Coffee, tea and alcohol should not be used.

CHRONIC ENDOCARDITIS AND ACQUIRED VALVULAR LESIONS

Valvular lesions are almost always the results of acute endocarditis and, therefore, indirectly of infectious diseases. The exact terminus of an acute endocarditis is hardly ever determined clinically. The possibility of its transition into an insidious chronic endocarditis is always to be taken into account.

According to a number of authors the primary chronic endocarditis of tuberculosis, developing at puberty, usually produces a pure mitral stenosis. In childhood arteriosclerosis, as a cause of heart lesions, is a negligi-

ble matter. By far the most common cause is acute rheumatism; the endocarditis associated with it, preceding the recognition of a valvular lesion, often runs its course unnoticed.

Valvular lesions occur almost without exception, only after the fifth year. The very occasional departure from this rule is noted between the second and fourth years.

The Clinical Picture.—Mitral lesions are by far the most common in childhood, exceeding aortic disease fifteen or twenty fold. Aortic lesions seldom appear until puberty. The clinical picture is governed, therefore, in large part by these mitral lesions and of these mitral insufficiency is much the more prominent. A pure mitral stenosis is found but rarely and then only in older children.

It is a quite characteristic fact that many heart lesions remain wholly latent for years and often only manifest themselves toward puberty. This is true in about one-half of the cases observed. The physician discovers the condition quite accidentally in the course of a general examination. The parents have not suspected any trouble with the child's heart or even that he is not so strong and healthy as other children. He has attended school and engaged in gymnastics, marching and other exercises. Frequently, however, the patient is found to be a little irritable; he tires easily, is pale, and now and then complains of headache and has frequent epistaxis. Seldom is his physical development delayed and this generally in the rare event of mitral stenosis.

Upon examination a distinctly visible and palpable apex beat is observed. It is to be noted that the patient himself does not notice the forcible beat and that the young child hardly ever complains of palpitation. If the condition has existed for a long time bulging of the thorax in the cardiac region may be observed, particularly in the very young patient or in initially severe forms of the disease. Many cases, however, show no deformity whatever. Percussion may reveal, for years, a quite normal relationship and at the most only a slight enlargement of the heart dulness to the left, as a result of a dilatation of the left ventricle which has occurred early and completely compensates the lesion for a long period. The nutritive integrity of the heart of the young child; the absence of alcoholic injuries and of arteriosclerosis; the low blood-pressure and freedom from the overexertion, unite to make complete compensation possible for years and to postpone congestion of the pulmonary or systemic circulation.

The murmurs are at times distinct or may be rough and sharp. They are heard not only in the usual areas but always, in the very young and generally in older children, over the back between the scapula and particularly at its angle. In mitral lesions the sound is lower and in aortic cases higher than ordinary.

When the lesion is no longer compensated, evidences of congestion in the pulmonary and systemic circuits soon appear. Enlargement of the liver, cyanosis, bronchitis, dilatation of the peripheral veins, a true venous pulsation due to relative insufficiency of the tricuspid valve, effusion into the various body cavities, and edema are the chief results. The heart is

notably dilated, especially upon the right side, so that upon first examination one may suspect the presence of a pericardial exudate. At first the pulse is but rarely arhythmic and, even later, if the myocardium is not affected arhythmia may not appear.

The symptoms of the various valvular lesions are generally similar to those that are seen in the adult and only a few points require mention.

Mitral insufficiency is by far the most common of these lesions and frequently occurs independently. The accentuation of the pulmonic second sound and a dilatation of the right ventricle may be absent for a long time.

Mitral stenosis generally follows upon mitral insufficiency after it has existed for some years. In these cases it is often found that the original systolic murmur has disappeared, being entirely replaced by a diastolic murmur with fremitus. The author has never seen a true primary stenosis develop until puberty. As in the adult the murmur may be audible only after exertion.

On the aortic side, insufficiency and stenosis are usually coincident. The valve is rarely affected until the tenth to the twelfth year. This form of disease is often of severe degree and dangerous. It may terminate in sudden death.

Tricuspid insufficiency is seen in association with severe mitral lesions in which compensation has failed, causing dilatation of the right ventricle.

Course.—From the writer's personal observation, in harmony with the reports of many recognized authorities, it may be said that in acquired valvular lesions, and especially in the slight mitral insufficiency of childhood, there is the possibility of complete restitution. This remains true even in cases in which the diagnosis is very well established (see page 391); and when only such cases are considered where a murmur has appeared very gradually, in connection with acute rheumatism, and has remained unchanged for months or even for a year or two, it has been known to disappear gradually and spontaneously. In most cases, of course, the lesion once developed remains, even though it may be fully compensated and may create no symptoms for many years and usually not until puberty, or even later. Often, however, serious disturbances arise, after many years of undisturbed well-being. These may be due to the gradual weakening of the heart muscle itself, or to the fact that the heart cannot respond to increased demands made upon it, or to an aggravation of the heart lesion incident to renewed attacks of endocarditis.

Frequently the disease proves fatal within a few months or years. In these cases, however, the preëxisting heart lesion is hardly ever responsible for the calamity, the cause of which is usually to be found in a supervening and often obliterative pericarditis, with an associated myocarditis.

In general it may be said that valvular lesions are compensated more fully and for a longer time in children than in adults. When, however, evidence of compensatory failure once appears the condition is always a serious one and death results more speedily than in older persons. Sometimes the dilatation of the heart and the congestion of the liver are enormous.

Diagnosis.—The diagnosis of valvular lesions is to be made in the same manner as in the adult, excepting for the fact that recognition of mitral insufficiency, difficult in older persons, offers insuperable obstacles in the child. Accidental systolic murmurs (see page 390), occur so commonly between the ages of five and fifteen, that we can hardly be careful enough in making a diagnosis of mitral insufficiency where the classical symptoms are not distinct and when the murmur is not loud and rough. Since in the mitral insufficiency of childhood the accentuation of the pulmonic second sound and the enlargement of the right ventricle may appear very late and, furthermore, since recovery is possible, the diagnosis becomes all the more difficult.

Next to a continuing systolic murmur in the mitral area, the lift and the resistant quality of the apical impulse, unchanged with the changing position of the body, and indicating hypertrophy of the left ventricle, is to be considered a symptom of the greatest importance. The attention of the reader has already been called to the similarity of the symptoms of mitral insufficiency and those of a congenitally open septum (see page 396). In anemic school children, the absolute cardiac dulness is often enlarged; and the coincidence of anemic murmur may easily lead to the mistaken diagnosis of an organic lesion if one does not realize that the enlargement of the absolute dulness, with unchanged relative dulness, may be due to a diminished expansion of the borders of the lung.

The prognosis is already clear. Prophylaxis should consist, first of all, in the avoidance of attacks of angina or acute rheumatism, by the observance of a general hygiene and especially by careful conservation of the heart. The very mildest case of rheumatism should be carefully treated by rest in bed and the use of salicylates.

Treatment.—An existing heart lesion which is fully compensated requires no treatment. If the physician accidentally discovers such a lesion he should inform the parents, without directing the child's attention to its presence. It is not necessary to change the patient's mode of life. Severe physical exercise, and especially bicycle riding, which is apt to have an injurious effect upon the heart, must be forbidden. Alcohol, coffee, and tea, all injurious stimulants, must be avoided. The patient should not select any means of livelihood which requires strenuous exertion. Considering the tendency of rheumatism and endocarditis to recur, all exposure to cold is to be safeguarded. Gentle measures tending to increase the patient's resistance, and the use of woolen underclothing in the cold season are to be recommended. Country life may serve to strengthen the child; a benefit to be gained equally, in the author's estimation, for those whose compensation is complete, from a long sojourn in the mountains, provided that strenuous hill-climbing is avoided.

When compensation begins to fail, long periods of rest in bed, with carbon dioxide baths, taken either at home or at some resort, may be tried. During the rest treatment, a special dietary consisting largely or exclusively of milk may be continued for seven or eight days with good results. If despite these methods there is continued failure or a want of full compen-

sation a systematic course of treatment with digitalis should no longer be delayed (see page 416). This drug and such similar remedies as camphor, caffein, morphin, etc., may be employed as they are in the adult.

ACUTE PERICARDITIS

Etiology and Occurrence.—Mild cases of pericarditis, in the form of slight exudates or small fibrinous placques, are very commonly found at autopsy following deaths due to the various infectious diseases of childhood. More intensive forms of pericarditis, of larger clinical interest, are also seen at this age. In the new-born and in young infants purulent pericardial exudates occur in the course of sepsis. These are commonly of streptococcic, and rarely of gonococcic origin. Even at a later period, as late, indeed, as the fifth to the seventh year, purulent exudates of the pericardium are quite usual, but they generally arise from inflammatory process of pneumococcic type in the neighboring lung tissue or pleura. Extension from peritoneal infections is less frequent. After the sixth to the eighth year, serous pericardial exudates become more and more common. These are due to rheumatism or tuberculosis, and undergoing resorption show a great tendency to obliteration of the pericardium. In rheumatism marked involvement of the pericardium usually occurs subsequent, only, to repeated cardial attacks. Accordingly in the child, both in type and origin, pericarditis presents a parallel to pleurisy.

Further it is to be remembered that at every age the several infectious diseases, scarlet fever, measles, erysipelas, etc., not infrequently cause pericarditis which, in its lesser degree, is often unrecognized and in more severe forms may develop purulent exudates, etc.

Pathologic Anatomy.—An exudate due to the streptococcus is usually of a seropurulent character. In pneumococcic infection, the exudate is less abundant, but both layers of the pericardium are apt to be covered with thick, ragged, fibrinous masses. In the tuberculous form there is a large amount of exudate, but comparatively few tubercles. The heart muscle is often enormously hypertrophied and dilated, a condition, however, usually resultant from rheumatism and due, in part, to valvular lesions.

Symptoms.—Acute pericarditis readily escapes notice when it is coincident with any serious primary disease; which, aggravated by the pericarditis, may even cause death without the recognition of the pericardial condition. It may be readily understood, therefore, that in sepsis or other serious infection, pericarditis may often go unobserved. It is equally true that in pneumonia or pleurisy the area of dulness incident to the primary disease covers that which may be due to pericarditis. Such a complication of pneumonia, by no means a rare occurrence in early years, is more often announced by the sudden aggravation of the general symptoms and the remarkably sudden failure of the pulse than by any physical signs.

In cases of purulent pericarditis alone making up the clinical picture, fever, restlessness, a sense of thoracic pressure, dyspnœa with very rapid respiration, a small and very frequent pulse, an anxious countenance, a

marked pallor with ashen color and, later, cyanosis occur. As in other forms of pericarditis sudden death may result.

In acute rheumatism, the picture of pericarditis is relatively clear. In the rheumatism of children, pericarditis is more frequently of early appearance than it is in the adult. Such general symptoms as fever, restlessness, headache and anorexia are first observed. In the course of several days, a slight friction sound may be discovered, perhaps, over the precordium. It is very soft and may resemble an endocardial murmur, but is of changeable quality; it is not transmitted and occasionally increases under the pressure made by the stethoscope. In contrast to an endocardial murmur, it is generally heard over the base. Simultaneously, or a little later, the respiration increases in frequency and becomes dyspnœic and sighing. The pulse grows small and frequent. Increasing pallor and restlessness emphasize the seriousness of the disease-picture, although complaint of thoracic or precordial pressure may still be lacking. Careful examination, however, reveals an increase of cardiac dulness in typical form, extending well beyond the apex to the left, peaking the area at the base and, a peculiar characteristic, widening well to the right and filling the cardiohepatic angle, a result not observed in simple dilatation of the right ventricle. The development of distinct dulness in Traube's space, below the apical impulse, when there are no indications of pleuritic effusion, is an extremely important point. Frequently friction sounds are heard in the precordial region when the exudate is large. This is readily explained by the fact that the abundant exudate forces the heart, which cannot be pushed backward, against the anterior thoracic wall. The greater part of the fluid gathers below, to the left, and to the right of the heart. If the exudate is very large, the left lung is compressed—a result which is indicated by dulness posteriorly and inferiorly, and by diminished breathing sounds, or bronchial breathing. These pseudopneumonic and pseudopleuritic manifestations are quite common. It often happens, however, that a pleural effusion develops prior to, or coincidently with, or subsequent to, a pericardial exudate. This may occur in the right, or the left, or in both pleural cavities; and if it is of early occurrence it may mask the evidences of pericarditis. The concealment is most apt to occur if the pleural effusion is on the left side. The apical impulse will often be felt longer than might be supposed. If the exudate is large a general undulation of the entire precordial region may be observed. Under these circumstances, and particularly in young children, this entire area may bulge if the condition has existed for a long period. The left half of the thorax lags in inspiration. A large exudate makes the work of the heart more difficult, especially in diastole. Such an excessive effusion occurs almost wholly in tuberculous cases and quite rarely in the rheumatic form.

The pulse in this condition may become smaller and smaller and may reach a frequency of 160 to 200. The congestion may cause increasing cyanosis, a distension of the veins of the neck, an enlargement of the liver which may be painful on pressure, and, occasionally, a general edema. In favorable cases the whole condition improves with the resorption of the exudate. If this does not ensue, the dyspnœa and the pressure symptoms

increase and death comes with heart failure and collapse after many days or weeks of suffering. Sometimes death occurs suddenly and unexpectedly.

The severity of the disease is often increased by a coincident rheumatic endocarditis. Even when the exudate is resorbed recovery is not necessarily assured. Quite often, and decidedly more often than in the adult, the disease results in more or less complete obliteration of the pericardial sac, which bars the possibility of convalescence and results in a long, tedious illness and ultimately in death (see below).

Tuberculous pericarditis pursues a course quite similar to that of rheumatic origin, excepting that it is much more insidious and hence of more gradual onset. It has a distinct tendency to the formation of a massive exudate, generally of a serous character. The disease commonly extends to the pleural and peritoneal cavities simultaneously. It often arises from so small a focus in the lung or the bronchial nodes that it appears to be primary in the serous membranes.

Diagnosis.—The diagnosis depends upon the same symptoms as in the adult. These consist of the large triangular area of cardiac dulness; the disappearance of the angle of resonance between the liver and the heart; and the filling in of Traube's space beneath the heart. The progressive increase of the area of dulness is especially characteristic, as is also the gradual approach of the absolute to the relative dulness. In a word, the absolute dulness enlarges more rapidly than the relative.

Occasionally in young children, when the exudate is small, a diagnosis is very hard to make. A fibrino-purulent pericarditis, associated with pleurisy and pneumonia, often defies demonstration. Roentgen examination is very conclusive in exudative pericarditis, showing a markedly enlarged, non-pulsating silhouette, with disappearance of the cardiohepatic angle. By means of the X-ray it is also possible to distinguish the extreme cardiac dilatation which may occur in older children in scarlet fever and other infectious diseases.

If the existence of an exudative pericarditis is definitely established, it is rarely difficult to determine whether the exudate is of a serous or a purulent character, and that even without a blood examination. If pus be present, a count will show a distinct increase of leucocytes. In early childhood a serous exudate is hardly ever seen, while later this is the predominant type on account of its usual rheumatic or tuberculous origin. At this early age the purulent forms are seen only in severe cases of infectious or respiratory disease.

An exudative pericarditis which is apparently primary will prove actually secondary either to rheumatism, to tuberculosis, or more rarely to nephritis. If endocarditic murmurs, with other coincident or earlier symptoms of rheumatism, are absent, the condition is usually tuberculous.

The prognosis of purulent pericarditis is almost inevitably a hopeless one. In any exudative form it is always very serious, since the disease frequently results in immediate death or in obliteration of the pericardium. In rheumatic cases it may be said that death usually results, directly or indirectly, from obliteration of the pericardial sac.

Treatment—The treatment of acute pericarditis, even in its purely fibrinous form, and most certainly in its exudative types, dictates absolute rest in bed. If there is reason to suspect its rheumatic origin, the salicylates should be given from the very beginning and will prove as useful as they are in the rheumatic exudate of pleurisy. To children of five years, 0.3 gm. (grs. v), of acetylsalicylate may be given, three times daily; while for children of ten years, 0.5-0.7 gm. (grs. viii-x), of acetylsalicylic acid, three times a day, may be substituted. With older children, an ice-bag may be placed over the heart if the fever is high. Often it has a quieting effect, but beyond this its value is rather doubtful. Later, applications of heat are to be preferred.

The patient's head should be kept high. All excitement and exertion are to be avoided. During the acute stage the diet should be light and readily digested, consisting chiefly of milk, gruels, soups, toast, rolls, eggs, apple sauce, and vegetables.

Cardiac weakness and increasing congestion require heart stimulants, in the form of digitalis, caffein, or camphor (see page 416). Morphin, by mouth or subcutaneously, is most serviceable if there is great restlessness and sense of thoracic pressure and will aid, also, in quieting the heart. Its use should not be too long delayed. If the pericardia exudate is very large, surgical interference must be considered. In the event of a purulent exudate, probably the only satisfactory measure is resection of a rib and drainage of the pericardium; but in view of the hopelessness of the prognosis it is a measure to which resort is very occasionally had. With massive serous exudates, however, paracentesis of the pericardium should be practiced more commonly than it is at present. The extra mammary method of Curschmann seems to the writer by far the best. An exploratory puncture is first made in the fifth intercostal space, at least one centimeter outside of the apical impulse, but in the area of absolute dulness. If the apex beat cannot be felt, the puncture is made just inside of the outer border of absolute dulness, the needle passing in a sagittal direction or slightly inclined toward the median line. If the exudate is reached, a larger but very sharp needle is inserted in the same place, permitting the fluid to pass off freely but as slowly as possible. The especially designed trocars of Curschmann may be dispensed with. The passage of the needle through the pericardium gives a sensation similar to that caused by pricking a distended bladder. The drainage of the exudate occurs more readily if the needle is connected with a fine rubber tube filled with salt solution, with the free end extending below the level of the body. This procedure is simple and, carefully performed, is quite free from danger and often achieves wonderful results in lifting the load from the compressed heart. The author has removed by this method 500 c.c. of fluid from the enormously enlarged pericardial sac of a six-year-old boy. In this instance there could have been no possibility of confusion of the pericardial exudate with a coincident pleural exudate, since the latter was of a different color.

PERICARDIAL ADHESIONS

The termination of pericarditis in the complete obliteration of both the visceral and parietal layers of the pericardium deserves special mention because it is relatively common in children between the ages of eight and ten years. In these cases the preceding acute pericarditis frequently goes unrecognized and the clinical picture is often overshadowed by symptoms other than those which point to the heart. Most of them occur in the course of rheumatic affections. Hypertrophy and dilatation of both ventricles are so great that the enlargement of the heart area seems to be fully explained, and a possible exudate, rarely of very great quantity, easily escapes observation. Second to rheumatism, tuberculosis plays an important rôle in the etiology of this condition. In this class of cases the valves are usually intact, the initial exudate is quite large, and the resulting obliteration of the pericardium is less readily recognized.

The pathologic findings usually show a firm knitting together and thickening of both layers of the pericardium throughout its entire area or over a large part of its surface. In recent cases, the gelatinous character of the exudate is shown, occasionally, in spots. If the process is tuberculous, tubercles or caseated masses may be found between the layers of the pericardium. Death, in cases of rheumatism, is usually a result of obliterative pericarditis, so that this is a very common finding at autopsy. At times the outer layer of the pericardium is adherent to the pleura, the sternum, or the mediastinum, the whole being converted into a dense cicatricial mass.

Symptoms.—Many cases are latent throughout their entire course and are discovered only at autopsy. This failure of diagnosis occurs chiefly in cases of rheumatic nature. In these patients it is often impossible to distinguish the symptoms which are consequent upon the existing heart lesion from those which relate to the pericardial adhesions. Valvular lesions and heart murmurs are almost always present; together with a marked and often enormous hypertrophy and dilatation of both ventricles, which press against a large area of the thoracic wall. A distinct impulse over the entire cardiac area and a bulging of the thorax are to be regarded as results of this hypertrophy. The apex beat is generally definite. A systolic retraction of the apical area is indicative of adhesions between the heart and the thoracic wall. This is not to be confused, however, with the more common systolic retraction of the intercostal space in the immediate region of a forcible apex beat (Romberg). The diastolic rebound of the intercostal spaces is pathognomonic, but it may be mistaken readily for the apex beat. It is apparent that signs by which obliteration of the pericardium may be recognized demand a very careful examination and, even at that, diagnosis may fail in the major number of cases.

The general symptoms of extreme pallor, superficial respiration, thoracic pressure, a small pulse, cardiac hypertrophy and dilatation, are just as common in endo- and myocarditis, or in cases of valvular lesion, as they are in pericardial disease. It follows that obliteration is to be suspected only when convalescence from pericarditis does not occur. In fact, the

child always becomes a chronic sufferer. As long as the patient is kept in bed, his condition remains fair, although the pulse is very small and frequent and the respiration becomes dyspnœic upon the slightest exertion. But as soon as the child is permitted to get up, the heart is found to be insufficient in spite of the enormous hypertrophy. The pericardial adhesions serve as a powerful obstacle to normal heart action and this obstacle is well-nigh insurmountable when the heart becomes attached to the sternum, a result which cannot be overcome by hypertrophy. The patient grows very weak, dyspnœic and cyanotic, and returns to bed of his own accord. Thereafter, periods of improvement and aggravation alternate; the child feeling fairly well for months at a time, or being able, perhaps, to go to school occasionally. Generally speaking, alike in the rheumatic and tuberculous forms, attacks of fever, with pleural exudate and peritoneal effusion, recur from time to time. The disease gradually becomes more and more aggravated and terminates with bronchitis and edema, as the results of cardiac insufficiency, after months or even years of time.

If the physician, and especially one who is inexperienced, has not observed the premonitory symptoms of pericarditis, his attention is often diverted from the heart by the frequent appearance of abdominal symptoms. Of these the most marked is the noticeable enlargement and induration of the liver, which is often tender to the touch. The hepatic border may be three or four fingers' breadth below the costal margin. Since this is often accompanied by enlargement of the spleen and ascites one may be tempted to suspect a primary cirrhosis of the liver. This is a very easy error to fall into, particularly when percussion and auscultation show fairly normal heart outlines, as happens frequently in tuberculous pericarditis. The mistake may be avoided if due attention is given to the usually evident cyanosis, to the small rapid pulse, the dyspnœa, and the readily demonstrated pleural effusion or thickening. Later on, in rheumatic cases, very marked cardiac changes, with a mitral murmur, tricuspid insufficiency, and dilatation are hardly ever wanting.

This so-called pericarditic pseudocirrhosis of the liver is probably to be considered as a symptom of congestion, in the causation of which the constriction of the inferior vena cava by the rigid pericardium plays a part.

This conception, however, is not to be considered as definitely established, for the liver lesion is quite frequently absent, while again it may occur independently of any cardiac affection. Nevertheless, exudative or fibrinous inflammatory processes in the pleural cavity and adhesions of the liver to its surroundings, as in tuberculosis, are found relatively often in relation to pericarditis. A symptom-complex due to chronic hyperplastic perihepatitis is also occasionally encountered. In many cases, and not uncommonly among older children, it seems necessary to assume that there is a form of polyserositis involving the pericardium and causing cirrhotic changes in the liver.

Diagnosis.—The difficulties in diagnosis of obliteration of the pericardium have been made sufficiently clear. In cases in which neither friction sounds nor evidence of exudate can be made out, the diagnosis must often

remain tentative. Even at autopsy the tuberculous nature of the disease cannot always be demonstrated.

In making a differential diagnosis true valvular lesions, with myocarditis and with indurated enlargements of the liver of varying etiology, in which lues is to be included, must be considered.

The prognosis is clear. The disease is fatal, it may be after weeks, months or even years of suffering. Partial adhesions only are compatible with long life but, antemortem, they are hardly ever definitely demonstrable.

Treatment.—Treatment is very unsatisfactory. Several authors claim to have seen marked improvement with the use of fibrolysin injections. Cardiolysis, the operative measure designed by Brauer, may prove useful if the pericardium is adherent to the sternum and may be considered if the general condition of the patient is not unfavorable. In the child, however, the hope of success is slight. Accordingly it is better, as a rule, to confine oneself to the usual symptomatic treatment of existing cardiac insufficiency (see page 416). Digitalis often entirely fails of results because the myocardium is seriously affected. Large pleural or peritoneal effusions must be tapped. Rest and the best of care and of food are, of course, essential. In mild cases carbon dioxide baths may be tried.

MYOCARDITIS AND CARDIAC INSUFFICIENCY

Acute myocarditis often accompanies various infectious diseases, especially diphtheria, scarlet fever and sepsis, and occasionally pertussis, typhoid fever, etc. In rheumatism, the disease often develops coincidently with endocarditis, so that the old term carditis or pancarditis is quite appropriate. But rarely is myocarditis primary, and even when it is apparently so it will often be traced to some general infection which has failed of earlier recognition.

Chronic myocarditis is much less frequent in the child than in the adult; since arteriosclerosis, so commonly associated with it, does not occur in the young. It is occasionally found in relation to the infectious diseases; most frequently, of course, accompanying valvular lesions and less often with obliteration of the pericardial sac.

Pathologically the heart muscle in acute myocarditis is found to be indurated and of a yellow color or streaked with yellow. The microscopic changes are often more pronounced than might be expected from the macroscopic findings. Degenerative changes of the muscle fibre, with round cell infiltration, occur. In the chronic form, placques similar to those occasionally seen in syphilis and in tuberculosis, may be found.

Symptoms.—The symptoms of acute myocarditis are often very indefinite and are apt to be hidden by, and quite indistinguishable from the symptoms of primary infectious disease with which it is associated. Its manifestations are definite only when they outlast the acute fever. Not infrequently the progress of the disease is wholly latent and sudden cardiac death may be the result.

The most important indications are the diminution of the heart's force and the small, rapid pulse. Bradycardia is rare. The weak pulse of acute

infectious disease is generally regarded as a sign of the weakness of the heart muscle. Very often, however, it is rather the result of a toxic vasomotor injury and the heart muscle itself may be entirely normal. Pallor, restlessness, dyspnœa, and even cyanosis may be caused by either form of disturbance. The apex beat and the heart sounds are usually weakened in myocarditis; the second sound may even disappear; and one or both ventricles may become dilated. Systolic murmurs are due to relative insufficiency. Enlargement of the liver is one of the earliest indications of resulting congestion. Peripheral edema, on the other hand, is rare. The blood-pressure falls; the pulse often becomes irregular, but is seldom slow. Protein is frequently found in the urine as a result of the primary disease. Sensations of constriction and pain in the chest, indicating heart disease, are much less common than in adults.

Myocarditis occurring in diphtheria presents a peculiar quality which will be discussed under that disease.

The serious cardiac disturbances sometimes developing in scarlet fever are probably traceable to myocarditis. The extremely rapid pulse frequently observed in ordinary attacks of this disease seems to indicate a peculiar affinity of the scarlatinal toxin for the heart muscle. This is further suggested by the bradycardia often found during the second week; by the cardiac dilatation and the transitory murmurs which, according to Stolte and Lederer, are of atonic origin. These cases in which a rapid pulse and cardiac insufficiency continue for many weeks after the subsidence of temperature are probably of myocardial character.

Typhoid fever causes myocardial degeneration much more frequently in children than in adults, in spite of its usually mild degree. It is rarely, however, a cause of death in this disease. The arhythmia which develops in convalescence from this and many other infectious disorders, even though the primary disease is not of severe grade, is probably due to the slight myocardial changes of a transitory type.

Chronic myocarditis occasionally develops after acute infectious diseases and most frequently after diphtheria. Tachycardia, arhythmia of the small pulse, very rarely bradycardia and, at times, dilatation are observed.

The most serious symptom is an increasing insufficiency of the cardiac muscle, seen in the later stages of valvular lesions, in which it is usually the ultimate cause of death. It occurs most commonly in acute and chronic myocarditis. Its well-known indications are the dilatation of the right heart, a decreased blood-pressure, dyspnœa, cyanosis, enlargement of the liver and ascites. Peripheral edema is very frequently absent or it appears late.

In rheumatic pancarditis, obliteration of the pericardial sac, with the obstacles which it places in the way of normal heart action, and the accompanying myocarditis are chief causes of insufficiency. Many conditions, on the contrary, which cause cardiac insufficiency in the adult, *e. g.*, pulmonary emphysema, arteriosclerosis, fatty heart and contracted kidney are wholly negligible in the child.

Diagnosis.—The diagnosis of myocarditis is often extremely difficult, especially during the fever period of acute infectious diseases when a feeble pulse is often due to vasomotor disturbances. Consequently it is often an obscure question whether the primary symptom-complex is to be attributed to an organic, a toxic, or a dynamic injury. Into the problem, the possible impairment of the peripheral or central nervous mechanism and of the intrinsic heart ganglia enter. In diphtheria it is especially difficult to determine to what extent a primary disturbance of the heart action depends upon disease of the myocardium or upon disease of the vagus.

The muscular tone response in the heart chamber, is shown by a retraction of the antrium, auricle or ventricle following a series of twenty strokes with the percussion hammer of from 1 to 2 centimeters inside of the previous outline. If the patient is horizontal and an attendant passively elevates the legs to an angle of 75-80°, and allows the child to lower them slowly, the auricles retract in a heart with normal muscle tone, while if there is myocardial weakness the auricles, or both auricles and ventricles dilate (Minerbi).

The symptoms of myocarditis are often similar to those of recent endocarditis. Of course the two diseases are frequently coexistent. In scarlet fever, acute dilatation due to nephritis is sometimes mistaken for myocarditis.

The prognosis of myocarditis is always doubtful. In diphtheria one must always be prepared for sudden death. The prognosis of cardiac insufficiency in the chronic heart diseases of childhood is even less favorable than in the adult. It must be said that compensated heart lesions in children have a more favorable course than they show in later life, but that when failure of compensation occurs it is less susceptible of permanent recovery and more rapidly fatal (Weill).

Treatment.—In the matter of treatment the uncertainty of diagnosis is not of so great importance, for up to the present time therapy is purely symptomatic and must be directed to the cardiac insufficiency and the consequent disturbances of the circulation.

The treatment of acute myocarditis and of cardiac weakness in acute infectious diseases must aim primarily to protect the heart and to avoid injuries from without. Absolute rest in bed and the avoidance of all excitement, such as may be attendant upon unnecessary painting of the throat in diphtheria, are demanded. Antipyretics, with the possible exception of quinine, and alcoholics are injurious and must be avoided entirely. Tepid baths act favorably upon the heart. In older children, with fever, ice-bags cold compresses, or ice-coils are often pleasant and quieting and will probably have a tonic influence.

The diet should be light, fluid or semifluid, preference being given to milk but it should be moderate in quantity and excess, particularly of fluids should be avoided.

Digitalis is of doubtful value in the cardiac weakness of acute myocarditis and should be employed only in cases in which other remedies have failed. Of the cardiac or vascular stimulants, camphor and caffein are most

satisfactory. Their exhibition should not be delayed too long if the pulse becomes markedly weak or if severe pallor, dyspnœa and cyanosis develop.

Caffein sodio-salicylate may be given in doses of 0.05–0.15 gm. (grs. i–iii), each day, to infants; 0.2–0.3 gm. (grs. iiiss–v), to children of three to five years; 0.4–0.8 gm. (grs. vii–x), to children of eight to ten years. These quantities should be divided into three to five doses a day. Sufficiently diluted with water, this remedy is taken without objection by the patient; it is well borne, and hardly ever causes excitement.

Camphor is preferably given subcutaneously. It may be administered from three to eight times daily in doses of seven to ten minims of the ten per cent. solution in oil. The official spirits of camphor may be employed in the same manner, but it causes necroses if not carefully injected intracutaneously.

If the circulation is very bad, strychnia may be tried, giving, once a day and subcutaneously, 0.0005 gm. (gr. $\frac{1}{120}$), in the first year; 0.001 gm. (gr. $\frac{1}{60}$), to children of three to six years; and 0.002 gm. (gr. $\frac{1}{30}$), to older children. With the strychnia, an epinephrin solution, (1:1000), may be given, using 0.5–1.0 c.c. (minims viiss–xv), in 10. c.c. (℥iiss), of physiologic salt solution.

Often the best results are obtained with the combined treatment, so that there is no objection to using caffein and camphor together, each in smaller doses and, if necessary, adding strychnia or epinephrin also.

If no results are obtained from this therapy, the liquor digitoxini solubilis (digalen), may be tried intramuscularly, using 0.5 gm. (minims viiss), for very young children; and 1.0 gm. (minims xv), for older ones.

As in adults, digitalis is the sovereign remedy in cases of cardiac insufficiency in chronic heart disease and in the event of the failure of compensation in valvular lesions. The usual caution, however, must be observed and even more religiously in children.

Children under three years of age may be given 6 c.c. (℥i), of the infusion, once a day for two days; while children of five to seven years may be given 12 to 25 c.c. (℥iii–v), in the same way. The action of the fresh digitalis leaves is even more certain, 0.02 gm. (gr. $\frac{1}{2}$), in the powdered form, being given three times a day to children under three years; 0.04–0.07gm. (gr. $\frac{2}{3}$–i), to older children. Its use may be continued for four or five days. As soon as the dyspnœa decreases, and a free diuresis occurs and the pulse is distinctly slowed, the treatment must be stopped. The newer preparations give very good results and they are borne better by the stomach than the original drugs. Liquor digitoxini solubilis, or digalen, may be given in doses of 0.1 c.c. (minims ii), three times a day, to children in the first two years; and, as often, in doses of 0.2–0.5 c.c. (minims iii–viii), to older children. This remedy may also be given intramuscularly or per rectum. Digipuratum is given in doses of 0.02 gm. (gr. $\frac{1}{3}$), to 0.04–0.06 gm. (gr. $\frac{2}{3}$-i), according to age, three times a day. It is a very reliable drug and as certain in action as the leaves. It does not have any bad effect upon the stomach.

It is best to give the digitalis preparations for three to five successive days only. If necessary caffein, in the prescribed doses or, preferably if

there be marked dropsy, a theobromin preparation, such as diuretin [0.1–0.5 gm. (grs. iss-viiss), three times a day], may be substituted in the intervals. If the stomach will not tolerate this medication it may be given by rectum. In cases of dangerous cardiac insufficiency, in which the usual digitalis therapy would act too slowly, strophanthin, 0.00025–0.0005 gm. (gr. $\frac{1}{250}$ to $\frac{1}{125}$), in a single dose only, may be used to advantage in older children.

In congenital lesions the use of digitalis should be delayed so long as possible, employing it only when increasing dyspnœa and congestion indicate the beginning of cardiac insufficiency. In cases of open septum, digitalis may be injurious on account of the increased pressure in the pulmonary circulation, when the stronger left ventricle forces more blood into the already overworked right ventricle.

In cases of cardiac dropsy, in which digitalis and diuretin have failed, a combination of digitalis with calomel [0.03–0.06 gm. (gr. ss–i.), three times a day] will often prove efficient and cause a free flow of urine after three or four days of treatment.

In obliteration of the pericardial sac the myocardium is often so completely exhausted that digitalis proves useless.

When severe cardiac symptoms, as restlessness, dyspnœa, orthopnœa, a sense of constriction, etc., occur, the little patient should not be denied the beneficial effect of morphin which, under such conditions, is peculiarly restful to the heart. It is best given subcutaneously in doses of 0.001–0.002 gm. (gr. $\frac{1}{67}$–$\frac{2}{67}$), for children of two to four years; and of 0.003–0.004 gm. (gr. $\frac{1}{20}$–$\frac{2}{33}$), for children of six to ten years, increasing the dose until the desired results are secured.

APPENDIX

BLOOD-VESSELS AND JUVENILE HEART

Diseases of the blood-vessels are not of great moment in children, since arteriosclerosis occurs only exceptionally and in the later years of childhood. It is true that a mild degree occurs occasionally in young children and even in infancy (Saltykow), but it is without clinical significance.

Aortitis and aneurism of the aorta, due to hereditary lues, have been seen in children of eight to twelve years.

Only about 20 cases of aneurisms of the thoracic aorta in children are on record. Rupture of the aneurism has been the cause of death in 8 cases (Bronson and Sutherland).

Arterial emboli, on the contrary, are not uncommon in acute and chronic affections of the heart, and particularly following diphtheria.

During puberty one frequently sees disturbances which are regarded as due in part to peculiarities constituting the so-called "juvenile heart." Children, so conditioned, complain of palpitation, a sense of pressure in the thorax, and of shortness of breath. Combined with these symptoms there may be a heaving apex beat and an accentuated pulmonic second sound and occasionally even a systolic murmur. An enlargement of the

area of cardiac dulness, together with firm, tortuous arteries may be found; the latter leading one, however, to think rather of a functional stiffening than of a juvenile arteriosclerosis. The blood-pressure is not increased. The Roentgen ray reveals no actual enlargement of the heart, as one may have been led to suspect. In fact the heart is more likely to be smaller than normal, and it may occasionally resemble the "drop heart," with aorta of small calibre, seen in narrow-chested individuals, presenting the signs of general infantilism. A true narrowing of the aorta is, however, extremely rare and its significance in relation to these accompanying conditions has not been explained. In these disturbances of the juvenile heart it is usually only a question of reduced functional capacity, relationally to the age of the child, or, in other words, a disproportion in growth. Such anomalies do not always disappear.

It must be remembered that masturbation may cause exaggerated and rapid heart action.

VI.

DISEASES OF THE GENITO-URINARY SYSTEM

C. NOEGGERATH

Revising the section by L. Tobler in the first four editions.

INTRODUCTION

The study of the diseases of the urinary apparatus is not completed by the mere demonstration of the presence, nature and amount of protein and formed elements in a given urine specimen. It is necessary to determine, further the location of the disturbance, whether it is renal or extra renal; glomerular, tubular or interstitial. This is shown by the clinical course. It is still more important, however, to determine the degree of secretory disturbance—just as it is essential to determine the compensation in heart lesion—and the careful study of other organs such as the skin (pallor, edema perspiration); the body cavities (transudates); the eye grounds, the nervous system; and the gastro-intestinal tract; is of utmost importance.

As a part of the latter, the determination of the blood-pressure is one of the more necessary steps. The usual methods, with cuffs of varying size to suit the age of the patient, are employed. The following, which may be considered averages for different ages, are given in millimeter mercury: nurslings: 108/80; three years: 110(118)/78; six years: 118 (125)/83; eight years: 124 (129)/85; after the ninth to eleventh years the values are the same as in the adult, 134 (136)/95. Here the first figure is the maximum pressure as determined by auscultation or oscillation, the second, in parenthesis, the palpation determination, and third the minimum. The pressure of the bed-ridden is lower and that of neurotic individuals higher.

Even in childhood the increased blood-pressure differentiates acute and chronic diffuse glomerulitis from the focal forms and from interstitial nephropathy and especially from tuberculous nephritis. In consideration of the rather wide range of the normal pressure and the great frequency with which slight variations are encountered, the single record is not nearly as valuable as a rise or fall in a series of measurements or a sudden change from a formerly recorded measuring of the pressure. A sudden rise may be a valuable early symptom of uremia.

In infancy and childhood, the various changes of the blood itself, such as concentration, reduction of the protein, etc., are similar to those of the kidney diseases in the adult. The more or less fixed sodium chloride content is 560 (540) mg. per 100 c.c. serum. The combined protein equals 28–40 mg. per 100 c.c. protein free serum—both values the same as in the adult.

The functional tests are to be obtained by determining the extra and intrarenal metabolism of water as shown by daily record of the body-weight. This is the only method by which it is possible to show the retention of water in the internal organs, the so-called pre-edema of Widal. To this must be added the observation of the specific gravity of the urine and the total daily quantity as compared with the amount of water ingested.

After the third or fourth day after birth, the total daily quantity of urine represents a definite proportion of the fluid ingested. While this varies in individuals and at different periods, the averages may be given as follows: For every 100 c.c. food injected the new-born secretes 60 c.c. urine; the nursing infant 68 c.c., and the child on partial solid diet and throughout childhood, 70 c.c. (Camerer). The total daily quantity of urine and the specific gravity are subject to great variations. During the first year 100 to 500 c.c. of a specific gravity 1.004 to 1.010 are secreted. During the second year 600 c.c. of 1.006-1.012 specific gravity. During the third to fifth years, 500-800; from the fifth to eighth, 600-1200 of 1.004-1.012 specific gravity; and from the eighth to fourteenth years, 800 to 1500 c.c. of a specific gravity of 1.002 to 1.024 (Holt).

Pollakiuria is physiologic in the nursling. The power of concentration and dilution of the urine is present at birth, but the concentration found in the adult does not occur before the end of the first or beginning of the second year. After this the interpretation of variations, except as shown by the normals above, is as in the adult. This is also true of polyuria, oliguria and anuria alone or combined with disturbances of dilution and concentration.

The tolerance tests differ but slightly from those used in adults. For several days a test diet of sufficient caloric value, with reduced protein, poor in sodium chloride and containing a small amount of water adapted to the age of the patient, is given. This means a milk and vegetable diet or a pure vegetable diet, or in very young infants, breast-milk. The substance for which the kidney tolerance is to be tested, water, sodium chloride, protein, is then added to this diet in quantities sufficiently great to more than supply the normal retention of growth but not so great as to cause injury to the patient. Thus 200 to 1000 c.c. water may be added, or 60-100 grams protein in the form of calcium caseinate, or 1 to 5 grams sodium chloride. These are given with the first morning feeding after emptying the bladder. The secretion of these substances is observed as in the adult and by means of it the specific renal efficiency may be determined. The retention of sodium chloride and water is indicative of tubular nephritis and the retention of the end products of protein metabolism indicates glomerular nephropathy.

UREMIA

The toxemia caused by substances of unknown origin, that may be formed in the urine, occurs in the two forms, as follows:

First: Eclamptic uremia, more common in childhood, and not accompanied by nitrogen retention, is probably due solely to the retention of sodium chloride and water in the tissues. The diarrhœa and vomiting is the natural attempt of the organism to free itself of the overload. If, however, the oliguria causes pressure and retention of sodium chloride in the cerebrospinal fluid and resulting brain edema, the symptoms of headache, slowing of the pulse, increased reflexes, Babinski's sign, and even unconsciousness to coma supervene. If the brain pressure is further increased, the blood-pressure rises suddenly and life ends in an eclamptic seizure.

The general tonic—clonic twitchings, wide fixed pupils and rapid pulse resemble the symptoms of true epilepsy or being monoplegic may be mistaken for Jacksonian epilepsy. Forms with chorea, ataxia, or disturbances of speech are seen.

Very frequently the condition does not advance beyond the early symptoms of disturbed reflexes. These often indicate the beginning of polyuria and recovery. Contrary to expectation, extreme uremic eclampsia is less often met with in tubular nephritis than in the glomerular form, from which it would seen that the central nervous system in the latter cases is more liable to edema.

Second: The form of uremia, characterized by the retention of nitrogen in the tissues and increased blood nitrogen (azotemia) dependent upon unknown urinary toxins is seen in cases of total anuria such as caused by double obstruction of the ureters, hydronephrosis, tumors, and in extensive destruction of the glomeruli in glomerular nephritis. The most important initiating symptoms, severe headache, sleeplessness, anorexia and vomiting, should serve to call the physician's attention to the steadily decreasing amount of urine and this demands prompt interference. Later we find the heavily coated tongue, an odor of urine on the breath and even, occasionally ulcerative stomatitis. The patient is tired and somnolent and, as a result of diarrhœa and nausea, especially when offered meat, becomes weak. Gradually a general anesthetic somnolence (hence "sleeping uremia") with reactionless narrowed pupils appears. This may be accompanied by restlessness, panting respiration, fear and cardiac distress. The reflexes are increased and Babinski's sign is present. Blindness of one or both eyes due to central lesions may be added and, finally, general uremic convulsions form the climax of the condition. Recovery may occur after the entire syndrome or during any part of the course. Or the outcome may continue in a more chronic form to a fatal end. It is not uncommon to find a mixed form of both the above conditions in young children.

As to the treatment of uremic poisoning: With severe headache, vomiting and marked oliguria, either hypo- or isotenuria, liberal phlebotomy (120-150-200 c.c.) is indicated. In chronic cases decapsulization of one or both kidneys is advised. Lumbar puncture promptly relieves the brain edema. A purely carbohydrate diet aids the removal of the poisoning. By the combination of these energetic measures the seriousness of acute uremia in childhood has lost some of its terrors.

Orthotic Albuminuria

Very small amounts of protein may be demonstrated in the urine of healthy individuals if the urine be evaporated to greater concentration and the more delicate tests be applied. The demonstration of protein by the ordinary clinical tests does not always justify the supposition that the kidney has suffered structural changes, even though the proteinuria be of true renal origin and is not caused by the mixture with the urine of such protein-containing fluids as blood or pus. Von Leube has found noticeable quantities of protein in the urine of one-third of the healthy soldiers

examined, the proportion being increased if the examination was preceded by physical exertion. Other authors claim to have found proteinuria after heavy meals or cold baths and even after excessive mental activity or emotional excitement. Attempts at palpation of the normal kidney often result in transitory proteinuria. If children of a certain age are made to kneel in an upright position (see Fig. 109) for a short time, one-third to one-half of their number will show protein in the urine, often in considerable amounts. Indeed a benign form of proteinuria is very frequent at this age. The phenomenon appears and disappears with a degree of regularity and

Fig. 109.—Relation of the amounts of protein (black) in the urine as affected by posture. Eleven-year-old girl with orthotic albuminuria; (1) Kneeling with marked kyphosis; (2) Natural standing position, with lordosis; (3) Increase of lordosis on kneeling; (4) Forced lordosis, standing.

without reference to any particular preceding injury. It may develop as large quantities of protein as a true nephritic proteinuria.

From these facts it will appear that a gradual transition may occur from the realm of health to conditions that must be considered abnormal and it is impossible and impracticable in this particular to draw a definite line between health and disease. Certainly it cannot be done by using the amount of protein in one or more specimens of the urine as a criterion. Comparison of the conditions under which a proteinuria appears and the general state of the individual's health will probably establish a more satisfactory basis.

The functional proteinuria of childhood has been distinguished from the nephritic form by several names. Pavy who first described the condition applied to it the term "cyclic," on account of the remarkable regularity with which the condition appears and disappears at definite hours of the

day. Stirling proposed the designation postural in order to emphasize its most essential characteristic, a dependence upon the erect position of the body. Orthostatic albuminuria, the name of more general usage, has practically the same meaning, while the older designation of Heubner orthotic albuminuria suggests more nearly the nature of the condition which is correctly described as a proteinuria caused by "raising oneself to an upright position," or rather as a result of changing from a horizontal to a vertical posture, in spite of the maintenance of which the proteinuria gradually disappears.

The functional proteinurias are common during the same period in which the orthotic type occurs. These functional forms have been described under various names, especially by the French authors. The latter not only distinguish between the cyclic and orthostatic types, but also claim to be able to differentiate hepatogenic, alimentary and pretuberculous forms.

All these proteinurias disappear with absolute rest in bed. Indeed they are all influenced by posture and it hardly seems possible or justifiable to group them upon solely theoretical grounds.

Occurrence.—Orthotic proteinuria is most common between the ages of seven and fourteen years. It is very rare in early childhood and its likelihood of occurrence decreases toward puberty. Its greatest frequency is coincident with the period of greatest bodily growth, from the eleventh to the fourteenth year. The affection is somewhat more common among girls than in boys. The figures of absolute frequency vary greatly. Reports of different observers record the condition in from 5 to 30 per cent. of all children of school age examined. It may appear in several members of a family and not uncommonly in succeeding generations.

It is more frequently seen in the large families of the needy, living in unhygienic surroundings, than among the well-to-do. Tuberculosis and neuropathy are among its hereditary antecedents, but no definite rules of such relationship can be laid down. Scrofulous and tuberculous children are found among those so affected, but they do not constitute a majority of its subjects.

Symptoms.—Orthotic proteinuria is often discovered in the course of routine examinations. The patient comes to the physician complaining of indefinite symptoms such as headache, lassitude, loss of interest in work or play, sleepiness, attacks of dizziness tending even to fainting spells, anorexia, nausea, and occasional vomiting. Palpitation of the heart, side-ache, and indefinite pains in the back and limbs, the so-called "growing pains," are frequent accompaniments. A history of epistaxis is common.

Orthotic proteinuria is often encountered in strong, healthy looking boys and girls who have grown rapidly and developed early. An individual predisposition, which we may anticipate, does not necessarily suggest itself in the general conditions of the child. The majority of these patients are thin, rather weak individuals with a poorly developed panniculus. Objective symptoms of anemia or chlorosis may be lacking in spite of their delicate appearance and usual pallor. It may be assumed that the distribution of the blood is inadequate. Other signs, in fact, indicate anomalies

of the circulation. This view of a functional vascular disturbance is best pictured by the term "irritable weakness." The peripheral circulation is feeble, the extremities are cold and moist and there is marked dermatographia and recurrent congestion. Erythema and urticaria are common. The pulse is labile both in volume and in frequency and is at times dicrotic. The heart is often slightly dilated, but this may be simulated by such a change of posture as a dependence of the head, or by a narrow thorax. Radiographically Reyher has demonstrated the small "drop heart" more frequently than the large heart. Impure heart sounds are heard at the apex and slightly above that point and soft systolic murmurs are common. The blood-pressure is generally normal, but arterial tension is occasionally reduced.

The total daily quantity and the general appearance of the urine do not differ materially from the normal. That a sediment of phosphates and urates may appear in the several specimens collected for examination is in part due to alimentary causes and is in part dependent upon increased concentration resulting from the orthosis. The marked cloudiness, especially in the morning specimen of urine in girls, at the developmental period, is a result of the desquamative catarrh of the external genitalia which is notably common at this age. A sediment consisting of numerous flat epithelial cells and leucocytes comes from the same source. Oxalate crystals are not uncommon. In a twenty-four hour specimen of urine the protein content is often minimal. A clear understanding of the condition may be obtained only from the examination of a succession of specimens. Urine excreted during the time when the patient is lying down is usually free from protein which reappears when the patient gets up. Since the proteinuria which develops while the patient is erect may at times continue for several hours after lying down, the morning specimen is found free of protein only when that excreted during the early hours of the night is separated from it and when the patient has not risen during the remainder of the night. The urine excreted during the first few hours after rising contains the largest amount of protein, which increases with the quantity of urine voided during this period. Under ordinary circumstances the curve representing the protein content sinks gradually from a rapidly attained maximum to normal. Even in children who have spent the greater part of the day on their feet, the urine is comparatively free from protein toward evening. If the child lies down during the day the protein content of the urine will again increase after the patient gets up. Thus the regularity of a cyclic albuminuria is dependent upon the daily routine and its apparent periodicity is governed by external influences. Upon continued rest in bed the urine of all these patients is free from protein and the morning maximum can easily be made to appear at night. The longer the horizontal position is continued, the greater is the influence of the change of posture. The amount of protein in the urine of the orthotic patient varies within wide limits in the given individual as well as in different cases. Generally speaking, the amount of protein remains within moderate bounds, but may run as high as 2-5 parts per thousand and, in exceptional cases, even more

The percentage of protein in the given specimen depends in great measure upon the dilution of the urine, excreted during the first fifteen minutes in the erect posture, by that collected in the bladder during the prone position.

In a degree the excretion of a protein body which can be demonstrated only by coagulation with dilute acetic acid in the cold specimen is quite characteristic of functional albuminuria. While this protein is hardly ever absent, its proportion to the total protein excretion is variable. Occasionally it exceeds other proteins.

In order that this significant finding be not overlooked the test should be carried out as follows: The clear filtered urine is divided in three test-tubes and is diluted with two or three parts of water. The first tube is used as a control. To the other two are added a few drops of dilute acetic acid and to one of them a few drops, also, of potassium ferrocyanide solution. The reaction often requires some time for its completion.

The quantity of urine excreted during the periods of proteinuria varies directly with the quantity of protein present and is governed by the same external influences, so that the output of protein always coincides with a decrease in the quantity of urine (Fig. 109). With the patient in the erect position, the urine is dark, contains much urobilin and bile salts but, with exception of an occasional hyalin cast, no formed element. The sodium chloride content is low. While in the prone position the quantity of the urine is increased with resulting pollakiuria and occasional enuresis. It is lighter color, less acid or even alkalin. Zondeck maintains that the otherwise normally functioning kidney is unable to handle heavy loads of protein and to be slow in the excretion of sodium chloride.

Nature and Pathogenesis.—In spite of the fact that for a long time the interest of the physician has been especially directed to the study of the nature and pathogenesis of intermittent proteinuria, many important points are still unexplained. The view that cyclic albuminuria represents an extremely insidious chronic nephritis, probably developing from the first in very small foci, has lost much of its support. The most damaging blow to this theory was recently given by the actual findings of Heubner and Langstein in a case at autopsy. In a ten-year-old girl subject to orthotic albuminuria, and dying of an intercurrent disease, no nephritic changes were found. Why the kidney functionates abnormally and why this abnormal function should be induced by a posture, are questions which still remain unanswered. The second question seems nearer a solution than the first. It has been determined experimentally that the change from the horizontal to the erect position is the determinating factor. But a further qualification becomes necessary. If the body-weight is eliminated by immersion in water, or if the body is stretched by supporting its weight from the head, the proteinuria does not appear. So that apart from posture, the influence of the body-weight upon the position is evidently a factor. In the normally erect position there is a slight lordosis of the vertebral column. Jehle has found that all his patients showed a distinct and typical lordosis of the lumbar spine and he considers this of etiologic importance. In order to affect the urinary excretion, the lordosis must be sharply curved with its extreme

point at the level of the first or second lumbar vertebra.[1] Jehle believes that such a lordosis distorts the renal vessels passing over its ventral convexity and thus hinders the circulation in the kidneys.

There can be no doubt that lordosis is an important factor in the etiology of orthotic proteinuria; but many more careful observations will be required to determine the actual measure of the part it plays. It has been definitely shown that the development of a typical lordosis may provoke proteinuria in convalescents, but a greater degree of deformity is required to produce this effect in such persons than is seen in the spontaneous lordosis of many patients with orthotic proteinuria. Then, too, it is to be remembered that typical lordoses without proteinuria occur. Further it is to be noted that in the proteinuria caused by lordosis in healthy children the proportion of acetic acid test bodies in the total protein is distinctly less than in true orthotic cases. From this it would not seem that an orthotic proteinuria could be hidden by a lordotic proteinuria. It is rather a question how the severe reaction of the orthotic patient can be explained by the slight lordosis.

The hypothesis of von Noorden, Weinstraud and others deserves some attention. They suggest that the tonicity of the trunk muscles in the erect position furnish a nervous impulse which in turn results in a spasm of the kidney vascularity with nephritic ischæmia and definite changes in the excreted urine. This is supported by the fact that the urine secreted while the patient is in an upright position resembles that obtained by temporary clamping of the kidney vessels. The author is inclined to suggest the term "angiospastic dysuria."

The mechanical factor of the lordosis apparently becomes operative when it is present in constitutionally weak individuals. Of the constitutional anomaly which this theory presupposes, it may be said that it appears within a definite period during the years of development and that it affects either the secretory or the circulatory apparatus of the kidney. It is a well-known fact that the slightest disturbance of the blood supply changes the kidney into a protein secreting organ. Vasomotor variations of the renal circulation, dependent upon a constitutional predisposition and induced by a lordotic posture, must be regarded from the present viewpoint as the etiologic basis of orthotic proteinuria.

Diagnosis.—The diagnosis of an intermittent proteinuria determined by posture is readily made. The careful examination of specimens from each urination for protein, and the observation of the cardiovascular and nervous symptoms, is all that is necessary. With this initial determination, however, the clinical interest in a case has only begun and diagnostic difficulties appear which cannot be met by the most careful analyses of single urinary specimens. For the orthotic type of proteinuria is not specific since nephritic proteinuria may, and not infrequently does, take on the similar form. A supposed orthotic proteinuria, giving small amounts of protein in the morning specimens, after all precautions have been taken,

[1] Pseudo-lordosis, in which the spinal column is sharply bent backward directly over the sacrum, is a very common condition in childhood.

and in which, moreover, the cycle cannot be interrupted by periods of rest in bed must always excite a suspicion of nephritis. This suspicion increases if the effects of a nephritic increase of blood-pressure are discoverable in the pulse and heart-beat. The diagnosis of nephritis is, of course, fully established when albuminuric retinitis, usually sought in vain, is discovered. Slight uremic symptoms make the differential diagnosis no easier, since they are not unlike those which patients with orthotic proteinuria exhibit. The slightest urinary sediment should be carefully examined. It is true that one or two hyaline casts in the sediment obtained by the use of the modern centrifuge are not very significant. If, however, well and regularly formed hyaline casts are found in large numbers, with other varieties of casts and red blood-cells, we must always consider them, according to Heubner, as manifestations, not of orthotic proteinuria, but of nephritis. If these distinctive signs are not found at once it may be necessary to keep an obscure case under observation for months in order to clear it up, for even in true nephritis long periods may elapse in which there is a minimal amount of sediment with only an occasional renal element.

The differentiation of proteinuria from the normal status is more simple; but since its therapy is of the passive order the effort loses interest. For traces of protein in the diurnal urine do not justify us in pronouncing a child ill who does not complain nor does it demand the interference of the physician. It can hardly be considered rational to place a child in a position of forced lordosis merely for purposes of diagnosis, even if it did not lead at times to harmful and mistaken conclusions. Especially is this true since so imminently a sensitive organ as the kidney may respond even in the healthy individual to such a trauma not only by way of proteinuria, but also by the appearance in the urine of large numbers of red blood-corpuscles and of hyaline casts. From a medical viewpoint interest attaches only to the question whether the demands of the child's daily routine cause proteinuria.

Prognosis.—The prognosis of pure orthotic proteinuria is always favorable and this marks the practical distinction between the orthotic type and all other forms of proteinuria which are affected by posture. If a case of orthotic proteinuria terminates in chronic nephritis, either an error in diagnosis or a combination of disease must be suspected. The latter is, of course, possible, but there is nothing to show that the kidney of the orthotic patient is especially predisposed to inflammatory changes. Uncomplicated orthotic proteinuria usually runs a very chronic course. Recovery even in the course of one or two years is not common. Usually the earlier it appears the longer it lasts, for spontaneous recovery, and there is no other, is commonly postponed until after puberty. The condition hardly ever persists later than the twentieth year. Intermissions, lasting for months, may occur.

Treatment.—The subjective disorders accompanying the symptoms of proteinuria are more amenable to treatment than the major symptom itself. Quinine or, when anemia or chlorosis is present, iron is often of great service. Such procedures as prolonged and absolute rest in bed, in the effort to suppress the excretion of protein at any price, are contraindicated.

The loss of protein is never great enough to endanger the metabolism even though the amounts are seemingly large. The rest-cure only delays the achievement of the purpose of saner therapeutic measures, the general upbuilding of the body. For this purpose, exercise is more important than rest; and fresh air with suitable games should be encouraged rather than denied. Dietetic treatment must be considered from the same viewpoint. The so-called nephritic diet is to be avoided and the appetite should be stimulated by a varied diet suited to the age of the patient. It is not necessary even to prohibit meat and eggs. Gymnastic exercises for the correction of the lordosis and the development of the muscles, especially of the trunk and abdomen, are to be recommended. Standing or kneeling for any length of time, tending to increase the lordosis, must be avoided. Orthopedic correction of the lordosis does not seem justified. Low-heeled shoes serving to counteract the lordosis may be worn.

TUBULAR NEPHROPATHY OR NEPHROSIS

Pathology.—A chronically progressive cloudy swelling with hyaline deposits, fatty deposits and with lipoid degeneration or necrosis of the tubular epithelium. Connective tissue and glomeruli may be affected in the early stages and always are in the late chronic cases.

Etiology.—The etiologic factor is frequently indeterminable. Diphtheria, lues, tuberculosis, or chronic purulent processes may be factors. Occasionally the condition may be brought on by acute poisoning with tar in ointments or salvarsan—in rare cases the colon bacillus or pneumococcus may be the cause.

Pathogenesis.—The knowledge of the pathogenesis rests upon various hypotheses, one of which lays the causation to the toxemia of the body cells by the products of decomposition of the diseased tubular epithelium. It is certain that this disturbance of the secretion of the urine is as definitely a constitutional disease as it is a true kidney disease.

The functional disturbance is characterized by the reduced excretion of sodium chloride and water, during the stage of edema, while there is no change in concentration power. With the improvement of the condition the excretion of these two substances increases and may be excessive. There is a distinct tendency to eclamptic uremia. The excretion of nitrogen remains normal or may be increased to as high as 2 to 3 per cent. in the twenty-four hour specimen and, for this reason, there is no nitrogen retention (80-100 mg. per 100 c.c. in protein free blood) and as a result no azotemia. During the formation of the edema, the blood is thickened and as the water is increased becomes thin again which can be recognized by the variation in the red cell count.

Symptomatology.—The outstanding feature of the clinical picture is the pallor and edema. As a result of the edema, we have the transudates into the body cavities, gastro-intestinal disturbances, eclamptic uremia and generally lowered resistance. The urine, in which there is a great deal of protein, at first contains large amounts of formed elements, chiefly lipoid and fatty casts. The absence of blood and the normal blood-pressure are of

diagnostic importance. The slight variations of temperature are of no significance. Frequently the two important symptoms of pallor and edema brought out in the rather indefinite history of the case suggest essential nephrosis. Slight swelling of the face points to the general anasarca, while sudden nausea, weakness and dizziness indicate brain edema. Diarrhœa vomiting or bronchitis may occur. After a week or so the urine shows a large amount of protein. At this stage the observer is impressed with the waxy pallor and examination reveals the edema. The heart rate and blood-pressure are unchanged or but slightly increased. The reflexes may be increased. The patient feels bad and has no appetite. Complains of pain on pressure especially over the tibia. The urine, dark brown, cloudy and acid, is highly concentrated (1039-1050 specific gravity). The twenty-four hour quantity is reduced to as low as 150 c.c. Large amounts of urates are deposited on standing.

The protein content is very great, amounts of 10-20-30 per cent. or even higher may be demonstrated. In the centrifugate are found epithelium, leucocytes, large amounts of various kinds of casts at first, and later epithelium and leucocytes showing fatty degeneration and lipoid bodies. In the later stages the number of casts is greatly reduced. The absence of red blood-corpuscles, which may be present in an occasional specimen, is diagnostic.

Course.—While an occasional mild, post infectious case may be encountered in infants, the majority of the cases have persisted for months or even years. With proper treatment the edema usually soon subsides but following it we have the second or chronic stage. In this the little patients, with dark circles under their sunken eyes and often greatly emaciated, look sick. Even though they begin to feel better as their appetites return and weights increase, the tendency to edema still persists. It may reappear suddenly at the slightest exceeding of the tolerance for sodium chloride or at times even without demonstrable cause. Such a recrudescence may even change the rather monotonous chronic condition to the eclamptic uremic form with gastro-intestinal disturbance. The proteinuria persists for a long time after the formed elements have disappeared. As in the edema of "flour-feeding injury," the greatest danger lies in the lowered resistance especially against pneumococci (peritonitis, empyema). Death may result from angina or bronchitis or erysipelas from an infected wound.

Prognosis.—The prognosis must be guarded and must take into consideration the chronicity and danger of infection. Either transition to a contracted kidney or eventual recovery are possible. Recovery is complete only, when, without proteinuria, the excretion of 10 grams of sodium chloride in twenty-four hours can be demonstrated.

Treatment.—The treatment must be directed first against the edema. During the period of formation, however, not much can be accomplished. Even when the tissues are filled such medicinal aids as heart stimulants and diuretics do not drive the fluid into the blood and to the kidney. At times five to ten grams of urea in water may help or a liberal venesection (100-200 c.c.) may be useful. The author has seen the anuria overcome in several cases by the use of large amounts of water (1 litre) given as Vol-

hard recommends. The hunger and thirst cure of von Noorden and Volhard has much to recommend it. This consists of a diet of sufficient fat in form of unsalted butter or cream, sugar in form of a fruit syrup, and a little salt-free bread—together with as much fluid as there was urine excreted on the previous day. Breast-milk may be used even in older children. Transudates and ascites may be relieved by tapping. The fluid obtained is milky owing to the lipoids. The relief of the anasarca by scarification or by drainage with hypodermic canula cannot be used in infants because of the danger of infection. Sweats produced by moist or dry heat are useful. The treatment of uremia has been discussed.

Fig. 110—General nephritic dropsy, ascites (University Children's Hospital, Munich, Prof. Pfaundler).

After the removal of the fluid the patient is usually hungry and the diet should be salt-free and not contain too much liquid, but must supply sufficient calories. It must be adjusted to the tolerance for protein, replacing that lost by proteinuria. Furthermore in the feeding the tendency to edema must be considered and changes made to keep pace with the gradually increasing sodium chloride tolerance and water excretion. The latter can be determined by the relation of body-weight and fluid intake and urine excreted. The determination of the excretion of sodium chloride is readily accomplished with the Strauss chloridometer, the use of which is as simple as that of the Esbach albuminometer. To recapitulate: the diet should consist of fat in the form of unsalted butter and cream, sufficient protein (cheese and meat), some carbohydrate (bread, gruels, etc.) and some fresh vegetables for the vitamine requirement.

Diphtheritic nephrosis presents all the symptoms of very mild tubular disease. It is rare in mild infections and common in the severe forms. It begins early, usually between the fourth and tenth days. The tendency to edema is an early symptom but the patients rarely present severe anasarca or ascites. Usually the retention of fluid is evidenced only by the rapid increase in weight which should lead to examination of the urine. The total daily quantity is decreased but rarely below 200 to 500 c.c. The color is dark yellow or dirty brown. Protein and formed elements may be present in small amounts at the beginning or entirely absent in the milder forms. The protein rarely exceeds 1 or 2 per cent. with the Esbach method. The

formed elements soon disappear. Red cells in the urine, and increased blood-pressure do not occur in the pure forms.

Usually this mild complication disappears in ten days to two weeks without symptoms of uremia although it may persist in varying degree of severity to the cardiac death caused by the diphtheria itself (low blood-pressure). Transitions to the chronic forms occur, and the addition of glomerular symptoms is not uncommon.

In ordinary cases, treatment is not indicated for the diet will usually be such as not to exceed the sodium chloride tolerance of the diphtheritic kidney. In the severe forms the treatment is that recommended for true nephrosis.

ACUTE DIFFUSE GLOMERULAR NEPHROPATHY, ACUTE GLOMERULAR NEPHRITIS

Pathology.—At first, congestion rapidly followed by distention; that is, elongation and widening of the glomerular loops with hyperplasia of the endothelium and swollen capsular epithelium. Later, obliteration of the capsule and scarification of the glomerulus may occur. The tubules and parenchyma may remain free or become involved to a varying degree.

Etiology.—The most important etiologic factor is scarlet fever. Angina, bronchial infection, pulmonary disease (pneumonia), skin infection (erysipelas), pneumococcus or staphlococcus infection are frequent causative factors. Purpura, lead poisoning and occasionally the factors listed under nephrosis must be kept in mind.

Functional Disturbances.—The marked reduction of the excretion of the nitrogenous waste products in the urine and their simultaneous increase in the blood and tissues is diagnostic of glomerular nephritis. The index of this condition is the increase of the non-protein nitrogen above normal limits. While the kidney still retains its concentration power and excretes urine containing 1.5-2.5 per cent. nitrogen the blood is sufficiently relieved. Large amounts of the urinary poisons are neutralized and made harmless in the retained fluid of the edema if this does not affect the central nervous system. When there is no edema, in the so-called "dry cases" or in cases with hypo- or even isotonuria accompanied by oliguria or anuria, uremia obtains. In the uncomplicated cases the sodium chloride and water are excreted, but in edematous cases the action is the same as in the nephrosis. Increase in blood volume without edema is a serious symptom of kidney insufficiency.

Symptoms.—The most characteristic symptoms are the urinary findings: blood, moderate amounts of protein, with oliguria and possibly increased specific gravity or anuria with uremia. There is an increase of the blood-pressure and not uncommonly a tendency to edema.

Course.—The course of the disease is protean. The glomerular nephritis may begin acutely at the very onset of the causative disease or may appear insidiously after two to four weeks. Any one of the characteristics, either alone or in combination with others, may usher in the onset. In the hospital the increase in weight may at times reveal the stage of the pre-edema or

attention called by the increased blood-pressure. In other cases the patient is brought for examination because of the visible changes in the urine, such as bloody urine, painful micturition or pollakiuria. In still others the pallor, lassitude, anorexia, and occasional vomiting and diarrhœa with thirst may indicate urea poisoning.

In its mildest forms the condition may be without symptoms, may or may not be accompanied by increased blood-pressure and rarely has more than the so-called "pre-edema." It is discovered only when a functional test is made. Because of its frequency and insidiousness such tests should be made for several weeks after recovery from all infectious diseases. The urine, light colored and but slightly cloudy, at first contains moderate amounts of protein. A few white or red blood-cells can be found microscopically. Later an increasing number of casts of all forms but without fat, are found. This form is cured in a few weeks without other treatment than rest in bed.

In moderately severe forms the picture is more distinct. These patients are pale, the skin edema is localized in various parts of the body; the face, sternum, shins, and spine are most frequently the site. The child is tired, has no appetite but is thirsty. The urine is red with a greenish iridescence or a dirty brown. The protein content varies from 3 to 10 per cent. The twenty-four hour quantity is markedly reduced to as low as 400-600 c.c. with a proportionate increase of the specific gravity. The blood-pressure may be normal or but slightly increased. The pulse, slowed, may be arythmic. The condition improves gradually in two to three weeks. During convalescence the blood-cells, and especially the white corpuscles, persist for a long time. In some cases a condition resembling orthotic proteinuria with albumen and formed elements, brought on by change of posture may persist. More frequently such a recrudescence may be brought on by exposure to cold or by acute angina. While most cases recover without further complications, the danger of transition into the chronic "pedonephritis" must be kept in mind. The contracted kidney is rarely seen as a sequel. In the treatment, the milk diet, formerly in general use, is no longer considered necessary. The diet should be largely vegetable, supplying sufficient protein for the requirement of the child. Absolute rest in bed is essential.

The severe form may be "dry" or there may be extreme general anasarca. At the very onset other manifestations may be added to those seen in the milder grades. The most urgent of these is pain in the kidney region probably arising from the tension of the distended capsule. The urine is usually a dark cloudy brown and only occasionally bright blood red. At first it contains vast amounts of the formed elements already described. These gradually become less as the condition persists. The protein content is not greater than in the milder cases. The total daily quantity is decreased rapidly to 200 or even as low as 50 c.c. But in the severe cases the specific gravity is not increased proportionately. Anuria persisting for several days is not uncommon. If there is no edema, the danger of uremia is very great. The blood-pressure is usually distinctly increased. Uremia may be merely indicated or it may appear suddenly, full-fledged. The more common

eclamptic form may occur mixed with the azotemic. The general symptoms are also variable. There may be irregular variations of temperature. The pulse-rate may vary in accordance with the fever or may be much lower. Arythmia is common.

Uremic kidney death or cardiac death may occur suddenly after eight to ten days or even after a longer period.

Prognosis.—But even most serious cases may recover in spite of anuria and uremia. With proper treatment a favorable outcome is characteristic of the glomerular nephritis of childhood. In such cases the fluid in the tissues is often drained off through the kidney, this process is accompanied by manifestations of eclamptic uremia, and as the edema subsides the child feels better. Nevertheless, the convalescent patient is subject to recurrences as in the milder forms and is sensitive to exposure and infections. The protein may reappear on change of posture. Transitions to contracted kidney and pedonephritis may take place.

Treatment.—In the treatment absolute rest in bed is imperative. The treatment of the edema is discussed under nephrosis. The use of urea as a diuretic must, of course, be omitted. Strophanthus, digitalis and camphor are indicated for cardiac weakness. The therapeutic measures combating uremia are discussed in the introductory part.

Great care must be exercised to avoid sudden chilling. When there is but a small amount of protein and an occasional red blood-cell in repeated urine specimens the patient may be permitted to get up unless there is a distinct orthotic reaction. Even in the latter cases it is well to permit a slight amount of exertion and change of posture as the orthotic proteinuria often disappears after two to three days. Nothing is gained by too long a rest in bed. The patient becomes fussy and loses his appetite. The diet similar to that suggested in nephrosis should be prescribed with the tendency to edema and danger of overloading with protein constantly in mind. In hospitals the food should be controlled by metabolism tests including the determination of non-protein nitrogen in the blood. The protein of the diet should be kept very low if the non-protein nitrogen is greater than 100 milligrams. In the home, however, it is generally necessary to be guided by the general condition of the patient and the amount of urinary sediment which very often gives a very accurate index of the nitrogen metabolism. During the first few days a purely carbohydrate diet is indicated. Later milk, which contains about 34 grams protein per litre, may be added in increasing amounts. It may be given in cocoa or Keller's malt soup. Breast-milk may be useful even in older children. Broth should be omitted because of the salt. Gradually light and dark meat and even eggs may be added. In other words the diet, in a general way, should consist of cereals, vegetables, fruit, and fat (unsalted butter). The total amount of liquid offered per day should be the same as the amount of urine excreted the previous day. After the free excretion of urine has been established the patient may be given as much fluid as he will take.

Scarletinal nephritis is by far the most common form of glomerular nephritis, although a pure form of septic interstitial focal nephritis also

occurs with scarlet fever. The frequency of nephritis with this disease varies within wide limits depending upon the nature of the epidemic. Furthermore, there is doubtless a distinct familial predisposition. Excepting for an occasional transitory, febrile or a more persistent hemorrhagic (interstitial) proteinuria, true nephritis does not appear before the end of the second or more commonly during the third week, during the so-called "second scarlet fever" or during later relapses.

It is not known whether this delayed kidney injury is to be laid to toxins in the form of waste products of the mechanism of defense formed gradually during the recovery from the infection, as is suggested by von Pirquet, or whether it is due to the actions of the toxins of the recurring attacks of the disease itself, according to Pospischill and Weiss.

There is no relation between the severity of the primary disease and the frequency of the kidney disease. It is the experience of most men, however, that after cases of scarlet fever without eruption or with very slight eruption the tendency to kidney complication is greater. The post scarletinal nephritis may appear unexpectedly. At times it may be preceded by a slight rise of temperature or the temperature of the exanthem does not become normal. After the nephritis has set in there is moderate fever or irregular variation of the temperature curve. This rise may discontinue in a few days or with recurrence persist for weeks. There is some question whether this fever is due entirely to the kidney condition. Usually there is a tendency to cervical lymphadenitis which usually manifests itself acutely before the kidney condition appears.

The symptomatology, treatment, and prognosis of scarletinal nephritis are identical with those of glomerular nephritis in general. No especial prophylactic measures are known. The old practice of exclusive milk diet is objectionable. Pospischill has demonstrated its worthlessness in a large series of cases in which he gave milk to part and general diet to an equal number. The effect upon the general well-being of the patient is not good. Some authors advise keeping the patient in bed for several weeks after convalescence but this also seems a rather harsh treatment of doubtful benefit. Absolute rest in bed is indicated only when there is fever or when leucocytes or other formed elements are found in the urine or when there is danger of exposure to cold. In other cases the patient is allowed to get up as soon as the temperature remains normal for a few days.

GLOMERULO-TUBULAR NEPHROPATHY (Mixed Form)

Just as glomerulitis may occur with nephrosis so we may have the tubular nephropathy with glomerular affection. In such cases the nephrosis may begin with the hematuria and increased blood-pressure of a glomerular process or both forms may alternate throughout the course and influence the entire disease-picture.

The pathologic findings show both forms of lesions without the definite differentiation seen in the individual diseases and without the typical clinical course. The interstitial tissue shows round cell infiltration and the

interstitial cells as well as the vessel walls show fatty degeneration. Scarification of the glomeruli and even contracted kidney may result.

Etiology.—Glomerulo-tubular nephropathy is the typical kidney disease of exudative children and especially of infants. It is the common sequence in cases of impetiginous eczema and otitis or may follow all of the conditions causing glomerulitis or nephrosis. It rarely follows angina, scarlet fever or diphtheria.

Functional tests and blood chemistry reveal reduction in the amount of water, chlorine and nitrogen excreted. Hypouria may occur. The degree in which each of these functions may be affected varies not only in different cases but also in the course of each case. In the individual case there is also a certain relationship between the protein, sodium chloride and even water retention so that for instance an increase of the sodium chloride intake may disturb the protein function and *vice versa*.

Symptoms.—The disease-picture is, naturally, not uniform. Usually the condition is very serious at first. In many cases severe general anasarca is the first symptom. The extremely pale waxy face is so swollen that the little patient can hardly open his eyes. Frequently the distended skin areas become the seat of impetigo with inflammation of the regional lymph nodes and fever. The patient lies very quiet and gives every indication of being extremely ill. Vomiting may occur and diarrhœa is common. The blood-pressure is increased. Difficulty is experienced in producing perspiration by the use of hot packs or pilocarpin even in rickitic infants. The characteristics of the urine are those described under glomerular nephritis. The persistence and obstinacy of the general edema is very significant. It may persist for weeks but usually passes off with polyuria. After its disappearance the patients often look as atrophic as those recovering from the "flour-feeding injury." During the long drawnout sickness the symptoms of uremia may appear at any time. Very often there is an indication of it in the diarrhœa, vomiting, headache and Babinski's phenomenon, when the edema begins to go down. Similarly the other manifestations encountered in the convalescent stage of nephrosis or glomerular nephritis such as orthotic albuminuria, recurrences on exposure to cold, and reduction of immunity are seen.

Prognosis.—In spite of the severity of the disease complete recovery seems much more common in children than in adults. Nevertheless, it must be remembered that we can speak of complete recovery only when, after the urine is free from protein and formed elements including white and red blood-corpuscles, the metabolism takes care of the full required amount of sodium chloride, protein, and water without producing disturbances of secretion. Some children die of later complicating infections or uremia. A small portion may have a contracted kidney or continue as cases of chronic or pedonephritis.

Treatment.—Apparently, treatment is somewhat more efficacious than in adults. At least I have thought that the straight sugar feeding recommended by von Noorden is of benefit, not only in the acute nephrosis, but also as an ameliorating influence upon the glomerular symptoms. It is

quite possible to feed a three-year-old child very satisfactorily on 250 grams of glucose in fruit juice per day and this may be continued for ten days or more. In some cases diuretics are valuable. During convalescence breast-milk is ideal even for older children for its sodium chloride and protein content is low and thus reduces the tendency to edema. It may be given as a major part of the diet with vegetables and fruit until such time when the kidney can stand the addition of protein and sodium chloride. The particulars of the treatment should be regulated by the suggestions given under nephrosis and glomerulitis.

KIDNEY DISEASES IN INFANTS

All of the various conditions described above, except contracted kidney, occasionally occur in infants. The nephritis frequently encountered in the course of parenteral or enteric disturbances of nutrition especially in intoxication and decomposition is of special interest.

The pathologic findings are not definite. There may be no lesion or only a slight fatty degeneration as in nephrosis. In other cases this fatty degeneration may be accompanied by foci of round cell infiltration in the intestinal connective tissue indicating a septic interstitial nephropathy. Clinically, the most important manifestation is the slight amount of protein, which may, indeed, be lacking throughout. It is accompanied by varying amounts of hyalin and granular casts, kidney epithelial cells and occasional leucocytes and red blood-corpuscles. Edema is rarely present although the face or hands and feet may at times seem slightly swollen. The sudden rise in the weight curve, very significant in infants, is also uncommon. The condition agrees most closely with the description of a septic interstitial nephropathy. Those cases in which there is no blood in the urine are probably very mild tubular nephroses. The classification of the remainder of the cases in which there is no pathologic lesion in the kidney must be left to further study.

The pathogenesis may be due in part to toxins resulting from infection or in part to poisons arising in the intermediate metabolic changes (acids?). Furthermore, the infantile kidney is very easily injured. Usually prompt recovery takes place but the kidney disease may persist for weeks after the disappearance of the causative injury. It is probable that some of the cases, especially the infectious interstitial nephritis, may go on as a form of chronic pedonephritis of Heubner. In the differential diagnosis the transient kidney irritation following alimentary intoxication, the edema and scleredema of the newly born and debilitated infants may be excluded by consideration of the general clinical picture. The edematous form of "flour-feeding injury" may be ruled out by the history and the absence of urinary changes. The treatment of these forms of kidney disease must be dependent on the cause. Typical cases require the same treatment described above. Breast-milk is especially effective.

CHRONIC KIDNEY DISEASE

Chronic nephritis may occur in childhood in the same forms as it does in the adult. Both the large white kidney and the contracted kidney have

been observed in children. Both disease-pictures are extremely uncommon in the first years of childhood. Their frequency increases toward puberty. Their clinical and structural features do not differ essentially from the chronic nephritis of later life and therefore they require no further description here. The only significant thing about the chronic nephritis of childhood is the rarity with which its typical forms appear. This may be due in part to the fact that certain etiologic factors operative in the adult, *e.g.*, gout arteriosclerosis, chronic intoxications, etc., play no part in the diseases of childhood. But even where chronic nephritis develops from the acute type which follows upon infections, its manner of appearance differs as between children and adults. In view of these facts, a distinct advance was made when Heubner defined the chronic nephritis most common in childhood by the term pedonephritis.

CHRONIC NEPHRITIS OF CHILDHOOD (PEDONEPHRITIS)

This disease is not uncommon in children between the second and the fourth years, but is most frequent during school age. In spite of its obstinacy its benign character is its essential and significant trait. Its benignity is illustrated not only in the mildness of its subjective symptoms and in its not infrequently favorable termination but also in the rarity with which its course is aggravated by severe complications. Uremia, retinitis, serious changes in the vascular mechanism, or even marked edema are seen only in exceptional cases.

Etiology.—It is certain that pedonephritis occurs most commonly in the wake of acute infectious diseases and especially of scarlet fever; yet, it is not always possible to trace its early manifestations to their origin and often its genesis remains obscure.

This etiologic difficulty is increased by the fact that its subjective symptoms as a rule are so slight that urinalysis is not suggested. The patient is pale and flabby; tires easily; is more or less fretful, but is usually fairly well nourished. Occasionally the child complains of headache, pain in the side, palpitation, anorexia, and thirst; but more often its general well-being is but slightly disturbed.

Symptoms.—Marked dropsical conditions are never seen. At the most only a slight fulness of the face may be observed from time to time. Changes in the heart and the pulse are neither frequent enough nor distinct enough to support a diagnosis, which must depend almost entirely upon the urinalysis.

The quantity of urine and its specific gravity remain fairly normal and there is but little cloudiness and a small amount of sediment. The proportion of protein is never very high, usually varying between 0.5-2. per thousand. The proteinuria may disappear at times and at others may assume the orthotic form. Structural elements are hardly ever lacking even in the protein-free urine. The findings may be limited for months to a few red blood-cells and an occasional cast. Sometimes hyaline, granular and epithelial casts, with leucocytes, are found in large numbers. Then again, acute exacerbations may result in microscopically bloody urine.

Generally speaking, however, the course is uneventful. For a long time the affected child might be considered normal if it were not for the urinary findings. These with other symptoms of the disease may gradually disappear after they have persisted for years and a lasting recovery may be achieved. A certain part of these cases do not recover during childhood. In still others, the transition to a contracted kidney occurs sooner or later with a resulting aggravation of symptoms. Since it is never possible to predict how the disease is going to terminate the prognosis must always be guarded. The differential diagnosis from orthotic proteinuria has been discussed under that disease.

Regarding the pathologic changes occurring in pedonephritis, precedent to the transitions into the contracted kidney, very little is known. It is probably a matter of small diffuse inflammatory foci in the renal parenchyma.

Treatment.—Treatment is not very affective and hence care should be taken not to do any injury by therapeutic measures. Rest in bed with restricted diet may be ordered to initiate treatment and to permit observation. But the symptoms of disease will outlast rest-cures and dietetic measures continued for months, while the general condition of the child and its measure of resistance will suffer in every way from the pursuit of such methods. Nor is the psychic injury of this sort of treatment to be underestimated. The general daily routine of the patient should not be interfered with any more than is absolutely necessary, in order that his attention may not be too closely fixed upon his illness. Play and moderate physical exercise should be permitted. A mixed dietary, including even meat, is to be recommended. A trial of a salt-free diet extending over a period of several weeks is justified. The use of alcohol or of strong spices, indulgence in severe bodily exercise and the employment of cold baths must be prohibited. Proper hygiene of the skin should be secured by warm baths and relatively warm clothing. A steady warm dry climate, and especially the desert climate of Egypt, seems to have a beneficial influence. Sun-baths may serve as a partial substitute. Karslbad and other similar resorts have also been recommended.

NEPHRITIS WITH CONGENITAL SYPHILIS

In the postmortem examination of congenitally syphilitic children renal changes are very commonly encountered. Probably they are never absent in the syphilitic fetus or new-born. With increasing age they are more uncommon and less important. Apart from some signs of degeneration in the parenchyma, these are of slight degree and characteristically involve the connective tissue framework of the organ and especially an increase of the cellular elements of the adventitia of the vessels. The formation of small cysts, and such indications of retarded development as the prolonged persistence of the neogenic cortical zone, and a diminution of the number and size of the glomeruli are remarkably frequent, but not characteristic features. Compared with the general pathologic findings in such cases, these changes are usually rather unimportant, as is equally true of the

clinical manifestations produced by them. In occasional, but certainly very rare cases, hemorrhagic nephritides of luetic origin, resembling those of the adult, are said to have appeared. The frequent but usually slight proteinuria with but little organized sediment, is completely overshadowed by the other symptoms of the luetic disease. Edema, uremia and other serious complications play no part.

Untreated, the urinary changes may persist for a considerable time, but under specific treatment they usually disappear with other symptoms. The question whether mercury is not at times an etiologic factor, rather than a therapeutic benefit must be considered.

PURULENT DISEASES OF THE URINARY TRACT AND THE KIDNEYS

Cystopyelitis, Pyelonephritis and Renal Abscesses

Pathologic conditions in the urinary organs, the common indication of which is the passage of urine containing pus, are not uncommon in childhood. The fact that these conditions have failed of the attention due their great importance can only be attributed to the alleged difficulties of obtaining a urinary specimen from the young child. Even in doubtful febrile cases this all important diagnostic aid has been neglected. Since 1894 when Escherich called attention to the fact, we know that cystitis and other related inflammatory diseases of the urinary tract are peculiarly common in early childhood. The disorder is most frequently met with during the first year. Toward the close of the first six months it rapidly approaches its greatest frequency. It is not uncommon, however, in the second year, but after that age it again shows a gradual decrease of occurrence with advancing age. It is peculiar in the fact that it appears more often in girls than in boys. Nevertheless, its frequency in the male sex, representing about one-fourth of all cases, is not to be underestimated.

The early observers of purulent urine in young children diagnosed cystitis, but later authors have called attention to the frequent extension of the disease to the pelvis of the kidney. As in the respiratory and digestive tracts, so in the urinary passages, inflammatory conditions are rarely confined to any distinct anatomic division. One part after another of the urinary tract may show well-developed structural changes, and yet diagnostically it is difficult to differentiate a catarrhal affection of the lower urinary tract from that of the upper. The extension of the disease to the calices of the kidney or to the renal tubules may give symptoms that make possible a differential diagnosis. So that cystitis, pyelitis, pyelocystitis and pyelonephritis may be essentially but different stages of one and the same process.

The findings at autopsy are often in sharp contrast with the clinical symptoms and especially with the urinary changes. In spite of a well established pyuria, but slight changes in the mucosa are found. In mild cases, these consist simply of a circumscribed hyperemia and a slight swelling of the mucosa. In one instance these manifestations may be more marked in the pelvis and in another in the bladder. In severe forms the structural changes are more distinct and, in addition to those already cited,

submucous hemorrhages, more or less extensive ulcerations and, more rarely, wide-spread fibrino-purulent exudates appear. If the kidney is also involved it is enlarged, cloudy and soft. Dark red hyperemic spots and yellowish areas containing pus give a mottled appearance to the surface and on section. Occasionally the purulent infiltration is confined to the papillæ or the calices, or it may spread in a wedge-like form toward the cortex. In a word, the condition is one of ascending infection.

A variety of organisms may be responsible for the appearance of pus in the urinary tract. Practically, cystopyelitis due to the colon bacillus takes the first and most important place. In the majority of cases this organism is found in pure culture. Staphylococci, streptococci and gonococci are found with the colon bacillus or alone. The tubercle bacillus is seldom a cause of the disease.

The invasion of the urinary tract by micro-organisms does not necessarily invoke disease. According to Langer and Soldin the streptococcus lacticus is a normal inhabitant of the urinary organs during infancy. The work of Kleinschmidt, Helmholtz and Miliken, however, tends to show that this organism is a contamination and even though found in the catheterized specimen does not necessarily have to get into the urine by way of the blood. The results of infection develop only when the factors of predisposition are present. This predicates, first of all, a marked decrease of the general resistance to infection as the result, for instance, of severe exudative diathesis. In such cases, and still more commonly in severe acute gastro-intestinal diseases, the infection of the urinary tract occurs as soon as the loss of strength has reached a certain ebb and often while the patient is still under observation for the primary malady. In consequence the prognosis becomes much more serious.

Besides these general predisposing influences, the mere diminution of the quantity of the urine, the consequent loss of the continuing flush of waste material from within outward and the retention of small portions of the urine in the bladder, are circumstances which pave the way for infection. Dilatations or constrictions of the ureters or any actual malformation of the genito-urinary organs may induce similar results. The writer has seen several cases in which symptoms of inflammation followed the excretion of urine containing large deposits of uric acid crystals and urates.

Pathogenesis.—We do not know how the bacteria reach the affected area in each and every case. This is entirely clear only when cystitis follows the practice of catheterization. There is no doubt that the infection may pass from without inward. The question whether the tract is traversed in this manner in the majority of cases cannot be definitely determined, despite the fact that a large number of these cases occur in females, in whom the urethra is shorter and more direct. It is generally denied so far as the male is concerned. This suggests a different etiology in the two sexes.

On the other hand, the infection is often believed to be hematogenous. Since the presence of various micro-organisms in the urine is not unusual and since bacteria, and especially colon bacilli, have been found in the circulating blood of infants suffering with intestinal diseases, the hemato-

genous origin of the disease seems well supported. The theory of direct migration of the bacilli from the colon to the bladder appears less probable; experimentally, at least, it requires extensive lesions of the intestinal mucosa. Too little attention has been given to a fourth possibility, *viz.:* infection by the lymph channels, unquestionably the paths best adapted to bacterial transmission. Lymph vessels draining the region of the ascending colon and the appendix and communicative with the lymph channels of the parenchyma of the kidney have been demonstrated. That these channels are actually less complete or even entirely absent on the left side may account for the greater frequency of pyelitis on the right.

Clinical Course.—When pyelocystitis appears in the course of some preëxisting disease its particular manifestations may be lost in the symptom picture of the primary disorder. A relapse during convalescence, or even an unaccountable rise in temperature may indicate the development of some complication.

Even in the event of an apparently primary, idiopathic pyelocystitis the characteristic symptoms of the disease are often absent. Cases are seen in infants in which there is no fever or only a slight rise of temperature. The first intimation of the condition is given by the examination of the urine. In older children there may be some difficulty of micturition. They suffer severely and, crying or screaming with pain, evacuate small amounts of urine at short intervals. As a result, many a child will refuse to go to stool and will soil its clothing although it has previously acquired habits of cleanliness. During micturition the patient draws up his legs, or presses his hands over the bladder region. These symptoms are rare in infants. Tenderness or pressure may be elicited over the kidney and bladder region in a few cases. The diagnosis of a supposedly enlarged kidney is due to the failure to recognize the physiologic low position of the child's kidney and to the increased sensitiveness to pressure. After the second year, the more severe forms of the disease are uncommon. After this period the disorder may set in almost unobserved and may show, from the very first, a more chronic tendency.

Acute primary cystitis follows a more severe course in infancy. It presents the picture of a serious infectious disease. The child becomes restless and fussy and the temperature rises rapidly to 39°-40° C. (102°-104° F.), or more. The fever may be accompanied by convulsions. The pulse and respiration become rapid. Vomiting during the first few days is common. Even though consciousness may not be impaired the child gives one the impression of being seriously ill. It is extremely cross and irritable; cries and often shrieks upon the approach of anyone and objects strenuously to being moved. A more serious condition may follow or persist from the onset indicating the involvement of the pelvis or even the kidney itself. Because of the extreme thirst the child grasps the bottle greedily, only to push it away after taking a few swallows. The loss of strength, hastened by the anorexia and the refusal of food, soon becomes noticeable. The initial period of irritability is followed by depression. The little patient lies absolutely still, save for the rapid respiration and for occasional slow groping

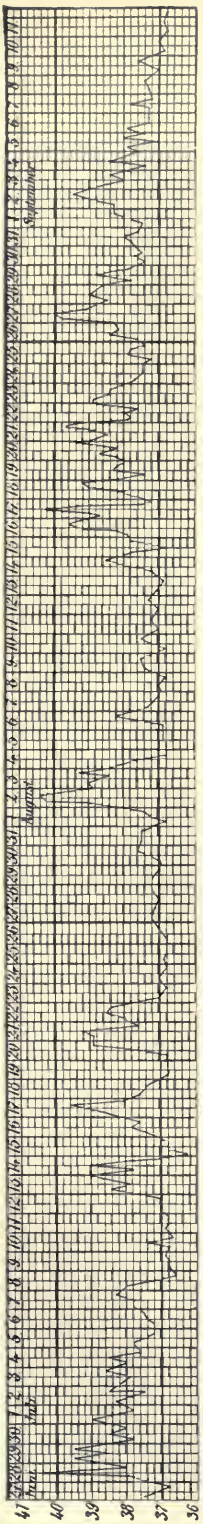


Fig. 111.—J. K., Age ten months. Temperature curve of a case of pyelocystitis.

motions of the hands; the face is contorted with pain and anguish; the eyes are wide open and bright.

Even in the breast-fed infant the digestive functions, as indicated by the consistency of the stools, are almost always affected. It sometimes happens that the picture of a severe acute nutritional disturbance, with typical signs of intoxication, is secondarily induced by the parenteral infection. Toward the end of the first week's illness, an almost characteristic yellowish pallor, together with an increasing flaccidity of the skin, becomes apparent. The abdomen is sunken and the skin loses its elasticity. If the irritation and pain is extreme a reflex muscular spasm of the back occurs and opisthotonus and other signs of meningitis are presented. If this irritation is more marked on the right side, the condition may simulate a perityphelitis.

Course and Termination.—Though the disease runs along with high fever and extreme illness with very gradual remissions and slow improvement, fatal termination is uncommon. But the patient may die after weeks of illness as a result of the continued wasting and the gradual weakening of the heart. In infants a transition to general sepsis is not uncommon. At times even the most serious cases take a turn for the better and after slow convalescence make a complete recovery. In fact, recovery is always gradual and the convalescence is interrupted by repeated relapses as serious as the first attack. Even in the most favorable cases the pus is found in the urine for weeks or months after all other symptoms have disappeared, so that there is a question whether some of the patients ever recover completely. And those that do recover have a certain tendency to relapse that may persist even after childhood. Relapses are most frequent during the first few months of convalescence.

Secondary pyelocystitis may persist longer than the primary disease or in milder cases may disappear when the primary symptoms disappear. Cystitis and pyelocystitis of older children is more liable to become chronic, persisting for years. It is fatal in exceptional cases only.

Diagnosis.—The microscopic examination of the urine is absolutely necessary in making a diagnosis. The fresh urine shows a diffuse cloudiness which, particularly, when shaken, resembles the curling of a pillar of smoke. Hemorrhagic discoloration is uncommon.

While the fresh urine has no characteristic odor, it seems to decompose more rapidly at the temperature of the bed and easily becomes so unpleasant as to be noticeable to the attendant. If infection is due to the colon bacillus, the reaction of the urine is, as a rule, acid, while an alkalin reaction is exceptional. Upon settling or centrifuging, a surprisingly large amount of whitish slimy sediment is often seen. Microscopically this is seen to consist almost entirely of pus corpuscles which often form in balls or clusters. In the acute stage a variable number of erythrocytes is found. A tenacious mucus-like substance may be recognized in cloudy or thread-like masses. A few epithelial cells, usually round and with distinct nuclei, are found among the leucocytes. Casts are generally absent, even when the kidney is affected. The amount of protein corresponds in a general way with the quantity of pus. In the fresh urine, short rods actively motile with rounded ends are seen in the majority of specimens. These grow rapidly upon culture media and are Gram negative. The complete absence of micro-organisms in the stained smear suggests tuberculosis. The differential diagnosis must be established by special stains and guinea pig injections.

These urinary findings alone suffice for diagnosis if the rupture of pus into the urinary tract from neighboring inflammatory foci, *e. g.*, perityphlitis, can be excluded. It is usually impossible to make a diagnosis without examining the urine. The grayish-yellow pallor is very characteristic. The routine examination for protein is insufficient for the amount of protein excreted is usually very small. Every child that is ill with febrile disease the microscopic examination of the uncentrifuged urine is imperative. The urine of infants suffering from acute digestive disturbances should be carefully watched for signs of pyelocystitis, even though there is no fever. This is the only way in which serious errors in diagnosis may be avoided. In older children the differential diagnosis may be confused with typhoid fever or perityphlitis while in infancy the clinical picture, which in the stage of irritability may be complicated by general hypertonia, especially of the muscles of the neck, may be mistaken for that of epidemic meningitis. Unless there are distinct local symptoms, a clinical diagnosis is impossible in infancy. The form as well as the presence or absence of certain epithelial cells, are extremely unreliable aids in diagnosis. An increase of protein in the urine is suggestive of pyelitis or disease of the kidney itself. When tuberculosis is suspected cystoscopy may be of use in older girls.

Treatment.—While much can be accomplished with suitable therapy, in severe cases a good prognosis cannot be insured. The patient should be kept in bed until the urine becomes clear. In infants and younger children the diet must be regulated according to the requirements of the parenteral infection. During the period of acute manifestations in both infants and older children, the symptoms of exudative diathesis must be treated. Liberal diet of coarse foods, fruit and cereals, is advisable to prevent constipation. Highly spiced foods may be irritating. With this, a liberal lavage of the kidney with large amounts of water, has long been used. Too much emphasis cannot be laid upon the importance of this water treatment. In infants the water may be given by stomach tube. In older children, the

addition of fruit juices and other flavoring may help to get in the required amounts. In some cases, more strenuous measures such as drop method per rectum, intraperitoneal or subcutaneous injection may have to be instituted. At least 1½ quarts must be given to smaller infants in the twenty-four hour period and as much more as possible to older children.

If temperature runs high, tepid baths 35° C. (95° F.) are refreshing and stimulating. If the body temperature falls, the temperature of the bath may be increased to 40° C. (104° F.). Internal medication with hexamethylenamine (urotropin), or phenyl salicylate (salol) is often of undeniable value. At present, hexamethylenamine is the more commonly used. Five to eight doses a day of the aqueous solution, representing a total dosage of 0.25-0.5 gm. (3-7½ grs.), for infants, or 1.0-1.5 gms. (15-22 grs.), for older children, may be given. The more obstinate cases are sometimes favorably influenced by phenyl salicylate given in the same doses. Finally, hippol [1.0-1.5 gms. (15-22 grs.) a day], or naphthalin, [0.5 gm. (7½ grs.), three times a day], may be tried. The author has found the alkalinization of the urine by means of liberal doses of potassium citrate, as recommended by Klotz, very effective. No other internal treatment is used. It may be necessary to give three to six grams per day in divided doses to infants and three to ten grams to older children, the amounts to be controlled by testing the urine with litmus. This, together with the increase of the volume of the urine as a result of the ingestion of large amounts of water, is the treatment most commonly used at present. Local treatment by lavage is applicable only in cases of pure cystitis and should be considered only when the disease continues for a long time and other remedies have proved useless. In the administration of lavage all pressure must be avoided. A 3 per cent. solution of boric acid is used first; this is followed by a solution of silver nitrate (1-4000) which should be withdrawn after its use has been continued for five or ten minutes, when the bladder is to be thoroughly rinsed with a physiologic salt solution. Local heat in the form of cataplasms may be employed to relieve pain and tenesmus. Vaccine therapy is of no value in cases of colon infection, but occasionally effects a cure when the disease is caused by other organisms. The treatment of tuberculosis of the genito-urinary tract consists of removal of the affected kidney after determining the function and normality of the other kidney. This applies no matter what portion of the tract is affected.

As a prophylactic measure it is essential that extreme care be exercised in preventing fecal matter from being forced into the urethra. The patient must be protected from cold and exposure.

Hematuria and Hemoglobinuria.—Hematuria, the evacuation of urine containing blood, is a symptom of various diseases of the urinary tract. The urine is abnormally colored, the shade varying from yellowish-red to a dark red or a brownish-red. It is cloudy and often dichroic. In the sediment large numbers of fresh or laked red blood-corpuscles are to be seen. Mild degrees of hematuria can be demonstrated only by means of the microscope.

Apart from hemorrhagic nephritis, hematuria often occurs after trau-

mata in the region of the kidney. It may accompany renal or cystic calculus, or neoplasms of the kidney. It is seen in tuberculosis of the kidney or of the urinary passages; in embolic or thrombotic conditions and in hemorrhagic diathesis. Mild hematuria may be an early symptom of infantile scurvy.

When it is impossible to treat the underlying conditon and this is usually relieved by surgical measures, the hemorrhage itself may be combated to advantage by rest and by the application of cold or, at times, by gelatin, either subcutaneously or by mouth, or transfusion.

Hemoglobinuria is the term used when discoloration of the urine is due largely to free blood pigment and some debris of red blood-cells. The former is usually found as methemoglobin and gives the urine a more or less reddish color. Hemoglobinuria may be caused by such ectogenous poisons as the chlorine salts, phenol, naphthol, hydrogen sulphide, anilin, mushroom toxines, etc. It is occasionally met with as a sequel of such infectious diseases as scarlet fever and malaria.

Paroxysmal hemoglobinuria is a disease in which hemoglobin containing urine is voided at varying intervals of time. The condition is rare, but it does occur in childhood. General symptoms usually usher in the attack. A chill, frequently followed by a rise of temperature; the excessive desire to urinate, pallor, mild cyanosis or icterus, and slight proteinuria are observed. It usually lasts but a few hours, when the patient again becomes normal. Apparently, the paroxysm is most frequently brought on by exposure to cold. The real nature of the disease is unknown. Congenital syphilis is present in the large majority of cases. The treatment consists in the prophylactic avoidance of the injury suspected of exciting the attack. In specific cases it should be directed against the general disease.

DIABETES INSIPIDUS

Diabetes insipidus, while rare, is relatively frequent in later childhood. It is probably congenital, developing gradually. The attention of the parents is usually called to the condition by the excessive thirst rather than by the large amounts of urine. The child may become so thirsty that, if sufficient water is not supplied, he will drink the water of the bath or toilet. There is no doubt, however, that polydipsia is not the primary symptom. The thirst is the result of the enormous excretion of urine. The increased quantity of urine in cases in which the kidney shows no pathology is supposed, according to E. Meyer, to be due to lack of the power of concentration with normal power of diluting the urine. Large amounts of fluid are required, therefore, to remove the debris of metabolism. The twenty-four hour quantity is increased to several litres (quarts). In severe cases, this may even equal the body-weight. The urine is very light colored and the specific gravity may be as low as 1.002 to 1.004. The total solids is usually about normal so that there is no retention. The frequency of micturition and the quantity of urine increases and enuresis is a frequent complication. The blood-pressure is normal.

The general health of the patient is affected because of the unquenchable thirst, which interferes with sleep and makes the child morose. Generally

the need of fluid causes anorexia. The child loses weight at first, and the general development is retarded. The secretion of saliva and perspiration may be greatly reduced so that the mouth and skin are abnormally dry. Because of the large amounts of cold water ingested, the body temperature may be subnormal. Trophic disturbances of the hair and nails are seen.

The duration is usually difficult to prognosticate. It usually persists for years and, in occasional cases, to old age. Spontaneous recovery may take place if intercurrent disease does not cause death.

The diagnosis is made by means of functional tests with sodium chloride or protein. A diet low in these food elements reduces the excretion. When they are again added, the amount of urine increases and both substances, or one or the other, are fully excreted without increasing the specific gravity of the urine or increasing the per cent. of their products. The concentration of sodium chloride and nitrogen in the blood may increase and this may be dangerous.

In the differential diagnosis various conditions must be ruled out.

In diabetes melitus, the specific gravity of the urine is high, it contains sugar and acetone bodies—contracted kidney is characterized by increased blood-pressure and retention. Pyelitis is differentiated by the presence of pus. Primary nervous polydipsia of childhood may be distinguished by the facts that the quantities of urine are not as great and the power of concentration is not lost.

The cause of diabetes insipidus is unknown. Heredity and nervousness may occasionally be factors. Commotio cerebri may bring on the disease. Tumor of the hypophysis or pressure on this organ may be a cause. Occasionally congenital adipose dystrophy may be associated. All of these latter points as well as the transient benefit obtained by the injection of extract of the hypophysis, according to the method of Velde and Johns, seem to point to the possibility of a functional disturbance of the pars intermedia of this gland. Some authors consider injury or abnormality in the region of the infundibulum. It may be possible that the function of the pancreas is involved.

Therapy.—Pituitary extract acts specifically in controlling the condition. A salt-free diet, not too rich in protein, which combines large quantities of fluid with its nutrient elements in the form of fruits, vegetables, milk, thick soup, etc., should be chosen. Attempts should be made to reduce the quantity of fluid taken very gradually. This endeavor demands patience and judgment, since especially in true diabetes insipidus the too rapid reduction of fluid may induce serious results. Measures directed to the stimulation of the peripheral circulation and designed to divert the patient's attention from the conspicuous feature of his general condition should be instituted. Fresh air, recreative exercise, warm or tepid baths and sun-baths may be suggested.

ENURESIS

The normal child acquires voluntary control over the urinary sphincter, during the daytime, by the end of the second year. Occasionally this con-

trol may fail, in the nighttime, until late in the third year. The period at which a child acquires essential habits of cleanliness depends largely upon the care that it receives and the pedagogic skill of its attendants, as well as upon its own physical and intellectual development.

When the influences of training fall upon inherently poor soil the results will naturally be delayed and may never be achieved. Debilitated and imbecile children are slow in learning to make their wants known, while the idiotic never learn. Even serious interferences with physical development occurring at an early period, will delay the acquirement of habits of cleanliness.

There are children, however, in whom none of these unfortunate conditions are found, in whom, notwithstanding the most tedious training, it is impossible to establish the volitional control of micturition or in whom its attainment is delayed for a number of years. There are still others in whom control, acquired with great difficulty, is again lost for an indefinite period as the result of some intercurrent disease.

The sudden and entirely involuntary voiding of large quantities of urine, without evidence of desire to urinate or even of excessive distension of the bladder, is termed enuresis. As a rule, it occurs only at night and is known as nocturnal enuresis. It happens very rarely during the day and only under the momentary influence of strong mental diversion, as during active play, excitement, fear or physical exertion (diurnal enuresis). Disturbances of micturition which are due to structural lesions of the cord, or are incident to loss of consciousness resulting from disease, are not included in this conception of enuresis. Neither does the term apply to those conditions in which the causative factor is a pathologic increase in the output of urine, with consequently frequent desire to urinate as in diabetes, cystitis, lithiasis, etc.

After the exclusion of all above pathology, the remaining condition does not in itself constitute a distinct disease entity. A number of children are found in whom involuntary micturition has not occurred since early childhood but develops after the fifth year. The accident does not happen regularly night after night, but occurs occasionally, or in a short sequence with long intervals of respite. In such cases the enuresis may be but a minor manifestation accompanying unobserved nocturnal epileptic discharges. This relationship should be suspected when such intermissions occur and when attacks of dizziness, peevishness, exhaustion upon awakening, and a bitten tongue are observed. (Pfister.)

Some cases of bed-wetting may be classed as manifestations of juvenile hysteria. In these individuals the symptom first appears in relation to external events and disappears rapidly and for all time under psychic treatment. This type of case is probably well illustrated in the psychic infection which has been known to spread the disorder among a large number of boys in a school dormitory.

The typical and common form of enuresis, however, affects children of all ages up to and even beyond puberty. Most of these patients have either never fully acquired habits of cleanliness, or have broken their acquired

habits in the course of intercurrent disease; a few among them have lost control for no accountable reason. In severe cases nocturnal micturition occurs every night, while in milder ones there may be intervals of days and weeks during which they do not wet the bed. The accident generally occurs in the early hours of the night and usually before midnight, but it may be repeated several times during the hours of sleep. The child is not awakened at all or maybe aroused by the cold wet bedding. The bladder is not necessarily distended. Micturition may occur at the hour when the child has been accustomed to empty the bladder, if it has not been given the opportunity to do so.

A large majority of children who suffer with enuresis belong to this class. The signs common in these cases, which unite them in a single group, do not always stand out clearly but may be looked for and developed by inquiry. Too much stress must not be laid upon any accidentally etiologic factor, but an attempt must rather be made to determine the basis for the fact that stimuli, not ordinarily causative of such a result produce the symptom. In enuresis, it is impossible to discover a local neurosis of the bladder. There are cases in which the local symptom is overshadowed by the general impression of an abnormally predisposed personality. In some instances distinct physical and psychical signs of degeneration are to be noted, while in others they are merely suggested or masked. Among the physical signs, anomalies in the formation of the head, face and teeth are observed, while the nervous and physical indications include exaggerated reflexes, vasomotor irritability, sudden localized sweats and such varied disturbances of innervation as errors of speech, sleep-talking, somnambulism, pavor nocturnus, capricious moods, and a secretive disposition. Finally, as a key to the entire situation, a bad family history may be found. In this picture the enuresis loses the importance of an independent functional disease. It takes its place as one among other symptoms, prominent merely in its frequency and in the attention it attracts, but serving as a sign of hereditary stigmata and degenerative inferiority.

It is in the soil of such a neurosis that the disturbance develops and in which it is cultivated by other and exciting influences. Oxyuris, masturbation, phimosis, vulvitis, balanitis, eczema of the genitalia, adenoid vegetations, etc., may be among such influences. An abnormal soundness of slumber cannot be regarded, in itself, as an etiologic factor.

Prognosis.—A prognosis is difficult in individual cases. The disturbance may disappear at any time without therapeutic interference. In fact, this has often occurred suddenly without known cause, although it is generally a more gradual improvement and related to some definite event or to change of environment. Almost any form of treatment may give surprising temporary results. In severe cases it is not well to put too great faith in the permanency of results. Most cases recover spontaneously during childhood. Occasionally an exacerbation occurs at puberty.

Therapy.—There is no specific therapy, and, for this reason, the list of remedies that have been advocated are endless. Since every successful remedy has only a psychic influence, there should be no trouble in effecting

frequent changes, since the psychic impression soon loses its efficacy. Whether we prohibit liquids at supper-time, or raise the foot of the bed, or prescribe the old favorite fluid extract of rhus glabra, or apply adhesive plaster on the bladder region, the mental suggestion always plays the most important part. The application of the faradic current, of sufficient strength to cause slight pain, or the use of hypnotic suggestion have more lasting effects and are more impressive. Occasionally regular habits are formed by rousing the child at stated intervals. Medicinally, strychnin and, more especially, atropin [0.02 gm. (⅓ gr.) in 10 c.c. (2½ drams) of water; one drop for each year of age], have come into favor.

With all the symptomatic therapy, the improvement of the general condition of the weak and delicate child by suitable nourishment, massage, stimulating hydrotherapy and fresh air, must not be forgotten. By far the best results are obtained by skilful psycho-pedagogic treatment. This method strives to increase the self-reliance of the patient by sensible encouragement and psychic aid, while the attention and the will-power are being trained by various pedagogic measures, Success may be greatly assured by removing the child to new and strange surroundings, as in placing it under institutional care.

VULVO-VAGINITIS

In girls who have not reached the period of puberty various etiologic factors may produce inflammation of the genital organs with pathologically increased secretion.

The desquamative catarrh of the new-born, in which a gelatinous or caseous secretion containing numerous epithelial cells and very few leucocytes, is but one manifestation of the general desquamation of the body surface which occurs after birth.

The mucopurulent secretion occurring with a low order of inflammation in older infants is usually an indication of the exudative diathesis. Contagious impetigo, herpes, and eruptions following vaccination may appear on the mucous membrane of the genitalia and may cause an atypical inflammatory secretion after the more or less characteristic primary eruption has subsided. The genital mucous membranes may also participate in the general eruption of scarlet fever, measles, or small-pox. Inflammation may result from the presence of foreign bodies or of oxyuris, or from masturbation.

Purulent gonorrhœal vulvo-vaginitis is of much greater consequence. Its micro-organism, the gonococcus of Neisser, is identical with that of gonorrhœa in the adult. Marked differences in the pathogenesis, the localization and the course of the disease made it difficult, at first, to recognize its identity; but to-day there can be no doubt that these differences are not dependent upon the organism but rather upon its different habitat incident to the age of its host.

While gonorrhœal infection hardly ever occurs in adult females except through direct contact in sexual intercourse, practically, it is never transferred in this way to the young. The majority of girls who have gonorrhœal

disease acquire it by indirect contact. The infection is carried by polluted hands, by the thermometer, by wash-cloths or by clothing. Careful inquiry will usually point to some adult of the family as the author of the infection. Generally the mother, suffering supposedly with leucorrhœa, is the source, or the infection is carried from the eyes of the child where it had been first implanted and whence it has been transferred to the genitals. The mucous membranes of the external genitals at certain ages are so prone, in fact, to this infection that the disease will pass from child to child in epidemic-like form in hospitals, resorts, public baths, etc., even though the general hygiene is good.

Occurrence.—A predisposition in female children, limited alike by age and by local conditions, is shown not only by the fact that small boys are almost invariably spared from uro-genital blennorrhœa but also by the evidence that in girls there is a period, between childhood and maturity, in which the disease almost wholly disappears. The condition is observed in the new-born of gonorrhœal mothers, the infection supposedly taking place during delivery; but vaginal disease in the child at this period is much less common than gonorrhœal conjunctivitis. In the former locality it is most frequent between the second and the seventh to the tenth years.

Symptoms.—The inflammatory process is localized in the mucous membranes of the external genitals and the vestibule, together with the orifices of Bartholin's glands; the navicular fossa and the labium minora are its most common seats. In contrast with its exhibitions at a later age the vaginal mucous membrane is more seriously affected. The transit of the disease process to the endometrium or even to the tubes is exceptional.

The disease is active after an incubation period of about three or four days with but slight manifestations. Subjective symptoms may be entirely wanting. Pain on walking or sitting and a burning sensation upon urinating occur only when abrasions or manifestations of eczematous irritation of the neighboring skin have been caused by a lack of care and cleanliness. The desire to urinate is at times increased. The general well-being is often undisturbed and the patient continues to look fresh and rosy. The attention is often first attracted to the infection by the yellowish spots of dried secretion appearing upon the linen.

In the acute stage inspection of the genitals may be painful. When the labium majora are spread apart their contact surfaces are found symmetrically reddened and covered with a stringy secretion which forms greenish-yellow crusts upon the external margins. The region of the vestibular glands and the urethral orifice are bright red and covered with a thick yellowish pus which is retained by the swollen and edematous hymen. Large quantities of pus can be expressed from the vagina by the finger introduced into the rectum, showing the marked involvement of the vaginal mucous membrane. The inguinal glands may be moderately swollen. The involvement of the urethra and bladder is rare—the infection of the endometrium to the tubes slightly more frequent; the anal mucosa on the other hand is frequently affected. Gonococci have been found on the nasal mucosa.

In the stained smear of the secretion are found numerous pus cells and

typically located Gram-negative diplococci of characteristic form. Their demonstration is essential to the differentiation of the disease from other causes productive of mucopurulent secretions. Various forms of diplococci are not uncommon in the genital secretions and the diagnosis depends therefore upon their form and position.

Course.—The course of the disease is distinctly chronic. Even though the acute symptoms of inflammation are soon overcome the secretion rarely disappears completely within four to eight weeks; or, greatly reduced in quantity, may continue for months. Cases, in which distinct remissions occur, may persist for years. The most careful and active treatment may not succeed in completely arresting the secretion, but only results in partial recovery. Thus, even though there is no actual danger to life involved, the prognosis is unfavorable because of the long duration of the disease. Complications by extension to the bladder, the internal genitals and the peritoneum are rare, nor is the infection very often carried to the eye even though the case is not given as scrupulous care as it should. The gonorrhœal infection of single joints is a more frequent matter.

Prophylaxis.—Preventive measure should guard young girls from careless contact with gonorrhœal adults or with articles used by them. They should sleep alone. The genital regions should be kept clean by frequent washing. In institutions the infected patient must be isolated. Individual toilet articles are indispensable.

Treatment.—In the treatment of the acute stage rest in bed is essential. To diminish the danger of the spread of the disease it is well to use a T-bandage and to have the patient wear closed drawers when out of bed. The unpleasant results caused by the free flow of pus may be avoided by frequent irrigation of the well separated parts with weak saturations of potassium permanganate either expressed from a piece of cotton or applied by use of an irrigator. With the patient in the cystotomy position, the vulva directed upwards, a 1 to 3 per cent. solution of protargol or 20 per cent. argyrol is instilled into the parts, from two to four times a day, from a cotton tampon, which is then applied externally and left in place for ten minutes. The remaining solution is not removed. Sitz baths, containing an astringent, such as tannin, aid in preserving local cleanliness. Irrigations of the vagina, to which there may be physiologic and pedagogic objections, are not essential. If they should become necessary they may be given with a soft rubber catheter and an irrigator, using a weak solution of potassium permanganate at body temperature. The use of iodoform vaginal suppositories and the dusting of the interior of the vagina with dry sterilized bolus alba have been frequently advised. It is customary to give various balsams, such as santal oil (5-15 drops three times daily) internally. Gonococcus vaccine is useful in the gonorrhœal rheumatism only. The overheating of the body by the use of hot baths is dangerous, intravenous injection of silver salts is useless.

The condition may be said to be cured when after repeated diligent search, following irritation of the vaginal mucosa with strong silver solution,

no gonococci can be demonstrated in stained smears and this must be repeated for several weeks.

PHIMOSIS, PARAPHIMOSIS, AND BALANITIS

It is generally impossible to retract the prepuce sufficiently to expose the glans penis to view. Upon close examination it is often found that the inner surface of the prepuce is closely adherent to the glans even as far forward as the funnel-like meatus. In some individuals the prepuce extends beyond the glans so that it is sometimes difficult to inspect the urinary meatus. Either condition may be considered normal and should not cause the least anxiety. In fact the synechia between the glans and the prepuce are due to congenital and entirely physiologic epithelial adhesion which disappears spontaneously from before backwards during the early years.

Even an apparently small preputial opening does not, as a rule, hinder the evacuation of urine because it distends under the pressure of the stream. The condition is pathologic only when the urine dribbles from the opening, while the child strains and cries, and the free end of the preputial sac is filled and distended with urine. This is due to a stenosis of the preputial canal, which is termed phimosis.

In these rare cases we occasionally find objective symptoms of retention of urine. Less severe degrees of constriction may cause disturbance of function if inflammatory changes develop in the tissues concerned. These may occur when smegma and small portions of urine decompose in the sac and produce irritations, redness and edema of the free surfaces. Then the distal part of the organ may become swollen to an extreme degree. A purulent secretion may be expressed from the narrow slit-like preputial opening, causing extreme pain. This condition is known as balanitis or *balanoposthitis*.

While these typical symptoms are easily recognizable and the functional disturbances caused by them are undeniable, the statements and the interpretations of the mother concerning them must always be accepted with utmost caution. At practically all levels of society the genital organs of the infant receive a special measure of anxious and careful observation. With the exception of the teeth, no other organ is suspected of so frequent and varied pathogenetic influence, nor is the physician more frequently consulted for disorder of any other part. The every day report that the child cries before passing urine has its probable explanation in the fact that the infant often empties the well-filled bladder while it is crying and straining from some other cause.

The opinions of the laity will surprise us the less when we consider how many physicians are prone to consider slight constriction of the foreskin an etiologic factor in severe and varying disturbances, especially of a nervous order, and treat their patients with this view.

The surgical treatment of phimosis is indicated in a small number of cases and, then only, when these cases show severe symptoms of recurrent balanoposthitis or when evidences of chronic irritation appear. In milder cases the prepuce may be stretched by manipulation or with blunt instru-

ments. The adhesions which cause the retention of large masses of smegma may be broken with a probe.

Inflammatory conditions should be treated with applications of a cold boric acid solution or by careful irrigation of the preputial sac.

Before advocating surgical procedure it is well to exclude or treat spasmophilia, for spasms of the urinary sphincter and even eclamptic attacks may be manifestions of this condition. In the differential diagnosis the congenital atresia or stricture of the urethra must also be taken into consideration.

HYDROCELE (Serous Periorchitis)

Fluid exudates, between the parietal and visceral layers of the tunica vaginalis of the testes, are extraordinarily frequent during the first year of life. As a rule the serous exudate is of moderate degree and involves only one side of the scrotum. Hemorrhagic or purulent exudates are very rare. Occasionally the exudate extends up along the spermatic cord or may be entirely localized in the funicular process. The swelling may be present at birth or it may develop gradually soon after birth. It does not cause any special symptoms, nor do any dangers arise from it. Hydrocele is relatively frequent in children who suffer disturbances of nutrition. It may disappear as gradually as it comes, with only indifferent treatment or with none. The skin should be well cared for; the genital region must be kept clean; and any existing intertrigo should be treated. Operative interference is not urgent. If the tumor becomes very large, or distends the scrotum or if it persists for months some measure of surgical aid may be sought, as by puncture or injection of Lugol's solution.

ANOMALIES IN POSITION OF THE TESTES

The migration of the testis in the course of development from the point of formation in the abdominal cavity to its final position in the scrotum is occasionally incomplete at birth and may be halted at any point in its path. If the interruption in its descent occurs within the abdominal cavity the condition is known as cryptorchidism or monorchism. The arrest in its course commonly occurs in the inguinal canal or at its orifice. The scrotum is empty upon one or both sides, while the misplaced testis may often be palpated in the neighborhood of the inguinal canal. This delay in development is not uncommon in the new-born, but the malposition may correct itself in the course of the first year or even much later.

The importance of the malposition, pathologically, lies in the fact that the retained testicle is prone to diseases of various sorts. Fibroid or fatty changes may develop in it or it may become the focus of severe inflammatory processes. In later life, malignant neoplasms are occasionally grafted upon it.

Inflammation of the testicle situated in the inguinal canal may be mistaken for an incarcerated hernia. It is also true that incomplete descent is not infrequently associated with an inguinal hernia and may be a factor in its causation.

Treatment.—If it can be palpated an attempt may be made to bring a retained testis to its normal position by manipulation. If its descent does not occur spontaneously nor in response to such manipulation by the tenth to the twelfth year, surgical measures, either by way of orchidopexy or extirpation, must be considered.

MASTURBATION

Latent voluptuous excitement in the realm of the genital organs may be actualized long before sexual maturity. In fact, its vague indefinite indications may be traced to the earliest infancy. These sensations, peripherally produced, become associated with potential mental processes to constitute the phenomenon of conscious sexual sense much later. The intensity and the period of this development are the subject of great individual differences.

The earliest voluptuous sensations arising in the genital organs are accidentally discovered. Probably they are frequently brought to the child's attention in the course of its play and as a result of its normal inquisitiveness. Just as its attention is attracted to its hands, feet, ears, or umbilicus, so it is led to investigate the genitals. Then too, itching or tickling sensations, brought out by inflammatory irritation or by unaccustomed or poorly fitting clothing, may bring the hands into contact with the genitalia. These accidents may cause spontaneous erections in extremely young boys. Not infrequently, latent sensations may be aroused by the manipulations of older children or unscrupulous adults. If the sensation of the first stimulus is pleasant, naturally it will be repeated and a vicious habit of improper and uncontrolled abuse develops.

It is difficult to form any definite idea of the frequency of masturbation. If mild degrees of habit are not excluded the percentage is likely to be too low rather than too high. While, generally speaking, masturbation is less common among girls, it is rather the rule than the exception among boys; varying, of course with age, environment, and training. Whether masturbation is pathologic under all circumstances is beside the question: there can be no doubt that it may become pathologic in proportion to the degree in which habit is practiced.

The hold which it has upon the child varies greatly. An uninterrupted incline leads from the single or occasional attempt to those cases in which unbounded abuse reigns, when all the thoughts and actions of the child become centred upon the practice early and late. The most unmitigated forms of abuse are occasionally seen in girls still in their early childhood.

In spite of the varied forms in which it may appear, masturbation is easily recognized by the careful observer. It is true that the physician in making a diagnosis is dependent largely upon the report of the persons constantly in contact with the child, who alone are able to observe the patient at all times without attracting the attention of the observed. The hands do not necessarily play a part in masturbation. Especially in girls, the compression and friction of the thighs is often sufficient. Sometimes the genital parts are rubbed against articles of furniture or pressed rhyth-

mically upon the bedclothes. In young children this hardly ever results in a true orgasm or even in any actual acme. When ejaculation occurs in sexually immature boys the material consists of the secretion of the prostate of the urethral gland and the urethral bulb.

The anxious parents are interested primarily in the question of the occurrence and extent of injuries to health resulting from masturbation. To the physician the primary question reverses itself; for excessive masturbation is not so much the cause as the effect of psychic abnormality. Almost all idiotic children and those with marked psychoses masturbate; and even in apparently normal children excesses may develop as the usual result of congenital neurotic tendency or of neuropathic stigmata. Occasional masturbation in healthy children is probably harmless and surely too much stress is usually laid upon the practice in such cases. Major degrees of habit, in which satisfaction is obtained by increasing stimuli and only with extreme exertion of the body and at the expense of the will may injure the nervous system and apparently affect also the function of the heart. The extreme influence which may be induced over the psychic conditions of the patient as the result of overanxious and senseless treatment seem to the writer of much greater consequence. Filled with the consciousness of past errors, tempted to future wrongdoing, frightened by punishment, with vague knowledge of the serious results of the habit, and forced into solitude by fear and shame, the masturbator passes from the pleasure of the moment to the resultant remorse and becomes constantly weaker in will with the renewal of good resolutions which are immediately followed by a repetitional fall. This disturbing inner conflict is probably the principal factor in placing the stamp upon the nature of the masturbator. The picture of the flaccid, tired, self-centred and shy melancholic is not seen in the years of undisturbed and active sexual life.

The milder forms of masturbation are overcome in the course of years and do not affect normal development. There is little hope of permanent recovery for severe cases; the possibilities of therapy are not very great. The affect of any punishment is soon exhausted and corporal punishment is not without a directly bad influence. Actually, masturbation can be prevented only by uninterrupted watchfulness. Restrictive apparatus or mechanical devices have a disadvantage, in that they continually attract the attention which should rather be diverted from the organs involved (Heubner). This desirable result can be achieved only by wise pedagogic influence exerted by an experienced outsider. The child must be entertained and kept constantly busy at physical and mental tasks. Complete tire, by means of work, play and sports, which serve to develop the energy and strengthen the will, cause the child to fall quickly asleep.

Boys should be taught to empty the bladder at night immediately before retiring and in the morning upon getting up.

NEW GROWTHS

In childhood, the genito-urinary organs are relatively often the seat of various kinds of new growths. This is especially true in early childhood.

Not uncommonly we find rapidly growing malignant tumors arising from the kidney or the adrenals. Sarcomata, carcinomata, myxomata, embryonal round-cell tumors and hypernephromata occur. They may develop almost without symptoms. The physician does not usually see these cases until the tumor is of a size to alter the shape of the abdomen. Careful determination of the position, origin and form usually give information from which a correct diagnosis may be made. This may be supported by the urinary findings (hematuria). Cystic degeneration of the kidney and occasionally congenital hydronephrosis may cause similarly large tumors. Smaller benign kidney tumors are more rare.

Tumors may also develop in the bladder and in the genital organs of both sexes. In boys, malignant growths are found, especially in the prostate and testes; in girls, in the vagina and the ovaries. Treatment rests with the surgeon.

VII.

DISEASES OF THE NERVOUS SYSTEM

BY

J. IBRAHIM,

Jena.

ORGANIC DISEASES OF THE NERVOUS SYSTEM

I. Diseases of the Meninges

Pachymeningitis Hemorrhagica Interna.—Several cases of this disease have been observed in infants weakened by disturbances of nutrition, syphilis, rickets or infections. According to Rosenberg hemorrhagic rhinitis of luetic, diphtheritic or other origin plays an important part in its etiology. This condition precedes the pachymeningitis by from two to four months. The connecting link may be a thrombosis in the region of the cavernous sinus. Symmetrical proliferations on the inner surface of the dura over the cerebral convexities in the anterior and middle cranial fossæ appear. These proliferations consist of fine multiple lamellæ which enclose newly developed capillaries or sanguinolent exudates. Large deposits of blood or serous fluid, amounting sometimes to one-fourth of a litre, may gather in cyst-like cavities and are known as hygroma of the dura mater. In more advanced cases the entire brain may be covered with a thick layer of exudate resembling connective tissue.

The clinical manifestations resemble those of internal hydrocephalus. Enlargement of the cranium, with tensity and protrusion of the fontanelle, may develop insidiously or acutely, but usually without the accompaniment of fever. All the symptoms associated with hydrocephalus, as described in a later page, appear.

The fluid withdrawn by lumbar puncture is usually clear but is under supernormal pressure. It may be of a brownish color and slightly hemorrhagic if the pia mater has been damaged and communication exists with the subarachnoid space. This finding is pathognomonic of the disease. Any accidental admixture of blood resulting from the lumbar puncture must be excluded as a matter of course. If this accident has occurred, it is possible to clear the fluid completely by sedimentation or centrifuging. The ophthalmoscopic picture of retinal hemorrhage (Goeppert), which is not constantly present, however, is of great diagnostic value. If it is not possible to remove a larger quantity of fluid by lumbar puncture and to so relieve the symptoms of intracranial pressure, cranial puncture may perhaps enlighten the diagnosis. In this procedure the hemorrhagic exudate spurts out so soon as the dura is entered. The puncture is made through the greater fontanelle and in doubtful cases serves a diagnostic purpose.

Treatment.—The disease is amenable to treatment. Repeated lumbar punctures with the evacuation of large quantities of fluid [50-150 c.c. (2-5 ounces,) or more], are to be recommended. Cranial puncture should be made only when lumbar puncture gives no relief, since it carries with it the danger of hemorrhage. The subcutaneous injection of sterilized gelatine (20 c.c.), coincidently with lumbar puncture, has proved helpful.

2. Tuberculous Meningitis[1]

Tuberculous meningitis is an inflammation of the soft coverings of the brain and cord developing in connection with miliary tuberculosis of these organs. It is always a secondary condition.

Etiology and Pathogenesis.—The disease is most common in early childhood. It is of greatest frequency between the second and the fifth years. In the first year of infancy it is rare. In childhood tuberculous meningitis is almost always a manifestation of general miliary tuberculosis. Children apparently in blooming health often fall victims to this disease. At autopsy some latent tuberculous focus, usually in the form of a caseated bronchial or cervical node, is found to be the point of departure for the disease. It may originate also from bone or joint tuberculosis. Pulmonary foci are less frequently its source, saving possibly in infancy when the bronchial or mediastinal nodes are always affected too.

In most instances infection of the meninges is probably hematogenic and is due to the rupture of some tuberculous mass into a vein. Much more rarely the infective agent may travel through the lymph channels. Occasionally the process develops by continuity from some neighboring organ or focus, as the middle ear, the cranial bones, the vertebræ, or a solitary tubercle of the brain.

A number of factors are recognized which play an important part in determining the development of tuberculous inflammation of the brain membranes, in that they serve as direct causes of the spread of tuberculosis from some latent focus quiescent up to the point of its appearance. In this relation measles and pertussis are prominent. Children survive these diseases, but they do not make a complete recovery and a protracted illness ensues which gradually spreads to the meninges.

Moreover, the results of traumata are of great moment, those directly affecting the head or the entire body being less important than those which act immediately upon an existing tuberculous focus and cause a spread of the tubercle bacilli. It is a well recognized fact that miliary tuberculosis and tuberculous meningitis not infrequently follow operative interference upon tuberculous disease of the hip-joint or for the removal of a caseated cervical lymph node, or for the straightening of a gibbous.

Breast feeding confers no immunity against the disease. At times an increase in the number of cases has been observed in the early spring months.

Pathologic Anatomy.—As a rule, the principal seat of the disease is found at the base of the brain. The entire brain mass, the blood-vessels and

[1] Known in older terminology as basilar meningitis, acute internal hydrocephalus, water on the brain, etc.

nerve roots in this region are embedded in a soft, gelatinous, grayish or, at times, greenish exudate. Accompanying this is a diffuse inflammatory edema which disappears when the brain is removed from the skull. The convolutions seem smoothed over and the entire brain appears to be saturated with the fluid gathered in the distended ventricles. The tubercles, small, gray transparent or opaque nodules, are generally found at the bifurcations of the small vessels and, as a rule, are most numerous in the sylvian fossa. In some cases inflamed foci, in process of disintegration are found on the cortex, constituting a meningo-encephalitis, and occasionally a few large foci in the form of yellow caseated plaques, scattered over the surface of both brain and meninges, are associated with the miliary tubercles. Often solitary tubercles are also found in the brain.

Clinical Picture and Course.—The disease usually has an insidious beginning, with such indistinctive symptoms as anorexia and listlessness. At the same time a loss of weight beyond that which the anorexia would support is noted. Slight rises of temperature may be discovered now and then, the child may cough a little and the anxiously observant mother soon decides definitely that the child is not well. Gradually the change in its demeanor becomes more definite and may be recognized even by the casual observer. The child, formerly happy, friendly, and always ready for play, becomes quiet, fretful and morose, looses its normal desire for diversion, prefers to sit in dark corners, resting its head against the wall or the back of the chair, and requires sleep during the day. It avoids bright light and loud noises and, upon occasion, may become very irritable and violent. In the child who ordinarily meets the physician with active resistance, the quiet indifferent manner with which he permits himself to be questioned and examined may attract the more attention. In typical cases two symptoms now appear which often dictate the employment of a physician, *viz.*, more or less severe and constant headache, particularly in older children, and vomiting. The latter may be active in character, resembling that of gastric disorder, or may be passive, entirely without nausea, and independent of the immediate presence of food. The latter type is the more significant of the cerebral nature of the disease. In infants, and in those cases with coincident intestinal tuberculosis, the stools are apt to be dyspeptic in character. In the typical course, however, obstipation, persisting perhaps to the end, soon sets in.

Even when these symptoms are accompanied by only slight variations in temperature the differentiation of the disease from disturbances of purely gastro-intestinal origin is often very difficult at this early stage. Only a careful consideration of the entire picture of the hereditary predisposition, of the changed demeanor, the loss of weight, and finally, the fruitfulness of treatment makes it at all possible.

Very soon manifestations of irritation appear, and of such a character that the attention of even the inexperienced observer must be directed to the central nervous system. Hyperesthesia of the skin and other sense organs becomes noticeable. The slightest touch causes pain. The sensitiveness to visual and auditory stimuli increases. Hectic flushes appear on

the cheeks and transient erythemata upon the body. A distinct dermatographia causes red spots, remaining for a long time, wherever the skin has been rubbed.

Slight indications of motor irritability develop which are not especially significant in themselves, since they may occur in any febrile affection. Grinding of the teeth, chewing and sucking movements, stereotyped repetitional groping about the head; picking at the lips, the bedclothes, or the genitals; blinking, and deep sighing and yawning, are observed from time to time. The last of these phenomena may prove of great diagnostic value.

At this period the sensorium is usually slightly clouded. The patient becomes soporose but will still give rational responses. It may be possible to demonstrate a slight rigidity of the neck muscles which is particularly noticeable when the head is bent forward. It is necessary to be quite certain that all active resistance may be excluded, making the test, if necessary,

FIG. 112.—Tuberculous meningitis. General tonic spasm. Involuntary movements of the left side of body. Scaphoid abdomen, extreme emaciation.

when the child is asleep. In infants the increased tension and protrusion of the fontanelle is demonstrable.

Even a careful examination of the nervous system gives but little definite information at this time. The pupils are usually narrowed; they react to light only occasionally and dilate again immediately; the reflexes are often increased and at times unequal upon the two sides of the body, a very important finding from the diagnostic point of view. Not infrequently a rigidity of the entire vertebral column accompanies the rigidity of the neck, an especially prominent symptom in older children, who, even at this stage, may be still up and about. Other groups of muscles, also, may be spastic, in which, upon repeated examination, a distinct change of tonus may be recognized. Kernig's sign, consisting in an inability to sit up without bending the knee or to flex the thigh, to a right angle with the body, with the knee straight when lying down, is usually distinct, and is a valuable aid in the recognition of the disease. This is alike true of Brudzinski's sign, a reflex drawing up of the legs upon repeated passive flexion of the neck, especially valuable as a diagnostic feature in children of over two years of age.

At this stage, the vomiting has usually stopped. The child is slightly

delirious at night; the fever remains moderate, with the temperature even normal for days at a time. Lumbar puncture is entirely justified as a diagnostic measure at this period and may change a suspicion to a certainty.

The cutaneous tuberculin reaction of v. Pirquet is positive in the

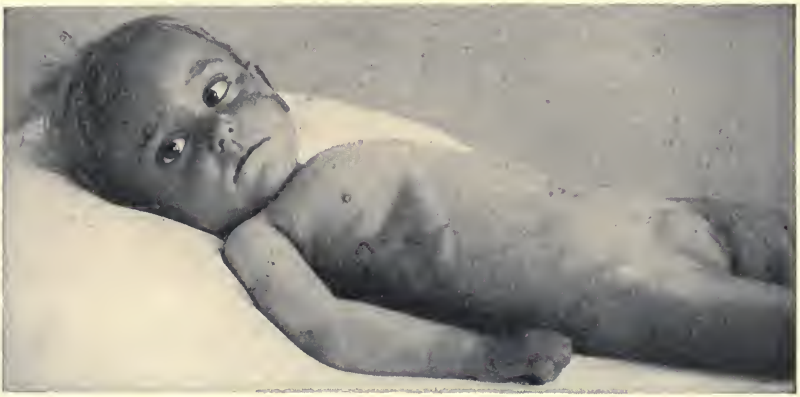


FIG. 113.—Tuberculous meningitis, patient ten months old. Convergent strabismus, somnolence, vacant stare.

majority of cases, but as in other forms of miliary tuberculosis it is occasionally absent.

Manifestations of intracranial pressure gradually become more noticeable, especially when they are caused by an acute inflammatory hydrocephalus. Coincidentally, or at a somewhat later date, paralytic symptoms are observed, and usually in the field of the cranial nerve supply. Doubtless these are due, in the main, to the formation of an exudate at the base of the brain.

The sensorium becomes increasingly clouded. With eyes widely opened the child stares into vacancy, reacts but little on call and is apparently wholly unconscious of its surroundings. Occasionally, it may utter a loud, piercing scream, the hydrocephalic cry, but I have seen a number of cases in which this often recounted symptom is absent. The limbs lie lax, as though paralyzed, or for hours at a time execute continually repeated, automatic movements, which are very often unilateral. Again a child is seen to maintain unusual fixed postures, as with the arms extended in extreme pronation with the hands clenched and strongly flexed. Tremor upon motion is a common symptom at this stage.

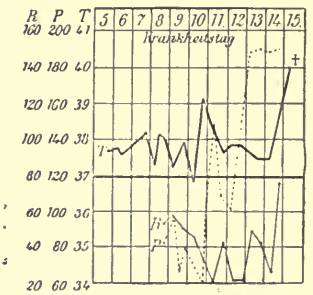


FIG. 114.—Tuberculous meningitis. Five-year-old girl. Typical condition of the pulse.

The pulse deserves special attention. If the disease is observed carefully throughout its entire course, the pulse will always show a remarkable irregularity and infrequency. Sometimes the pulse-rate is reduced from

140 or 160 per minute to 100 or even to sixty. It is irregular and often intermittent. This, as a symptom of irritation of the vagus in consequence of the increasing intracranial pressure, usually lasts but a few days and is replaced by a rapid and often an unusually high rate of pulse

As a rule, paralysis of the cranial nerves is first manifested in the eyes. An inequality of the pupils, strabismus and nystagmus, resulting from involvement of the abducens or the motor oculi, are of earliest appearance. Ptosis of one or both lids is usually coincident. Winking becomes infrequent and a mucoid secretion gathers in the eyes. The facial nerve, also, may be involved.

Examination of the fundus often reveals a slight hyperemia of the papilla, and in older children, when the fontanelle is closed, the typical choked disc of intracranial pressure is seen. Choroidal tubercles are but rarely found upon the closest examination.

The respiration which has been irregular with deep sighing inspirations,

FIG. 115.—Tuberculous meningitis. Sopor, ptosis, strabismus, scaphoid abdomen.

early in the disease, gradually takes on, more or less distinctly, the Cheyne-Stokes type. Long intervals of rest vary with superficial and deep breathing. In typical form, respirations develop, after the respiratory pause, from short and superficial to more and more powerful breathing efforts until they reach an apex, after which they again decline until another pause is reached.

Often marked difficulty in swallowing develops and the danger of aspirating food is imminent. The abdomen, in most instances, is markedly retracted, or of the scaphoid form. Emaciation is extreme. Bed-sores are avoided only by the exercise of extreme care, since the patient loses control of the bowel and the bladder.

The reflexes, which at first may have been markedly exaggerated, are gradually lost. Similarly, the rigidity of the neck which, in infantile cases, may reach a very definite degree, usually disappears before the end is reached.

Thus the patient goes on to final dissolution, which usually comes as almost a relief to all concerned. And yet just at this point in the surely fatal illness a deceptive ray of hope often gleams. Recovery seems about to

set in most unexpectedly. The child suddenly awakens from its deep stupor, appears to recognize its mother, begins to speak, demands food; everything, in fact, seems to indicate the beginning of improvement. But this false attempt at recovery lasts only a few hours or at the most a few days, and the patient again sinks into the former condition.

In almost all cases, clonic epileptiform convulsions appear in the final, and sometimes in the earlier stages of the disease. The pulse then jumps up to a rate of 180, or 200 or more, per minute and the temperature may run very high. The forehead and face are covered with a cold sweat; and life ends with a cardiac paralysis, often in the midst of a convulsive attack.

As a rule, the disease lasts two or three weeks, but may continue longer. The duration of its different stages may vary; in fact, the greatest variations from the type described may occur.

Atypical Course.—Cases which run a rapid course and end in death within a week are seen. This type is especially common during the first two years of life. Such a meningitis may develop as the last phase of a general tuberculosis. Its course is almost an afebrile one in infancy. In such cases autopsy shows a condition more nearly resembling miliary tuberculosis of the meninges than a tuberculous inflammation of the brain membranes.

An atypical onset is not uncommon. Cases commencing with epileptiform convulsions of a frequently unilateral character, or beginning with coma which overshadows the entire disease-picture, are recorded. Others have been described repeatedly which show early monoplegia, hemiplegia, or even crossed hemiplegia, with such other focal symptoms as aphasia. In these atypical forms meningo-encephalitic plaques, or an excessive inflammatory edema over the motor area, or encephalitic foci of the cerebral vertex or in the region of the internal capsule, in connection, sometimes, with an arterial thrombosis, have been discovered.

The absence of obstipation and of scaphoid abdomen is not uncommon in cases during the first year. The characteristically slow pulse may also be wanting. Spinal symptoms, by way of shooting pains at the level of various cord segments, are not unknown. Retention of the urine, in particular, is of note. Long continued remissions extending over weeks or months, have been described a number of times, justifying the suggestion of a temporary recovery and recurrence of the disease. These cases, however, are extremely rare.

Diagnosis.—At the onset, the diagnosis may present very serious difficulties. In the differentiation of the disease from simple gastro-intestinal disturbances, the observation of changes in character and disposition, and of variations in sensory irritability, together with a study of the hereditary predisposition of the patient, are of value.

At a later stage of the disease, the neck sign (page 460), is especially important, but is not pathognomonic in infancy. In older children, Kernig's sign, the sighing respiration, and the discovery of changes in the reflexes are important. So in infants is the bulging of the fontanelle. It should be remembered that apathy, stupor, and even irregularities of the

pulse occur in severe digestive disturbances. The absence of dermatographia contraindicates meningitis.

Typhoid fever may resemble tuberculous meningitis very closely. Diarrhœa, a dry red tongue, and leucopenia are symptoms of typhoid. The diazo-reaction is often present in meningitis. An early meningitis may often be mistaken for typhoid if somnolence and meteorism are present from the first. The absence of rose-spots, of low-fever with severe general symptoms, and of leucocytosis, indicate meningitis, while the evidence in its favor becomes stronger when the neck sign is present. Uremia may have to be considered if the meningitis occurs with oliguria and nephritis.

In its severe forms spasmophilia may produce a picture similar to that of meningitis, with spasm of the neck muscles and of other muscular groups, with variations of the pupils, eclamptic convulsions and even bulging of the fontanelle. On the other hand, it is known that many symptoms of latent and manifest spasmophilia, the facial or Chvostek phenomenon, tetany, and carpopedal spasm, may appear in the course of tuberculous meningitis. In many cases the diagnosis can be made only by aid of lumbar puncture.

Brain tumor may have to be considered when focal symptoms usher in the disease. All transitory symptoms argue against tumor.

Pyelitis, particularly in the first two years of life, not infrequently produces a pseudomeningitic picture which leads to its confusion with tuberculous meningitis. It is essential to examine the urine in all doubtful cases.

Pathognomonic Symptoms.—In every case in which the diagnosis of tuberculous meningitis must be considered, two methods of examination should be applied. 1. The ophthalmoscopic search for choroidal tubercles, a search, however, that is often made in vain. 2. The lumbar puncture, by means of which one is enabled not only to establish the definite diagnosis of meningitis, but also to differentiate its several types.

In tuberculous meningitis the fluid obtained by lumbar puncture is under increased pressure; it is clear or very slightly clouded, or occasionally opalescent, and carries more than its normal content of protein. If possible, 10-20 c.c. of the fluid should be allowed to stand at room temperature, or better in an incubator. Usually a fine cobweb-like fibrin clot is formed which may be spread upon a slide and stained by ordinary methods. This clot contains cells, chiefly mononuclear lymphocytes, and if sufficient care has been taken in its preparation tubercle bacilli can be found in the large majority of cases and even in the early stages of the disease. At times, this result is attained only after a long and tedious search. Similar findings may be made from the centrifuged sediment of the spinal fluid.

The prognosis is almost inevitably of a fatal result. Exceedingly few instances of recovery are known. The not infrequent spontaneous improvement described in the course of the disease will not give the experienced physician much hope.

The treatment, although fruitless in most cases, may accomplish much in relieving the severe suffering of the patient and the anxiety of relatives.

The best care and the greatest possible quiet and rest must be secured. The diet requires particular attention from the beginning. If the diagnosis has been well established and the disease progresses uninterruptedly it is probably more humane to discontinue all remedies tending to prolong suffering. Nutrient enemata and tube feeding should be used only during the early stages. Regular lumbar puncture, repeated once a day or every other day, is often useful in relieving symptoms. A constant, mild measure of congestion of the head, obtained by placing a small rubber band about the neck is said to have proved helpful. Internally, calomel may be given at first in frequently repeated doses [0.03-0.05-0.1 gram; (grain ss-i-iss), every hour]. The inunction of the back of the neck and the shaven occiput with mercurial ointment and the internal exhibition of potassium iodide [1.-2. grams (grs. xv-xxx) each day], may be tried, especially in those cases which offer the slightest suspicion of syphilitic meningitis. Daily painting of the head with collodion containing 15 to 20 per cent. of iodoform has also been recommended. The headache may be relieved by an ice-cap so suspended that it will not weigh upon the head.

When symptoms of motor irritability become prominent, chloral hydrate [1 gm. (grs. xv), per rectum], or, if necessary, hypodermic injections of morphin may be used.

3. Purulent Meningitis (Meningitis Simplex)

Purulent meningitis is an inflammation of the brain coverings and of the brain itself which may be produced by various micro-organisms. It is hardly ever a primary disease, being due either to direct continuation of a purulent process or to metastatic infection.

Etiology and Pathogenesis.—The pneumococcus (diplococcus lanceolatus), is the most common cause of purulent meningitis, with the exception of the meningococcus producing epidemic meningitis which will be considered in a subsequent section. The disease may be caused by the streptococcus, and, more rarely, by the staphylococcus, by Cohen's bacillus, the influenza, typhoid, or colon bacilli, or the bacillus proteus or pyocyaneus. These organisms reach the meninges, either directly, as in fractures of the skull, or by infection of spina bifida; or by way of the blood, by metastases, as when a meningitis occurs as a part of a general sepsis or polyserositis, or probably as a sequela of lobar pneumonia. In the majority of instances, however, it develops by direct contiguity through the lymph channels. The most common path of the infection is from the nose through the cribriform plate into the cranial cavity. Particularly in infancy, meningitis is commonly forerun by a purulent coryza. Second in order of frequency, the middle ear serves as the primary focus of infection. This is especially true of otitis following measles or scarlet fever. A purulent inflammation of the meninges may also come from infective processes in the orbit, from erysipelas of the head, from brain abscess, infected cephalhematomata, etc. The disease is very frequent in infancy and occurs even during the first days of life.

Pathologic Anatomy.—The purulent, seropurulent or fibrinous exudate is heaviest, as a rule, over the convexity of the brain and often resembles a

cap. In meningitis arising from infection of the middle ear, the exudate is occasionally confined to the base. There may be a coincident thrombosis of the sinuses. When the ventricles are filled with purulent fluid the condition is termed pyocephalus.

The Clinical Picture and Course.—The onset of the disease is usually sudden, attended by high fever and terminates fatally within a few days. Occurring in infancy, the picture is often completely overshadowed by the development of convulsions. In the intervals between these early convulsions, the child lies panting and with face distorted with pain. Later, more or less complete sopor develops, occasionally associated with persistent tonic spasms. Paralyses, similar to those which occur in tuberculous meningitis, and even symptoms which suggest involvement of the base of the brain, as inequality of the pupils, strabismus, etc., may appear. A sudden twitching of the body when any part of the surface is tapped may be very noticeable. The fontanelle is tense and bulging, while the rigidity of the neck is merely suggested in some cases and never takes a prominent part in the symptom-complex.

The older the patient, the more nearly do the symptoms approach those exhibited in the adult. Terrific headache and often torturing thirst are chief among these features; but in such cases convulsive attacks also occur. The picture of inflammation of the brain membranes in these more mature patients unfolds itself with great rapidity, but in the same order as the more gradually developed symptoms referred to.

Atypical Cases.—Convulsions may be entirely absent even in infants, and somnolence or stupor and fever, for the time being, may be the only symptoms observable. The disease may run an essentially latent course and if so, especially when it supervenes upon other severe febrile affections, it is very apt to be overlooked. At times the sensorium may remain unclouded for a long time. A rather protracted course, extending over several weeks, has been noted, particularly in meningitis due to an influenzal infection.

The prognosis is usually clear. A few children have survived, but most of such survivals have suffered permanent injuries, in the way of deafness, blindness, psychical disturbances, etc.

Diagnosis.—In infants the examination of the fontanelle is important, for it remains tense even in the intervals between convulsions. Occasionally lobar pneumonia, especially of the upper lobe, may simulate meningitis during the first few days of its course. Careful examination and the speedily ensuing history should clear up the differentiation. Disappearance of the patellar reflexes indicates pneumonia (Pfaundler). In all doubtful cases, lumbar puncture, which in meningitis yields a fluid more or less clouded, or of even a creamy purulent character, containing proteins in large quantity, with large numbers of leucocytes, and in which the causal micro-organisms may usually be demonstrated by ordinary methods of staining, is a final and conclusive test.

Treatment.—Rest, breast-feeding in infancy, and adequate nourishment given by tube if necessary, are important. In older children the dietary should be fluid, non-irritating and in the form of milk, soups, and

fruit juices. The head should be cooled by means of the ice-cap or by cold applications. Narcotics, such as ethyl carbamate or chloral by rectum, when signs of serious motor irritability are present, and inunctions of the nape of the neck and head with mercurial ointment are advised. Internally, potassium iodide may be tried. The continued exhibition of hexamethylenamine, in doses of two to three grams, or thirty to fifty grains a day, is recommended. One-half of this quantity may be given to infants. Hot baths, once or twice a day, are often given. Repeated lumbar puncture can be only beneficial. In pneumococcic meningitis this may be accompanied with the intradural injection of pneumococcus serum. Subcutaneous, lumbar, or intradural injections of optochin (ethylhydrocuprein) may be useful (Wolff and Lehmann). In meningitis arising from infection of the middle ear some hope lies in operative interference.

4. Meningococcus Meningitis or Epidemic Cerebro-Spinal Meningitis.

Meningococcus meningitis is a purulent inflammation of the brain membranes, occurring sporadically and in limited or widespread epidemics. Early childhood is particularly liable to the infection, which is caused by the diplococcus intracellularis.

Etiology and Epidemiology.—Epidemics commonly occur in the cold season and reach their climax in the period between February and May. They usually cease during the summer months. Sporadic attacks, which may well be considered as isolated cases in a very mild epidemic or endemic, have the same seasonal relationship. Severe epidemics frequently continue for five or six months. Evidently the disease is not spread by articles with which the patient has come in contact nor by direct contagion, but is spread by means of so-called meningococcus carriers. Carriers are understood to be persons, who entirely normal in themselves, or but slightly affected, as with a mild form of pharyngitis, yet nevertheless harbor virulent organisms—in this instance in the nasopharynx. It has been shown, by numerous experiences, that close and long maintained contact is necessary to enable one person to acquire this infection from another, or to carry it to a third person. The familial transmission of the disease by fathers, employed in mines or living in barracks, who become carriers of the meningococcus and infect their children, is in point. This tendency is especially observed among families in restricted circumstances. Unhygienic social conditions play their part in the spread of the disease. The viability of the disease germs is prolonged in moist air, although they perish rapidly when removed from the human body.

Individual predisposition appears to be a factor in the acquirement of the disease; children, especially during the first three years, are extremely liable to attack. It is possible that the status lymphaticus affords an especially favorable soil.

Pathogenesis.—Epidemic meningitis develops in the following manner. The meningococci first invade the upper respiratory passages or the nasopharynx alone, where they excite a pharyngitis or a retronasal angina

accompanied by a peculiar swelling of the pharyngeal tonsil. Sometimes, again, the pharynx is spared and the deeper respiratory passages are infected. Laryngitis, bronchitis or even pneumonia may develop, the infection being transmitted to the meninges from these areas. These latter routes are, however, of relatively rare travel by the meningococcus.

Numerous observations made during recent large epidemics, both in Silesia and in North America, indicate that infection of the brain membranes is doubtfully a matter of direct transmission of the disease germs through the cribriform plate, but that they pass into the blood and reach the meninges through the vascular channels. We are justified in regarding meningitis as the result of a frequent and usually a very early metastasis of a meningococcic sepsis. Cases of sepsis of this type, in which the brain membranes have escaped, have been recognized.

Pathologic Anatomy.—When death occurs in the fulminant stage of the disease hyperemia of the meninges is the only finding. In any other stage, a purulent or mucopurulent exudate is found, distributed variably over the outer surface of the brain or spinal cord and without any characteristic preference for either the base or the convexity. The pia is clouded and edematous even in the areas uncovered by pus. The ventricles are usually distended and contain a cloudy inflammatory exudate. Typical Gram-negative diplococci are found everywhere. The brain and cord tissue, particularly in the optic thalamus and the nerve root zones, are extensively involved. In cases in which death has occurred during recovery or convalescence, the disappearance of the purulent exudate has been noted, but the cloudiness and secondary fibrous changes of the meninges persist.

The Clinical Picture.—The clinical picture of meningococcus meningitis has so many characteristic features that it is often possible to make a diagnosis without lumbar puncture and even to differentiate the type from other forms of meningitis.

Its onset, usually sudden, and attended by high temperature and often by vomiting, soon passes into the meningeal symptom-complex. This is distinguishable from that of other forms of meningitis by the exceptional intensity of motor and sensory irritation, as seen in the rigidity of the neck and the vertebral column, by a marked hyperesthesia of the skin, a tenderness of the spinal processes and the extremities, severe headache and backache and, despite the severity of the symptoms in general, by more or less complete retention of consciousness. It is marked by very definite and constant variations and remissions of the fever and of the distinctively meningeal symptoms as well. Even with an occasional continuance of the disease for weeks or months, it may terminate either in death or recovery. Labial herpes is quite typical of the meningococcic form of meningitis. It appears in the early days of illness in from one-third to one-half of the cases occurring in children of over three years of age. Often serious complications, involving both the eye and ear, may accompany or follow the disease.

Special Symptoms.—The disease is ushered in suddenly with high fever, an intense headache, vomiting and, occasionally, with a chill or a

convulsion. The consciousness is usually clouded at an early period. In older children active delirium may ensue. But rarely, and particularly among infants, the onset is more gradual. The frequency of herpetic eruptions in older children has already been mentioned.

In most cases the sensorium clears up during the first week. Long continued sopor is always an unfavorable sign.

The temperature curve shows in the beginning a series of staircase elevations, but later assumes a more continuous line broken by lysis, to which new fever periods succeed. These rises are of varying height and continuity, sometimes persisting for several days, or longer, and giving to the temperature chart a very irregular outline. The curve may even show intermittent or remittent phases in which afebrile intervals of days are alternated with fever periods.

The pulse is frequent and very labile even in convalescence. The respiration is much increased, especially in infancy. Vomiting, after the first few days, occurs only in older children. As a rule, the appetite is seriously

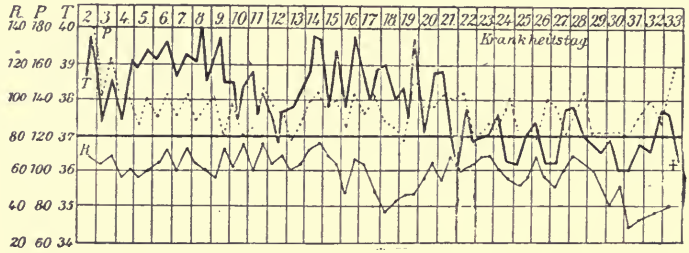


Fig. 116.—Meningococcus meningitis. Six-month-old boy. Protracted course. Repeated attacks of fever. Persistent rapid pulse and rapid respiration. Death occurred in extreme emaciation in the hydrocephalic stage.

impaired. A high grade of cachexia, marked by a scaphoid abdomen, is common in protracted cases.

The spleen is not usually enlarged. Albuminuria is common during the first week, but nephritis, on the other hand, is rare. Even with high fever, the urine is light in color and abundant. Examination of the blood often shows a distinct neutrophilic leucocytosis.

The skin is not only subject to herpes, but to other exanthematous eruptions, sometimes at an early stage, in the form of typical petechia and, again, in the second or third week, resembling measles and, more rarely scarlet fever.

Special Symptoms of the Nervous System.—While convulsions and paralysis of every kind may develop, certain individual symptoms usually assume a prominence which enables the experienced observer to make the diagnosis. The rigidity of the neck is especially marked in many cases. The head is retracted as far as possible and pressed into the pillow. Every attempt to bend it forward develops definite resistance and severe pain. In some instances the effort induces tonic or clonic spasms or trembling of the limbs. Opisthotonos may so affect the muscles of the back as to cause the formation of a true *arc de cercle*. It must not be forgotten, however, that

the rigidity of the neck is not constant and is subject to the same variation as are other symptoms of the disease. It is not present in all cases; it is quite occasionally absent in children under three years of age.

When this important symptom is absent, the observation of other evidences of irritation is of the utmost importance. Among these an excessive sensitiveness, manifested by pain upon the slightest movement of any part of the body, is particularly notable. The legs often seem to be especially sensitive, the small patient screaming whenever they are lifted. Not infrequently passive motions are followed by reflex trembling. In older children an oversensitivity to light and sound is also very apparent. Dermatographia, as in all forms of meningitis, is very prominent.

The reflexes do not follow any definite rule of variation. The skin reflexes are increased at first. Kernig's sign (see page 460), is almost always present and in older children is of some diagnostic value. This is also true of Brudzinski's neck sign.

Fig. 117.—Meningococcus meningitis. Four and one-half-year-old boy. High grade fixed opisthotonos. Sensorium free throughout. Kernig present. Emaciation. Recovery with serum treatment.

Convulsions play a minor part in the disease-picture, excepting at the onset of the attack. General convulsions of later development are often an ominous event. Tonic contractions of individual muscle groups, twitching of the ocular muscles or of the fibres supplied by the facial nerve, are of more common occurrence. Strabismus and inequality of the pupils may come and go. Paralyses of the eye muscles are rare in this disease as compared with tuberculous meningitis. In fact, other paralyses, as in the facial and hypoglossal distribution, and paraplegia of the legs are seen only occasionally.

Finally, it must be remembered that the fontanelle presents a typical appearance; it is tense and even bulging. In infants with other symptoms indistinct, this is an important sign.

The fluid obtained by lumbar puncture is always cloudy, and sometimes is thick and purulent or resembling mucus, so that it will not flow from the needle. In these cases the fluid must be secured from the lumen of the needle. Analysis always shows an increased protein content and usually gives a negative Trommer's test. Microscopic examination commonly discovers masses of polymorphonuclear leucocytes; but at times these are but few in number. Occasionally large numbers of typical biscuit-shaped Gram-negative diplococci, in part of extracellular and in part of the pathognomonic intracellular type are found. In cases progressing toward recovery the spinal fluid becomes clearer with each successive puncture, but months later may still contain leucocytes, an increased amount of protein, but

rarely meningococci. Fibrin forms in the fluid only in the early stages of the disease and again during convalescence.

Complications and Sequelæ.—Rheumatoid, painful swellings of the joints, sometimes mono-articular and again multiple, are among the benign complications of the disease. They usually disappear spontaneously after a few days. Occasionally abscess formation ensues.

Complications of special sense organs and particularly of the eye and ear are of frequent occurrence and serious moment. Optic atrophy, keratitis, and otitis media may be considered true accompaniment of meningitis. Other and especially serious coincidences of meningitis, which should probably be looked upon as primary metastases of the meningococcic sepsis, developing early in the course of the disease, are panophthalmia, iritis and iridocyclitis. They are commonly unilateral and often cause blindness in the affected eye.

An involvement of the labyrinth of the ear is sometimes seen and usually develops in the first week of the disease. It occurs even in mild cases; it

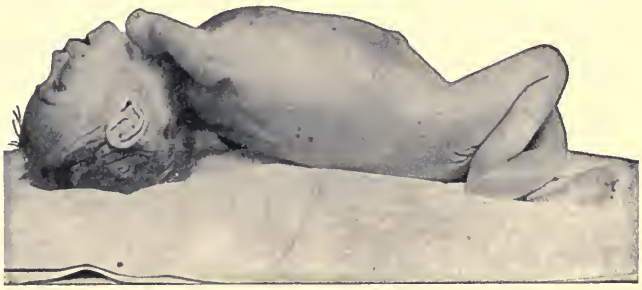


Fig. 118.—Epidemic cerebrospinal meningitis, rigidity of the neck.

is always bilateral. Its mild forms cause subjective noises and phenomena of dizziness; while in severe cases complete deafness results and may be responsible in young children for deaf-mutism.

Pleural empyema, endocarditis and pericarditis are rarer complications. On the contrary, chronic hydrocephalus is a frequent and a very significant sequel. An acute form of hydrocephalus may be noticeable even during the course of the meningitis. After the primary disease has completely disappeared and the spinal fluid is entirely clear and almost protein-free, the head may continue to enlarge and finally presents the picture of a typical hydrocephalus with eyes directed downward and inward, with spastic extremities, and mental retardation and idiocy.

Course and Termination.—There are fulminant forms (meningitis cerebro-spinalis siderans), which terminate fatally within a few hours. In these cases, doubtless, one has to deal with the most severe form of meningococcic sepsis. On the other hand, abortive forms, diagnosed only in the midst of epidemics, may recover in a few days. Usually the disease lasts for weeks or months and exhibits the variations and remissions already described. In the course of these variations it will often be observed that changes in the general well-being and in the nervous and psychic symptoms

do not always conform to the rise and fall of temperature. Recovery may be definitely announced only when there has been a complete subsidence of fever for a long period and when the patient has become psychically normal. A tendency thereafter to headache, irritability, and weakness of reasoning power is occasionally permanent.

In severe cases, death often results during the early days, but more commonly during the second and third weeks. It is usually preceded by coma and convulsions. A fatal termination may occur, however, after weeks or months, either as a result of extreme emaciation and exhaustion, or from hydrocephalus with its common symptoms of intracranial pressure.

The meningitis basilaris posterior, of English and American literature, probably represents a protracted form of meningococcic meningitis, occurring in early childhood, in which severe opisthotonos, projectile vomiting, tonic extensor spasms, and a tendency to post-meningitic hydrocephalus play a prominent part.

Diagnosis.—A diagnosis is readily made only when the chief symptoms are apparent. Of these, special attention should be directed to the rigidity of the neck, to the hyperesthesia, the herpes, the disturbances of consciousness and, in infants, to the bulging of the fontanelle and the tremor induced by passive motion. If the diagnosis of meningitis be once established it is usually easy to differentiate between its various forms. Lumbar puncture should be performed very generally so soon as epidemic meningitis is even suspected, not only for the sake of establishing the diagnosis, but also to permit of preparation for serum therapy should this procedure be found necessary.

During the first or second day an apical pneumonia with meningitic symptoms may result in a mistaken diagnosis. The severe nervous phenomena, the herpes, and the intense headache should obviate confusion with less serious gastro-intestinal disturbances. The slight enlargement of the spleen, the leucocytosis, the herpes, if present, as well as the sudden onset of the attack, are evidence against typhoid fever. The rose spots of typhoid are of no value in the differential diagnosis. Influenza may produce a very similar picture, but the sensorium is usually clearer and the hyperesthesia is less marked. The clouding of consciousness and the leucocytosis are points distinguishing meningitis from acute poliomyelitis during the early days. The high fever, the phenomena of irritation, present even during remissions, the bulging fontanelle, and the rigidity of the neck are evidence against the eclampsia of spasmophilic character. Of course it is often possible to elicit the signs of spasmophilia, the facialis phenomenon, the high electrical reactions, etc., even in children who are suffering with cerebrospinal meningitis.

Lumbar puncture, in view of its lack of danger and the ease of its performance, particularly in childhood, should never be neglected in cases of possible doubt.

Prognosis.—The prognosis is in part dependent upon the nature of the epidemic. Generally the mortality lies between 30 and 60 per cent. A certain number of the survivors suffer such serious permanent injuries as

deafness, blindness, hydrocephalus, and idiocy. In the individual case it is often very difficult to make a prognosis, since either death or complete recovery may result after months of doubt. Restoration is possible even when enlargement of the head has become marked. Long continued unconsciousness, the occurrence of convulsions even after the first week, hemorrhages in the skin, and trismus, are considered unfavorable indications in the prognosis.

Prophylaxis.—Parents of young children should avoid close contact with meningococcus carriers. They should especially avoid the residences of, and the attendants upon, cases of meningitis. Direct transmission of the contagion in hospitals has not been observed.

Treatment.—Rest, the best of care, and the maintenance of a good state of nutrition by means of an adequate and suitable dietary, are of the greatest importance. For this reason breast-milk is especially indicated for infants. On account of the lack of appetite it may be necessary at times to feed small quantities at frequent intervals, thus encouraging the patient to eat. It may be even necessary to feed with the stomach-tube. Especial care should be directed to the prevention of secondary infections, cough, decubitus, etc.

Methods of treatment which seem to give reliable results, particularly in sporadic cases, are hot baths and lumbar puncture. The baths should be given once or twice in the twenty-four hours, after the first few days. They should be at a temperature of from 37°-40° C. (98.6°-104° F.). The patient should remain in the bath for ten minutes and afterwards be kept warm in order to induce perspiration. Lumbar puncture may be done every two or three days. According to the degree of pressure, from twenty to fifty cubic centimeters should be withdrawn. Oftentimes this procedure has a very favorable influence upon the condition in general and particularly upon the nervous symptoms. In the hydrocephalic stage regular lumbar puncture is undoubtedly of value.

As a matter of internal medication, hexamethylenamine may be recommended as a harmless and probably useful remedy. One to three grams (15-45 grs.), a day may be given and if necessary continued for weeks. The customary doses of dimethylamino-antipyrin, of antipyrin, or acetphenetidin may relieve the severe pains. For older children morphin may be used. Analeptics, camphor, etc., may be necessary. In the event of persistent vomiting, atropin is to be advised. Ice-bags, ice-coils, etc., often become burdensome or painful and may be better avoided.

Serum Treatment.—The polyvalent antimeningococcic serum is specific. Its use predicates an absolute diagnosis, confirmed by the microscopic and cultural demonstration of the meningococcus. The presence of a Gram negative, intracellular diplococcus in spinal fluid that gives a positive Nonne test and has a cell count over thirty justifies its use. The demonstration of the organism should later confirm the findings. The serum must be fresh and, as with all serum treatment, the earlier it is given the better the prognosis.

The injection should be both intraspinally and intravenously. Recent

experience has taught that large doses (60-120 c.c.) intravenously are of great benefit. The organism has been demonstrated in smears and culture from the mucous membrane of the nose and pharynx and the intravenous injection clears up these foci at once. In epidemics, when the diagnosis can be made early, this procedure may prevent the advance of the meningeal invasion.

Spinally the serum is given after as much spinal fluid as possible has been withdrawn. It is advisable to give a little less serum than the amount of fluid removed, so that the final pressure is lowered. The serum, warmed to body temperature, is allowed to run in by gravity. Usually thirty cubic centimeters twice in twenty-four hours for six doses, with 60 to 120 c.c. intravenously is sufficient. A second course may be given after twenty-four to thirty-six hours if the symptoms do not subside or if the organism is still demonstrable in the spinal fluid. The results of the treatment should be carefully checked by cell counts, bacteriologic examination, and the clinical course of the case.

If only small amounts of fluid can be withdrawn from the spinal canal, the injection of the serum may be repeated more frequently. Some authors recommend the washing of the spaces by the injection of warm physiologic salt solution under slight pressure and allowing it to run out before putting in the serum. The distention of the meninges by the use of oxygen has been suggested. In small infants, in whom the canal is not easily found in repeated puncture, or if the puncture is dry, ventricular puncture through the fontanelle may be tried. The general care must be personally directed.

5. Serous Meningitis

Serous meningitis is an acute or subacute inflammation of the pia characterized by the formation of a clear serous exudate.

At autopsy only an edematous flooding of the meninges and brain with the serous exudate, and the consequences of such increase of fluid in the flattening of the convolutions and the distention of the ventricles, are noted as gross findings. Microscopically, inflammatory changes in the meninges or in the choroid plexus or in the ependyma are also seen.

The disease is not altogether uncommon among infants, but up to the present time it has not been exhaustively studied. It occurs also in older children. It usually develops as a disease secondary to pneumonia, pertussis, gastro-intestinal disturbances, measles, influenza and other infections. Purulent inflammation of the middle ear is regarded as an important point of origin. Probably this serous meningitis arises from the invasion of the meningeal spaces by very few non-virulent organisms. In some instances it may be the result of purely toxic action.

Clinical Symptoms in Infancy.—In the infant a disease-picture may develop which is entirely dominated by fever and by severe attacks of eclamptic convulsions. This is usually the result of an acute attack. It may terminate rapidly in fulminant form with hyperpyretic temperatures (apoplexia serosa). Again the disease may begin less stormily; signs of disturbance of consciousness and intracranial pressure appear more promi-

nently and its course may resemble tuberculous meningitis very closely. The latter form probably depends largely upon ventricular inflammation and goes by the name, also, of acute hydrocephalus.

Between these two opposite types a number of transitional forms are seen. Fever does not necessarily occur even in cases attended by convulsions. Paralyses do not play any very important part, while pupillary variations, strabismus and particularly spasms of the muscles of the neck and limbs, with increased reflexes, are, on the contrary, frequent. The convulsions are characterized by a very long duration; they may continue for hours or even days. They are attended by wild, terrified cries which the infant frequently utters. A careful observation of the fontanelle is of the greatest importance; it is always tense, unless the infant is extremely cachectic.

The fluid obtained by lumbar puncture, an examination of which establishes the diagnosis, is usually under high pressure. It is clear, but the protein content is definitely increased and upon standing a fibrin clot is formed as it is in the fluid of tuberculous meningitis. Microscopically but few lymphocytes or leucocytes and even very few bacilli (influenza or colon), or cocci (pneumo- strepto- or staphylococci), are discovered. A culture is negative or shows the same organisms. Of course, special precautions must be taken to exclude tubercle bacilli.

Complications and Termination.—The most important complication is undoubtedly spasmophilia (tetany). If serous meningitis develops in a spasmophilic child, especially in conjunction with pertussis, the life of the patient is seriously threatened on account of the frequency and intensity of the convulsions and the danger of the coincident hyperpyrexia. Among other diseases chronic disturbances of nutrition notably affect the prospects of recovery.

In many cases, death results within the first few days. The fatality is less in the subacute form.

Recovery may be complete, but the transition into chronic hydrocephalus sometimes occurs and more or less distinct mental injuries ensue, which are often first recognized when the child reaches school age. According to Quincke, a tendency to recurrent attacks of acute exudation, traceable to angioneurotic causes, may remain throughout life. These attacks may occur in conjunction with traumata, overexertion, excesses, or infectious diseases. In older children, the symptoms resemble those seen in the adult more and more closely. A less distinctly circumscribed disease-picture is to be recognized, in which vomiting, headache, intracranial pressure symptoms, and visual disturbances, choked disc, optic atrophy, etc., have a part. In this way, the picture of a brain tumor which cannot be localized may develop, a condition to be distinguished from this disease only by the tendency of the latter to recovery after lumbar puncture and by its frequent spontaneous remissions. Recently, symptoms have been described which are regarded as incident to an injury of the hypophysis, and include obesity and retarded development of the sexual characteristics, (Goldstein). In acute cases, again, the picture approaches that of tuber-

culous meningitis, from which it may be distinguished only by its favorable termination and by the absence of tubercle bacilli in the spinal fluid.

Treatment.—The treatment of the basic disease is important. This is particularly true of otitis media, if it be present. The methods of treatment recommended in other forms of meningitis, and particularly the inunctions with mercurial ointment, the hot baths and the administration of hexamethylenamine may give satisfactory results. The most important item of treatment remains in lumbar puncture, which may need to be repeated several times. In cases of continued sopor, cold douches applied to the head while the patient is in the hot bath may prove beneficial. Chloral hydrate, in doses of 0.5-1.0 gm. (15-30 grs.), given per rectum, is indispensable for the control of continuous convulsions. If congenital syphilis is to be suspected antiluetic treatment is demanded.

With coincident spasmophilia, all forms of sweat-producing packs are contraindicated, on account of the danger of hyperpyrexia.

Dietetic treatment of spasmophilia, to be described later, must be instituted, together with the therapeutic measures already proposed. In already existing hyperpyrexia energetic hydrotherapy, to cooling results, is urgently indicated.

Meningism, Hydrocephaloid

Under these terms are recognized meningitis-like conditions accompanying which no actual changes can be found. They usually represent a part of the symptom-complex of so-called intoxication in the digestive disturbances of infancy (*q. v.*) In some cases, which begin with high fever, the results are probably due to the non-radiation of heat, as in the so-called summer diarrhœas, while in older children they may be the effects of insolation. In these instances the fontanelle is not tense, but, on the contrary, is usually sunken. A fuller description and an account of the treatment of these conditions are given in the chapter upon Disturbances of Nutrition.

6. The Meningitis of Congenital Syphilis

Congenital syphilis often begins as a leptomeningitis, demonstrable only by the microscope; but it may also cause an acute or chronic meningitis pursuing a course resembling that of the serous type, on the one hand, or that of tuberculous meningitis on the other. In every case occurring in early childhood, in which the etiology is not quite clear, a course of treatment with mercurials and iodides is urgently indicated, particularly when there are other signs of congenital lues or when a positive Wassermann reaction is obtainable.

7. Sinus Thrombosis

Thromboses of the cranial sinuses, developing usually as the result of general sepsis or of local ear infection, do not differ in late childhood from the course they take in the adult. In infancy, however, septic phlebitis of the longitudinal sinus causes a series of violent symptoms, including high temperature, coma, jactitation, tonic convulsions interrupted by short sharp spasms, and tachypnœa. The fontanelle bulges. Edema and local venous congestion are rare.

Sometimes the diagnosis may be made from the spinal fluid which is brownish or greenish-red. It shows a reddish sediment upon standing

consisting of crenated red blood-cells, while the supernatant fluid remains of a yellow or brownish-yellow color (Finkelstein). Hemorrhagic pachymeningitis, which may present similar characters in the spinal fluid, usually gives a different clinical picture (*q. v.*). The disease in question is always fatal.

Operative interference may give relief, but only in cases of otic phlebitis.

II. CHRONIC HYDROCEPHALUS

Under the term hydrocephalus are included all of those conditions in which a considerably increased quantity of fluid gathers in the cranial cavity. These cases may differ etiologically, but their clinical pictures resemble each other so closely that an etiologic diagnosis is oftentime entirely impossible in any individual case. A separate description, therefore, the so-called congenital and the acquired cases will not be attempted.

1. External Hydrocephalus

External hydrocephalus describes the form in which the fluid is found outside of the brain and beneath the dura mater. This form is extremely rare and may be resultant upon hemorrhagic pachymeningitis.

Hydrocephalus e vacuo occurs in cases of retarded development and atrophy of the brain, with a consequent disproportion between the cranium and its contents.

2. Chronic Internal Hydrocephalus

In chronic internal hydrocephalus, an increased quantity of fluid of non-inflammatory origin, gathers in the distended ventricles of the brain. It is a result of a persistent disproportion between the processes of secretion and resorption of the cerebrospinal fluid.

Etiology and Pathogenesis.—The majority of the cases of acquired hydrocephalus must be regarded as the result of meningococcic meningitis or, more occasionally, of serous meningitis. In a few instances the accumulation of fluid may be due to a blocking of the outlets consequent upon an abnormal position of the Aqueduct of Sylvius, or of the foramina of Magendie or Monro. The not uncommon secondary form of congestive hydrocephalus, occurring in cases of brain tumor in young children is probably due similarly to mechanical factors. In this type compression of the veins of Galen may also play a part. In many a case, however, no mechanical explanation is possible and we are entirely at a loss to decide whether it is a question of embarrassed resorption or of increased secretion, and whether the true seat of the disease is in the choroidal vessels or in the ependyma of the ventricles. In so far as these cases are not of meningitic origin, we know only that a certain number turn upon a congenital tendency and that some of them are quite fully developed even at birth. Alcoholism in the progenitor may play a significant part. A considerable share of the acquired cases and probably a notable few of the congenital form are closely relational to prenatal syphilis. Further, a relationship of the milder grades

of hydrocephalus to rickets is apparently traceable, although not yet susceptible of explanation.

Pathologic Anatomy.—The ventricles, as a rule, are symmetrically distended. The lateral ventricles are most markedly affected. They may contain from one-fourth to one-half of a litre of fluid and there are cases on record of far greater quantity. The cerebral hemispheres show the most marked changes from intrinsic pressure. In the more severe stages the surface configuration is progressively obliterated, until the cerebrum comes to resemble two soft distended bladders with very thin walls. The central ganglia and the cerebellum may be affected by the compression or may be simply displaced. The floor of the third ventricle may protrude like a bubble and press upon the optic chiasm. The pyramidal tracts are often injured in consequence either of delayed development or of secondary

FIG. 119.—Congestion hydrocephalus due to brain tumor.

degeneration. Inconstantly, the meninges, the ependyma and the choroid plexus show inflammatory changes or their results; while still other abnormalities are common in congenital hydrocephalus. The coincidence of the disease with spina bifida is most important.

Clinical Picture.—The more severe grades of chronic hydrocephalus, occurring in the first or second year, usually present the following symptoms: The gigantic head immediately arrests attention. Its diameter, distinctly exceeding that of the thorax, may reach sixty to seventy centimeters (24-28 inches). Large blue veins, clearly visible, wind over the scalp, on which the hairs are widely scattered, and are traced downward over the base of the nose and the forehead. When the child cries these veins may swell until they stand out in full relief (Fig. 119). The abnormal and diminutive proportion of the face to the cranium is very striking. The face, small and delicate, suddenly widens at the level of the eyes, giving it a triangular appearance. The superimposed, massive, dome-shaped cranium gives the entire head the form of a huge inverted pear. The dis-

tance from the root of the nose to the hair equals the distance from the nasion to the chin. The bridge of the nose itself is wide and the eyes are spread. The auricles are obliquely placed and the auditory canal becomes a fissure running horizontal to the cranium. The fontanelles are widened to the utmost and the sutures show as wide channels. The frontal bone gapes to the glabellum. Round eminences appear over the temples, representing the lateral fontanelles opened by the flare of the bones. The eyes converge and appear to be pushed downward. The widely distended pupils, more or less unreactive, are partially covered by the lower lids. A crescent of sclera becomes visible between the upper lid and the cornea, and with its dead white accentuates an unnatural stare. The child is playful if approached in a friendly manner; it greets its attendants gleefully and follows them with its eyes. In so doing, a horizontal nystagmus becomes noticeable. The child cannot turn the huge head, which lies like a motionless mass to which the small body is attached.

Fig. 120.—Chronic internal hydrocephalus, age ten months. The condition began during the first months of life. Nystagmus. Pear-shaped head. External auditory canal at an angle. Choked discs. Head is transparent.

If the child becomes excited it screams lustily, contorts its face, and spasms of the limbs, often noticeable at rest, become especially marked in its attempt at slight atactic movements. The reflexes, especially those of the lower limbs, are increased.

The patient takes nourishment freely and rapidly and digests it well, but almost always vomits a part of one or two feedings in each day.

Special Symptoms and Course.—The onset of the disease may occur at any age. By far the greater number of cases are observed during the first two years. If the large head has developed during intrauterine life it may prove a distinct embarrassment to its passage through the parturient canal. More often, children, in whom a congenital tendency is really the cause of the disease, are apparently normal at birth, the enlargement of the head appearing after an indefinite period of faulty development. It generally sets in, however, during the first year. The post-meningitic forms develop gradually as a hydrocephalic stage of epidemic meningitis (see page 472). An increased protein content of the cerebrospinal fluid usually points to an inflammatory origin in the early stages of the disease.

The enlargement of the head which, similarly with an arrest of growth, can be judged only by regular and comparative measurements of the temporal and other cranial dimensions, is usually gradual. There are cases, nevertheless, in which a weekly increase of one to one-and-a-half centi-

meters (0.4 to 0.6 inch), has been noted. In children of two or three years the fontanelles fail to close and the sutures may be reopened.

The form of the head is at first round or spherical. In the more severe cases, lateral protrusions in the parietal and temporal regions, giving the pear-shaped head, and even a marked bulging of the frontal bone and a horizontal placing of the occipital bone appear. Mechanical influences, such as continued pressure in the dorsal position, may cause asymmetrical alterations, a flattening of the occiput, etc. The cranial bones show marked enlargement, with softened edges and thin, compressible, parchment-like centres.

The malposition of the eyes already described is very frequently present, is very typical, and is often an early symptom. Its causes have not been fully determined. In many instances, the flattening of the bony roof of the orbit may play a part. Nystagmus and strabismus are common. The pupils are generally dilated to an abnormal degree, but are rarely unequal in size. Optic neuritis and atrophy of the optic nerve are more common results with increasing years.

Rigidity of the neck, opisthotonos, is not uncommon in post-meningeal cases. Spastic conditions and exaggerated reflexes, especially in the lower limbs, are almost the rule and may develop as early symptoms. Adductive spasms, causing a crossing of the legs, are not unusual, but paralyses are rare.

In the arms, ataxia and tremor are often noticed and occasionally peculiar sterotyped positions are taken.

Convulsive attacks of eclamptic quality may occur, at times, in very young children, but are not frequent and may be absent even in the severest cases.

The psychic functions are not always affected, particularly in those milder cases in which the head ceases to enlarge. Commonly, however, mental deficiency or idiocy, in the most variable degrees, results. The powers of speech and locomotion develop very late. The gait is often spastic and difficult.

In breast-fed infants the physical development is often very good, but in the artificially-fed, nutrition may be extremely faulty. Vomiting is often periodic or of a recurrent type. Dentition is usually delayed.

As a rule, the cerebrospinal fluid, obtained by lumbar or ventricular puncture, is as clear as water; now and then it is slightly greenish or yellow. It contains but little protein, less than 0.1 per cent., and very few formed elements. The pressure of the fluid upon lumbar puncture is markedly increased so long as the hydrocephalus is still progressing; it is usually over 20. mm. mercury. This increased pressure, however, only obtains when the communication between the ventricles and the subarachnoid space remains open. If this is not the case but a very small quantity of fluid is obtained and the tension of the fontanelle is not affected by the withdrawal. In many instances the fluid is replaced with startling rapidity even though a large volume, amounting to 250 c.c. or more, has been removed at one time.

Termination.—Cessation of growth may occur at any stage of the disease. In very few cases may one speak of actual recovery since certain

physical and mental weaknesses usually remain. Spontaneous recovery, by evacuation of fluid as the result of trauma or of rupture through the nose, eye, or ear, is extremely rare.

Death commonly results from intercurrent disease (decubital phlegmon, etc.), from disturbances of nutrition, or even from operative procedures which have been undertaken in the hope of relief.

Chronic Hydrocephalus of Older Children.—A great bar to the abnormal growth of the cranium is introduced by the final closure of the sutures and fontanelles. For this reason alone, hydrocephalus presents an entirely different clinical picture in late childhood. Pressure symptoms are much more distinct and take an immediately prominent place. The entire cranium may grow with comparatively great rapidity as compared with the normal, so that the actual circumference of the child's head may assist the observer's judgment materially in making a diagnosis. By gradual stages of development the disease is related to the serous meningitis of this period of life.

Fig. 121.—Ten-year-old boy, healed chronic hydrocephalus arising during the first year. Circumference 61 cm. (24.4 inches). Moderate imbecility, in school for feeble-minded.

In such cases as these the symptoms are usually ushered in by disturbances of sight and gait. Optic atrophy is much more common than in infancy. It may begin with bilateral hemianopsia and often results in early blindness. More or less severe spasms of the legs with exaggerated patellar and tendo Achillis reflexes follow. The picture typical of Little's disease may develop. Extremely severe paroxysmal headaches continuing for a few days may ensue. Dizziness, vomiting, tinnitus, paralysis of the cranial nerves and, more rarely, tremor and cerebellar ataxia complete the picture. Finally, loss of memory and a state of stupor supervene; in short, a history closely resembling that of brain tumor, from which it may be distinguished only by the increased growth of the cranium and by its tendency to occasional intermissions. Again, the picture may be dominated by epileptiform convulsions with gradually increasing mental weakness, exciting a suspicion of true epilepsy.

Diagnosis.—A fully developed hydrocephalus cannot pass unrecognized. Its beginnings are often hard to distinguish. The physician must proceed cautiously and avoid frightening the parents unnecessarily by a premature diagnosis. Not to fall into the error of overlooking a familial trait, the parental heads should be carefully observed. Among the most important early symptoms are the peculiar position of the eyes and the exaggerated

patellar reflexes. Special precaution is required in reaching a diagnosis in the premature infant, in whom all these symptoms may be present without a subsequent development of hydrocephalus. The relative cranial growth is always indicative, but the absolute measurement of the temporal circumference is equally important. Normally this circumference in the first month is 55.5 cm. (14.2 inches); at three months it is 41 cm. (16.4 inches); at six months, 43 cm. (17.2 inches); at one year, 46 cm.(18.4 inches); at two years 48 cm. (19.2 inches); at seven years 51 cm. (20.4 inches); and at eleven years, 53 cm. (20.8 inches).

In the diagnosis of congenital hydrocephalus the recognition of other malformations is essential.

Syphilitic hydrocephalus is usually of moderate degree and is distinguished by its scaphocephalic form. The Wassermann test should be taken in every case.

Low pressure of the spinal fluid and rapid cessation of its flow, together with other symptoms of hydrocephalus, indicate obstruction of the drainage from the ventricles.

Differential Diagnosis.—Brain tumor, with hydrocephalus due to congestion, frequently escapes diagnosis. Early optic neuritis and especially optic atrophy in hydrocephalus, developing during the first year, is rather indicative of brain tumor.

The rickitic skull is more squarely built; it frequently presents periosteal proliferations of the cranial bones; the fontanelles are less tense, while the actual circumference of the head is but slightly increased. The nervous symptoms are lacking. Hydrocephalus, however, is often combined with rickets, not a surprising fact when one considers the frequency of rickets. Indeed there is a true rickitic hydrocephalus in which the cranium never becomes very large and which is always benign.

Meningitis is distinguished by fever, by the higher percentage of protein in the cerebrospinal fluid, and particularly by the latter's content of fibrin and the presence in it of formed elements and micro-organisms. The diagnostic points in the recognition of hemorrhagic pachymeningitis are detailed on page 459.

Prognosis.—The prognosis in general is very bad. Recovery occurs most frequently in the luetic and post-meningitic forms. The tendency to recovery is best determined by the aid of frequent measurements of the circumference of the skull.

Treatment.—Antisyphilitic treatment is the primary indication in every case of proved or suspected luetic origin. Mercurial inunctions should be continued for weeks and potassium iodide, in doses of 0.25 gm. (4 grs.) a day, for some months, together with injections of salvarsan or neosalvarsan administered in the customary manner.

Lumbar puncture should be made regularly. In the post-meningitic forms of the disease it has often proved useful. It should be repeated every three to six weeks and from twenty to fifty cubic centimeters of fluid should be withdrawn upon each occasion. There are cases in which it may be necessary to puncture from thirty to fifty times or more in the course of

several years. In two cases of idiopathic hydrocephalus the author has secured good results after lumbar puncture by compression of the head with a circular strip of adhesive plaster.

Puncture of the ventricles is easily accomplished through the open fontanelles, and it may prove necessary if sufficient quantities of fluid cannot be withdrawn by lumbar puncture.

A number of surgical methods have been devised for continuous drainage of the ventricles, such as tapping beneath the veins of Galen, trephining the occiput, scarification of the dura, etc. The most interesting attempts are those of Payr, who tried to establish drainage of the ventricles by the free transplantation of blood-vessels to the sinuses or veins of the neck. So far, however, all these attempts at surgical interference have given small satisfaction. The comparatively simple expedient of puncturing the corpus callosum (Anton, Bramann), probably deserves further trial. If the disease is very far advanced, it may be more humane to undertake no treatment that will simply prolong suffering, since the cerebral functions, once lost, are not regained.

Special attention should, of course, be given to the general care and feeding of the child. The infant should have breast-milk, if possible. Decubitus of the scalp must be safeguarded from the outset. When recovery is in prospect the motility of the limbs should be encouraged by massage and warm baths. Imbecility and idiocy may be improved by pedagogic methods of training.

3. Hydrencephaly

This term is applied to any malformation of the brain associated with hydrocephalus e vacuo, and also to a peculiar form of congenital hydrocephalus dependent upon early interference with fetal development. In the latter form, the cerebral hemispheres are converted into thin-walled sacs, but the cranium itself is little or not at all enlarged and may be even smaller than normal (micro-hydrencephaly). This condition is to be suspected when the lateral fontanelles are patent and is more definitely recognized when the head is translucent, transmitting a red glow when it is placed before a powerful light in a darkened room (Strassburger).

III. RETARDATION OF DEVELOPMENT

1. Gross Malformations of the Brain

These deformities are not fruitful subjects for discussion. Of the more frequent types are acephaly, anencephaly, hemicephaly, arhinencephaly, cyclopia, the absence of the corpus callosum, aplasia of the cerebellum, and parencephaly, all of which will be fully described under the head of hereditary ataxia and the cerebral paralyses of children. Congenital hydrocephalus and hydrencephalus have been discussed previously. Congenital idiocy is fully treated in the chapter on the psychoses.

2. Microcephaly

Microcephalus is that condition in which the size of the cranium and of its contents remain distinctly below the normal in size. Almost invariably it depends upon prenatal disturbances. It may be purely a matter of perverted development, the microcephalia vera of Giacomini, which includes types presenting abnormal convolutions, microgyria, and the like; or the fetal brain may be injured and atrophied as the result of inflammatory or vascular lesions, a pseudo-microcephaly. The latter form of microcephaly belongs to the group of prenatal brain paralyses, and will be mentioned again under the head of cerebral diplegias. As a rule, cases of this type present the manifestations of general muscular rigidity, with or without paralysis, athetosis, etc., while the true microcephalics, on the contrary, are often very active and lively. An extremely high grade of idiocy, with a characteristic shape of the head, is common to both forms. The small skull with retreating forehead and particularly large nose suggests the head of a bird. The cranium may be normal as to size at birth, but in this event the fontanelles close abnormally early and the sutures stand out as prominent ridges. Microcephalus is not amenable to treatment.

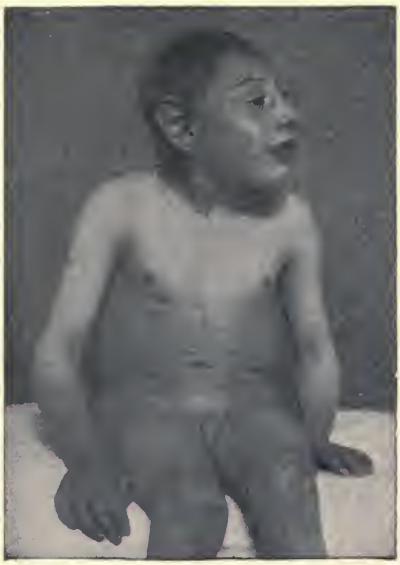


Fig. 122.—True microcephaly (familial).

3. Spinal Bifida (Rachischisis), and Cephalocele

Spina bifida or *rachischisis* is the congenital formation of a fissure in the vertebral canal often combined with a hernia or protrusion of the meninges or of parts of the spinal cord.

A spherical or oval tumor usually lies in the exact median line of the back. It may be sessile or pedunculated. It is most frequently found in the lumbar or sacral region, but is occasionally seen in the neck. It varies in size from that of a hazel-nut to that of a child's head. It is usually filled to distention with cerebrospinal fluid. When the meninges alone participate in the hernia it is termed a meningocele; when the central canal widens at the point of the fissure in the vertebral canal, so that the dorsal portion of the neighboring segments of the cord lies in the sac, a meningo-cystocele. In the most severe form, the so-called myelocele or meningomyelocele, the spinal cord itself is involved in the fissure formation. In this type the cord lies open upon the summit of the tumor. The mass presents three zones. The first is a dark red layer, resembling granulation tissue, lying in the centre,

the zona medullo-vasculosa. This represents the spinal cord and from it the involved nerves pass out. Outside of this lies a thin, grayish, bladder-like membrane, the zona epithelio-serosa, representing the spinal pia mater. This, in turn, is covered by normal skin, the zona dermatica, covering the tumor in varying thickness from the base.

Cephaloceles are similar protrusions of the cranial contents through circumscribed apertures in the cranium. They are situated either on the neck or at the root of the nose and contain prolapsed brain. A ventricle of the brain may be included in the tumor, constituting an encephalo-cystocele.

Spina bifida anterior consists in the very rare formation of a fissure on

Fig. 123.—Spina bifida, meningocele. Area medulla-vasculosa, zona epitheliosерosa, and dermatica distinctly bounded. Paralysis of lower extremities.

the ventral wall of the vertebral canal resulting in the development of a tumor in the pelvis.

Other malformations, such as anomalies in the development of the cerebellum or of the medulla oblongata, club-foot, internal hydrocephalus, etc., are found in association with each other and with spina bifida. Wieland especially has called attention to the frequency of the combination of congenital apertures of the cranium with the tendency to hydrocephalus which they undoubtedly favor.

Symptoms.—The symptoms vary with the location and content of the tumor. Paralysis of the sphincters, bulging of the anal region, more or less distinct motor paralyses, sensory disturbances and malformations of the lower extremities and even of the abdominal wall are frequently encountered. If the child lives, the tumor may become entirely covered with

skin. Death often occurs during the first few weeks as the result of purulent meningitis, or of infections of the skin, the urinary passage, etc.

Diagnosis.—The diagnosis is difficult only when the tumor is entirely covered by skin. Lipoma, teratoma, or a tuberculous abscess may also occur in the median line. An exploratory puncture may be necessary. In spina bifida it is often possible to feel the fissure in the vertebral column and usually the hernia may be partially replaced by pressure.

Treatment.—The treatment is surgical. It gives especially brilliant results in the type of pure meningoceles. When there are serious paralyses present we render no service either to child or parents by the attempt to preserve life. Not infrequently an internal hydrocephalus arises after the removal of the tumor.

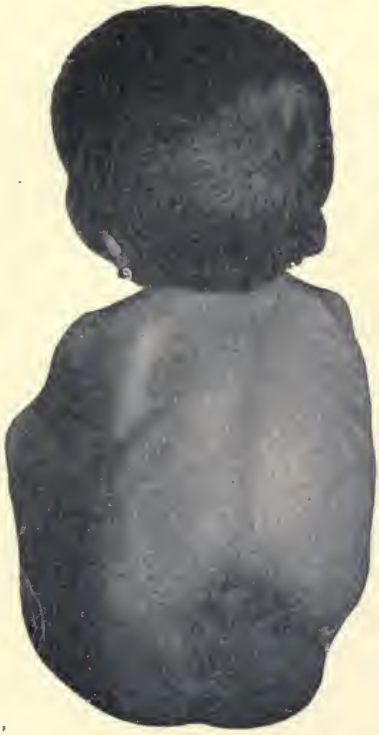


Fig. 124.—Spina bifida occulta. Doubling of the spinal cord. Club-foot.

Spina Bifida Occulta

This term is applied to those cases in which a tumor does not present. The lesion almost always lies in the lumbar region. Its location is sometimes marked by a soft pillow-like bulging and much more frequently by an abnormal growth of hair. At other times its existence may be indicated by scars or by a funnel-like depression in the sacrococcygeal region. In many cases there is a distinct curvature of the spine.

Usually diagnosis may be made by careful palpation or in doubtful cases by the Roentgen picture. Even when no palpable bony aperture exists, malformation of a vertebra and of the lower segments of the cord may be found, the myelo-dysplasia of Fuchs.

Occasionally club-foot or other malformations of the feet, syndactylia, flat-foot, etc., occur coincidently with spina bifida.

The symptoms discernible are pain, slight symmetrical paralyses of the feet and, particularly, disturbances of the sphincters resulting, for instance, in enuresis, dribbling of urine, and incontinence. Frequently, anesthetic zones, leading to the formation of indolent ulcers, are found on the lower extremities. Anomalies of the skin and the patellar reflexes are usually observed. Symptoms often do not appear until late childhood or even after puberty and are easily overlooked. Their late appearance is probably due to a persistent strain upon the tissues intervening between the spinal cord

and the skin, this strain being due to the rise of the cord in the vertebral canal coincidently with the growth of the patient.

Treatment.—The operative removal of the intervening strands of tissue and of compressive fibrous bands which may be present is to be recommended.

4. Hypertrophy of The Brain

Hypertrophy of the brain, or a congenital, abnormally massive and weighty brain is a rarity and is probably not an actual disease. It may produce a clinical picture resembling that of chronic hydrocephalus. No symptoms may be apparent, or these may appear, by way of convulsions, sopor, etc., only after the closure of the fontanelles. The oblique position of the

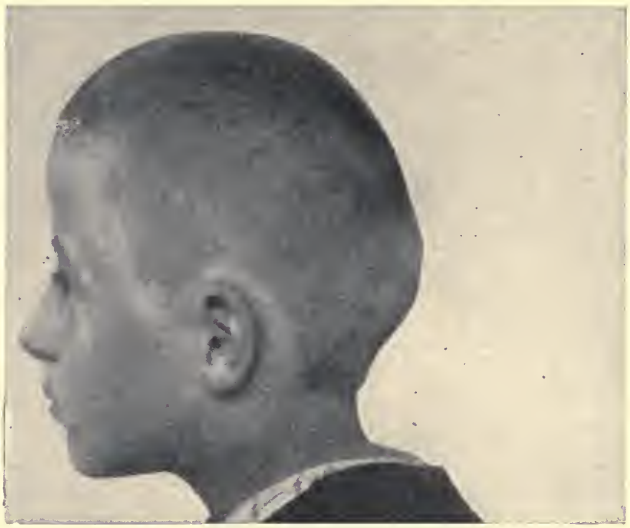


Fig. 125.—Pyrgocephalus.

eyes, typical of hydrocephalus, is absent. The finding of a large quantity of cerebrospinal fluid upon lumbar puncture contraindicates hypertrophy of the brain.

5. Pyrgocephaly, Oxycephaly

Pyrgocephalus (Turm Schädel), is a peculiar deformity of the cranium marked by the steep rise of the parietal and occipital bones with the formation of a high forehead. The entire head looks as though it had grown upward (see Fig. 125), while the base of the skull is often narrow. The sutures, and especially the sagittal, are palpable and form a distinct ridge. The region of the greater fontanelle is often particularly prominent and almost cone-shaped. The pointed head, termed oxycephalus, is a modification of the tower head.

The circumference and size of the cranium, as a whole, may be normal or even small. Signs of rickets are not inevitable. These peculiar and not

infrequent malformations of the head are often combined with disturbances of sight and exophthalmos. Adenoid vegetations of the nasopharynx are often coincidental with them. The visual disturbances are slowly progressive. Generally, they do not become apparent until the end of the first year, but they may suddenly lead to amaurosis. Males are far more frequently affected by the disease than females. Of its nature we have no clear knowledge. The cause of the optic atrophy is supposed to be, in part, an increase of intracranial pressure and in part a matter of direct pressure arising either from the abnormal form of the optic foramen or from the malposition of the internal carotid which is pushed into the posterior part of the optic canal (Behr). Up to the present time no treatment that will influence the disease has been proposed.

6. Congenital Functional Defects of The Cranial Motor Nerves

Congenital Aplasia of the Nucleus; Absence of the Nucleus; Infantile Nuclear Atrophy

Congenital paralyses of the areas innervated by the cranial nerves and, especially, by the nerves supplying the eyelids, the facial muscles and more rarely the tongue are seen in various combinations The most frequent of these is a uni- or bilateral ptosis and an abducens paralysis. The absence of the lachrymal secretion has also been noted in connection with these paralyses The position of the ocular bulb is normal even in unilateral paralysis of the abducens, nor does diplopia occur. In cases of ptosis it is often noticed that the eyelid is raised when the mouth is opened. These paralyses always remain absolutely stationary. They are often observed in the brothers and sisters of a family or in successive generations and, at times, are associated with other bodily malformities.

In some of these cases, the condition is one of aplasia of individual motor nuclei in the medulla (Möbius, Heubner). In others, the defects are due to other causes, dependent either upon insufficient germinal matrices of the peripheral nerves or upon congenital defects of particular muscles, notably in the ocular field,

An early diagnosis is readily made, but definite conclusions as to the nature of the disease and as to its cause, either in the absence of the nucleus or in muscular aplasia, are hardly ever possible. Nor is it always possible to distinguish from these the paralyses, particularly of the facial nerve, acquired in early childhood as the results of birth traumata.

7. Congenital Muscular Defects

Congenital defects of individual muscles are not by any means infrequent. The most common is a defect of the pectoralis; the absence of the trapezius, the serratus magnus, or the quadriceps is more uncommon. Some of the congenital functional defects attributed to the cranial nerves, the so-called nuclear aplasia are really due to muscular aplasia. Occasionally one sees a child in whom several muscles may be absent. In such a case it is characteristically true that the missing muscles are not those of the bilaterally symmetric groups. There are apt to be other coincident malfor-

mations. If defect of the pectoralis is present, other malformations are often found in the arm or chest on the affected side, such as a congenitally high shoulder, an aplasia of the mammary gland, a dystrophy of the ribs, webbed fingers, etc. As a rule the functional failures are slight.

8. Congenital Myatonia

Congenital muscular atony, first described by Oppenheim, is characterized by a bilateral, symmetrical flaccidity and the partial or complete absence of spontaneous movements of the lower extremities, which lie as though affected by a flaccid paralysis. The joints are limp and hyperextensible. The arms are usually less markedly affected and the diaphragm, the muscles of the neck, and those innervated by the cranial nerves in general are not commonly involved. In some instances the intelligence may be somewhat retarded. The patellar reflexes are greatly diminished or entirely absent; the limbs are atrophic and the electrical excitability is reduced quantitatively, even to an entire failure of response, but without giving the reactions of degeneration. Sensation is not disturbed. Gradual improvement and final recovery may take place, but such children usually succumb to some intercurrent affection. The disorder is probably one of delayed muscular development.

Fig. 126.—Myatonia congenita, three and one-half-month-old infant. Typical position of arms, visible insertion of diaphragm.

Diagnosis.—Flaccidity of the limbs and myopathy of a rickitic quality must be taken into consideration. The general affection of all the muscles and the character of the electrical reactions make the exclusion of poliomyelitis and birth paralyses an easy matter. The typical pose of the upper extremities in myatonia, to which the limbs return after any passive change of position, have been described many times (Goelt). This peculiarity may be taken into account in the difficult differentiation of the disease from early infantile spinal muscular atrophy. (Werdnig-Hofmann.)

Treatment.—The treatment should consist in careful massage and if necessary electrical stimulation, together with measures for the substantial improvement of the general health. Thyroid preparations may also be tried.

IV. DISTURBANCE OF THE CEREBRAL CIRCULATION

Anemia and hyperemia of the brain do not exist in childhood as primary symptom-complexes. When there is reason to suspect either one of these errors the treatment should be addressed to the basic disease by the same methods as are applicable to the adult.

Fainting spells, which are rather common in some children, may be especially mentioned. Sometimes they occur very often as a result of a neuropathic or hysterical constitution.

Chronic hyperemia of the cerebral vessels may develop as the result of improper methods of clothing, obstipation, the use of alcohol, habitual working by the strong light of kerosene lamps, etc.; and it may disappear with the removal of the cause.

Cerebral hemorrhages are very rare in children. Meningeal hemorrhages, occurring during the passage of the infant head through the parturient canal, are discussed elsewhere under Diseases of the New-born. Other forms of hemorrhage are practically always of traumatic origin and present symptoms analogous to those seen in the adult. Aside from conditions arising within the brain itself, intracranial hemorrhages occasionally occur in infectious diseases, among which pertussis and purpura are of the most common causal relation. If the child escapes death as the result of the lesion, the consequence may be the development of a spastic infantile hemiplegia. Recently, cases of meningeal hemorrhage occurring in older children and followed by recovery have been recognized. In these instances, a meningitic picture is added to the acute symptoms which attend the lesion, a condition beneficially influenced by repeated lumbar puncture which reveals a hemorrhagic spinal fluid.

Embolism of the cranial vessels is somewhat more common. In this event, the circulating toxins attendant upon the infectious diseases are the chief etiologic factors. The accident occurs most commonly during or after diphtheria and scarlet fever, more rarely after pneumonia, measles and articular rheumatism. The symptoms resemble those seen under similar conditions in the adult; an acute apoplectiform onset, with convulsions, sometimes of unilateral character, and coma. Focal symptoms, such as aphasia, hemiplegia and sensory disturbances follow. Embolism rarely terminates fatally, but usually leaves a typical cerebral paralysis. Complete recovery is also possible. Its differentiation from encephalitic processes may be very difficult or even impossible. The treatment is similar to that of encephalitis.

Thrombosis of the cranial vessels is very rare. Occasionally it occurs as a result of marasmic conditions. Infective sinus thrombosis is discussed above.

CONCUSSION OF THE BRAIN (Commotio Cerebri)

Concussion of the brain is indeed rare as compared with the numerous traumata that affect the cranium of the child. It occurs usually only after the first year. In severe degree its course does not differ from that in the adult and almost always gives a good prognosis. Loss of consciousness, vomiting, a slowing of the pulse, with later loss of memory of the occurrences which immediately preceded the accident are its chief symptoms. Sometimes transitory aphasia and retention of the urine occur.

The treatment consists in bodily and mental rest, the application of

ice-bags to the head, due attention to the bowels and the kidneys, catheterization if necessary, and careful observation of the heart action.

Mild forms of concussion are observed in which the traumatic origin may not be known or is discoverable only upon careful inquiry, since the patient often suffers a loss of memory and witnesses to the accident are apt to be silenced by fear. In such comparatively trivial cases, dizziness, headache, apathy, vomiting, tremor and other cerebral symptoms may appear. Again, rest is the most important therapeutic measure. Physical and mental exertion should be avoided for some time after the disappearance of the immediate symptoms.

Traumatic Pseudomeningocele.—This accident occurs only in children, and to its occurrence it is necessary that the injury to the cranium cause a tear in the dura as well as a fracture or fissure of the bone. The appearance of cerebrospinal fluid between the fragments of bone and the scalp follows usually within a few days. Not infrequently a pulsating, distinctly fluctuating tumor, more or less distended with fluid and covered by normal skin, develops gradually. It does not contain meninges or brain substance, although, exceptionally, traumatic encephaloceles have been reported. The bony fissure at the base of the tumor expands to a larger opening, with raised edges which may be palpated if the sac is not tense. If the trauma has involved simultaneously an injury to the brain substance a communication with the ventricles through the cranial fissure may be established. This is indeed a fairly frequent occurrence. Of course, in such cases paralyses, epileptiform phenomena, and other cerebral symptoms commonly develop. Gradual spontaneous repair has been recorded, but usually there is a tendency to progressive growth.

Diagnosis.—Cephalocele is congenital and occurs either at the root of the nose or in the cervical region. The pulsation, the respiratory fluctuation of the swelling, and the fact that it can be reduced by pressure, exclude cephalhematoma, dermoid cyst, atheromata and abscess. Unless the history is known, it is more difficult to exclude cavernous angioma or soft sarcoma of the dura which have eroded the cranial cap. The temporary disappearance of the tumor after lumbar puncture is characteristic of meningocele (Schindler).

Treatment is not very promising. Puncture does not induce healing and may sometimes make the condition worse. A cap may be worn as a matter of protection. A plastic bone operation to cover the defect may be tried.

V. ACUTE ENCEPHALITIS

Acute encephalitis is an inflammatory disease of the brain, affecting chiefly the gray matter. It is most frequent in early childhood.

Etiology.—Secondary encephalitis complicates certain infectious diseases. It is especially common in pertussis, scarlet fever, influenza, and diphtheria; more rarely in pneumonia, typhoid, cerebrospinal meningitis, erysipelas of the scalp, or umbilical sepsis.

It is the result either of direct injury by the specific disease germ, or of

the indirect action of bacterial toxines. A toxic encephalitis, due to chemical agents, as in lead poisoning, is also known.

Primary polioencephalitis (Strümpell), undoubtedly occurs sporadically and at times epidemically, in the course of an outbreak of poliomyelitis (*q. v.*).

Pathologic Anatomy.—All parts of the brain and the medulla may be affected. Small localized areas and, more frequently, wide areas of diffused inflammation develop. The points of predilection seem to be the basic ganglia and the cerebral cortex. The disease is not confined to the gray matter. The changes first affect the smaller blood-vessels, producing a cellular perivascular infiltration of the vessel walls, with hemorrhages and thromboses and subsequent degenerative changes in the ganglionic cells. Microscopically, the meninges may also show inflammatory changes. These alterations may be too minute to be seen macroscopically; but on the other hand, numerous small yellowish foci or even hemorrhagic spots, the so-called flea-bite encephalitis, may be visible. In still other cases, larger foci of softening appear, as a result of which considerable sections of the brain may show a cream-like consistency. Such changes are seen only in those rare cases of encephalitis which are rapidly fatal. In surviving cases, subsequently coming to autopsy, contraction and sclerosis of the affected portions of the brain, or scars, cysts, and yellow foci of softening are found as the end results. Thus, in the less characteristic final stages of the disease, changes occur which are scarcely distinguishable from such purely vascular lesions as embolism, thrombosis or apoplexy.

Symptoms and Course.—The onset of the disease is usually acute and attended with high fever, convulsions and disturbed consciousness. These convulsions may take an eclamptic form or may have the quality of tonic extensor spasms. They sometimes involve the laryngeal muscles and are accompanied by rigidity of the neck. Deep sopor is usually present from the beginning. The pulse-rate is greatly increased, and respiration is occasionally of the Cheyne-Stokes type. In pure cases of polioencephalitis in infancy the fontanelle does not bulge. The eyes often show a persistent deviation to one side and upward.

This serious condition, which is commonly mistaken for meningitis, may prove rapidly fatal, but, as a rule, the patient survives. Recovery may occur within a week or after several days. A great variety of disturbances may remain depending, as to their nature, upon the location of the encephalitic focus. These sequelæ are more fully discussed in the chapters upon cerebral paralyses of infants. A change toward recovery is usually signalled by a fairly rapid fall of temperature, following which a hemiparesis usually develops. Fever, coma and paralysis may persist for weeks, and focal symptoms, as aphasia, frequently tremor, ataxia or paresis of an extremity or of the facial nerve, become apparent very gradually. Since the disease may be localized in the pons or the medulla, a resulting crossed paralysis, hemiplegia alterans, or a bulbar paralysis, is possible. Such cases, however, are extremely rare.

The onset of the disease, although acute, is not necessarily abrupt. The fever, vomiting, and headache may be mild. The convulsions and the

resulting paralysis may be the chief symptoms. In fact, an extremely insidious form of attack has been observed, resembling more or less the clinical course of a brain tumor, in which, however, the spastic hemiparesis or the hemiataxia may remain or disappear entirely, to be followed, perhaps, by epilepsy.

Again, the course of the disease may develop by a staircase of exacerbations, the vomiting, headache, strabismus, fainting, or paresis of one arm, appearing in turn, and after an interval of days or a week the more turbulent course, already described, setting in.

Diagnosis.—In some cases it may be impossible to distinguish the disease from cerebral embolism. A high and more continuous fever indicates encephalitis. Meningitis may be excluded if increased tensity of the fontanelle is lacking and by the demonstration of the freedom of the cerebrospinal fluid from inflammatory products. Encephalitis may be regarded as probable as soon as focal symptoms and particularly those of a hemiplegic character appear. Unilateral convulsions affecting repeatedly the same side are also of diagnostic value. In the event of a very insidious course, brain tumor or cerebral lues is to be suspected. A choked disc and a progressive development indicate the former; the absence of pupillary reactions, and the beneficial results of antisyphilitic treatment, indicate the latter. If the Wassermann reaction is negative syphilis is improbable.

Prognosis.—The prognosis is very bad in respect of the fact that more or less severe physical disabilities remain and that frequently such psychic injuries as idiocy and epilepsy follow.

Treatment.—Rest; the application of ice-caps, ice-water packs, Leiters' coil, or other cooling devices, to the head; and the complete evacuation of the bowels with calomel [0.03-0.1 gm. (grs. ss-iss), every two hours], are the only attempts at treatment that can be made during the first few days. Local and general blood-letting have been frequently recommended. One or two leeches over the mastoid process on the side of the suspected focus may be tried. In the event of high fever, hydrotherapeutic measures, assisted, if necessary, by acetylsalicylic acid, antipyrin or quinine, given by enema, are useful. If convulsions are long continued or persistent restlessness obtains, chloral hydrate, ethyl carbamate, or even bromides are recommended. Lumbar puncture does not seem to have any favorable influence upon the symptoms and should be employed only for diagnostic purposes or in case the fontanelle is very tense. The feeding is a very important and often a very tedious matter; small quantities of concentrated nutriment, frequently given, and sometimes by means of the stomach tube, being required. It is hardly necessary to say that energetic antisyphilitic treatment should be instituted at once if a luetic cause is even suspected. After the acute stage of the disease has subsided, the treatment is the same as in other forms of the cerebral paralysis of childhood.

APPENDIX

Acute cerebral tremor (Zappert), is a disease which is seen in children during the first year of life. It occurs more frequently in boys. It is characterized by a rapidly developing tremor of the limbs and neck, of

course or medium grade, with which may be associated slight spasms and increased reflexes in the affected limbs. Unilateral forms have been described and occasionally a slight paresis has been demonstrated in the arm and facial muscles. The tremors increase markedly upon excitement and do not always cease completely even in sleep. A careful study of the disease is essential to prognosis. All the cases reported up to the present time have recovered completely within a few weeks, although the mental faculties were slightly impaired in a few instances. The cause of the disease is believed to lie in a toxic infective brain injury. Some authors however, consider it a neurosis. In its etiology, preceding disorders of the digestive and respiratory tracts, or certain infectious diseases, such as measles and varicella, probably play a part.

A diagnosis is usually made very readily. In the unilateral forms it may be difficult, at first, to rule out brain tumor or, more often, encephalitis, and all the more so since the disorder very probably represents a form of the latter disease.

Acute cerebral ataxia is a rare disease. A general ataxia, without paralyses, may appear in the course of any acute infectious disease, such as typhoid, scarlet fever, measles, etc., and particularly after a comatose stage of the attack. Associated with it, occur disturbances of intellection, loss of memory, monotonous syllabic speech sometimes followed by aphasia, and exaggerated skin and tendon reflexes. The sensory mechanisms are unimpaired, with the exception of the stereognostic and, occasionally, the muscle sense. Such manifestations of motor irritability as jerking of the neck may be observed. Most of the cases recover in the course of a few weeks.

In their differential diagnosis, polyneuritis, in particular, must be taken into account. The origin of the disease is probably an encephalitis and in cases showing bulbar and spinal symptoms may be an encephalomyelitis.

VI. EPIDEMIC ENCEPHALITIS

(Lethargic Encephalitis, Epidemic Poliomyelo-Encephalitis, Sleeping Sickness)

In 1917, Economo of Vienna, described a number of cases of encephalitis under the group name of "Lethargic Encephalitis." The outstanding symptoms of the group are paralysis of the ocular muscles and a tendency to sleep or lethargy. Two-thirds of the cases recovered and about one-third died. The pathology of the disease as revealed by autopsy was largely microscopic. The changes consisted of inflammation of the gray matter with, what v. Economo took to be, secondary small cell infiltration about the vessels. The destruction of the ganglion cells as a result of neuronophagia was regularly observed. Furthermore, small hemorrhages in the medulla were usually demonstrable. These changes were usually found widely distributed over a large part of the gray matter but most commonly and most intensely in the region of the midbrain, the ventricular lining, the optic thalamus and the lenticular nucleus. In these regions the white matter was frequently also affected. These findings are confirmed in general

by later observers. It has also been shown that the medulla and spinal cord are affected in some cases.

Clinical Picture.—The clinical picture of the disease is extremely variable. The onset is usually very sudden and most often with high fever. The somnolence, which later in the disease becomes the most characteristic feature of the symptom-complex in the majority of the cases, is not always present at the beginning. In fact, in children, manifestations of increased nervous irritability such as disturbed sleep and the like, are more frequent. In typical cases, these symptoms are succeeded by ocular phenomena such as ptosis, strabismus and double vision. At times, there may be signs of moderate meningeal irritation such as rigidity of the neck, Kernig's sign, etc. After this, the somnolence. In this state the child can be roused, will answer questions, is fairly well oriented, but goes back to sleep as soon as left alone. This may persist for days or even weeks. The temperature may soon fall to normal and recovery set in. In other cases, the somnolence goes on to distinct coma which may terminate fatally after a varying period of time. There is a large group of symptoms, however, which is added to the above picture. In some cases, these additional symptoms are so prominent as to overshadow the symptoms described above, forming types that may be classified as subgroups. So that we may speak of encephalitis of a choreic, athetoid, convulsive, myoclonic, catatonic, amyostatic, or hemiplegic type. Of these the myoclonic, rather common in children, has especially peculiar characteristics. In this type, persisting uninterruptedly for months, individual muscle groups are subject to lightning-like clonic contractions. The neck muscles, separate groups of the musculature of the extremities and, most frequently, the abdominal muscles are affected. The choreic type may resemble chorea minor very closely, but may be distinguished from it by the affection of the ocular muscles or by the appearance of other symptoms not found in chorea. The amyostatic or catatonic types are characterized by a peculiar rigidity of distinct muscle groups without other symptoms of disease of the pyramidal tracts, that is, without increased tendon reflexes, positive Babinski, etc. They further show a distinct reduction of mobility or limitation of motion, with the mask-like face. Occasionally some tremor may be added to this. All of these types may occur separately in a single case, or may be combined in various ways.

Lumbar puncture often shows distinct increase of pressure and the cell count may show a lymphocytosis. The globulin content of the fluid may be increased. The blood findings are not diagnostic. A certain amount of physical and mental lethargy may persist even after the acute symptoms have subsided. Pfaundler, Hofstadt and others have called attention to the persisting disturbance of sleep. This symptom is very common in children and may remain for six months or more after apparent recovery. It may not appear at once, but set in gradually several weeks after the acute stage. The patient does not go to sleep at the usual hour and when sleep does come it is disturbed by constant thrashing about in bed, talking, gritting of teeth, and even tearing the bedclothes. Usually, restful slumber comes on in the early hours of the morning and the child wants to sleep the

entire forenoon. Even so, they lose many hours of sleep with the usual ill effect upon the general well-being.

Etiology.—The question of etiology is still open to much study. Several authors have described micro-organism but none of these works have been confirmed. At present, the bacteriologic findings rest upon a filterable virus and the presence of globoid bodies in the lesions. The relation between encephalitis and influenza is generally regarded as a coincidence. Much work has been done to prove the relation between encephalitis and poliomyelitis. Both of these conditions may attack any part of the central nervous system. Poliomyelitis is more distinctly a disease of childhood and encephalitis more commonly a disease of adult life. The author sees a special significance in the apparent absence of contagion in encephalitis. While the disease occurs in epidemics, there is very little record of its direct transmission.

Diagnosis.—The differentiation from meningitis may present some difficulty in the early stages. In infants, it may be confused with spasmophilia and even with acute disturbances of nutrition.

Treatment.—Up to the present, the treatment has been very meagre. Hexamethylene, subcutaneous injection of foreign protein and other things have been tried without result. The intravenous or intramuscular injection of convalescent serum gives promise of some help. The post-encephalitic disturbance of sleep is also most intractable. Hofstadt recommends hot dry packs, continued for long periods, and completely immobilizing the patient.

VII. BRAIN ABSCESS; PURULENT ENCEPHALITIS

Brain abscess is not wholly unknown among children, or even in infancy. It is a localized purulent dissolution of the brain tissue, always of infectious origin, and of streptococcic, staphylococcic, pneumococcic,meningococcic, or pyocyaneal type. Multiple abscesses are, usually the results of metastasis in the course of bronchiectasis, pulmonary gangrene, etc. The most important form developing in childhood is the otogenous abscess. Traumatic abscess may also occur and is usually situated in the cerebrum. Months and even years may elapse between the causative trauma and the active manifestations of abscess. The recognition of this long latency and the fact that fever may be entirely absent are important points in the diagnosis. Indeed the clinical picture of brain abscess resembles that of brain tumor so closely that only by attention to the etiologic factor of possible injury to the head or of a chronic otitis media will the observer keep upon the right path.

Otogenous brain abscess develops from chronic otitis and particularly from those forms which are complicated with disease of the mastoid cells or with the formation of cholesteatoma. In this type also, a latent stage, often extending over weeks or months, is the common rule. During this period of latency there exist, at most, only an indefinite impairment of the general health. The stage of active development sets in quite rapidly and is characterized by intense headache, vomiting and disturbances of the sensorium. The latter may be of any degree of severity, from mere loss of

memory and inability to concentrate the attention, to a definite stupor or sopor. Delirium, like the headache, of unusual intensity, is not at all uncommon. Sometimes particularly painful areas over the cranium may be outlined by percussion. These may be of value in determining the localization of the abscess. Even at this period there is often an absence of fever. A slow pulse is usually a marked feature.

To these general disturbances, focal symptoms are added in many cases. These are of the greatest importance as guides to the topographic diagnosis and treatment. An otogenous abscess is situated either in the temporal lobe or in the cerebellum. In abscess of the temporal lobe, especially upon the left side, a sensory aphasia or word-deafness has been frequently observed, as well as a crossed paresis of the facial nerve distribution or of the extremities as results of pressure upon neighboring nuclei. An abscess of the cerebellum causes intense pain in the occiput and, eventually, opisthotonos, vomiting, dizziness and an atactic gait and pose. Secondary effects, by way of crossed paralyses of the extremities and the most variable pareses, result from pressure upon the medullary centres or upon nerve roots arising from them.

Meningitis may be excluded by lumbar puncture. In the differentiation from brain tumor, the history of possible etiologic factors, the continuing presence of otitis, the development of focal conditions in the temporal lobe or in the cerebellum, are of especial importance. Neisser's brain puncture may sometimes establish a diagnosis. In doubtful cases operation, which in so large a number of brain abscesses results in complete recovery, should always be advised.

VIII. CEREBRAL TUMOR

The clinical picture of brain tumor in childhood is quite similar to that in the adult. For this reason we shall merely touch upon a few points of special interest from the viewpoint of the pediatrist.

Brain tumors are fairly common among children and especially during the first years of life. Brain tubercles are of most frequent occurrence and their usual location is the cerebellum. Gliomata come next in frequency. Tumors of this type diffusely infiltrate variably large areas of the brain tissue, without being sharply circumscribed from the surrounding normal structures. They are often found in the pons but they may occur in the cerebrum or cerebellum. Sarcomata, myomata, angiomata, and cysticercus cysts are rarer. The last mentioned form may occur as free growths in the ventricles. Syphilitic brain diseases or gummata are of importance. Tumors of the meninges and of the cranial bones are also seen.

Symptoms.—The symptoms of brain tumor fall into two groups: those due to increased intracranial pressure, and those of true focal character. In a large number of tumors, especially those situated in the occipital fossa or the cerebellum, the indications of a congestive hydrocephalus appear. In infancy, indeed, an internal hydrocephalus may dominate the entire picture (see Fig. 119).

Symptoms of intracranial pressure, precede, as a rule, the appearance

of focal symptoms for a variable period. They usually include intense headache, cerebral vomiting, dizziness, and such psychical disturbances as persistent peevishness, loss of interest in play, mental apathy, and constant drowsiness which finally goes on to distinct sopor. Even in these early stages, not infrequently mistaken either for gastro-intestinal disorders or beginning tuberculous meningitis, choked disc may often be demonstrated. Its discovery is of the first importance in the early diagnosis of brain tumor. It is, of course, often absent, and particularly in tuberculous tumors of the pons.

General convulsions may make their appearance at any stage, but they are frequently lacking.

The focal symptoms are of two groups: first the phenomena of lost function due to direct injury or destruction of brain tissue; and, second, the more remote manifestations resulting from pressure of the tumor upon neighboring structures. If the tumor is situated in the so-called silent areas of the brain, the absence of function does not cause any readily recognized disturbance and all the focal symptoms may be lacking. Paralysis is often preceded by evidences of irritation, such as tremor, twitching, or athetosis. Localized clonic contractions are among the important focal symptoms, and may even terminate in general epileptic attacks, or Jacksonian epilepsy.

The topographical diagnosis of brain tumor cannot be undertaken here; the reader is referred to text-books on internal medicine and neurology.

Course.—Recovery is said to occur in those forms of tumor which are due to syphilis and cysticerci and, uncommonly, even in those of tuberculous origin. In cases of brain tubercle, death usually results from tuberculous meningitis or miliary tuberculosis. Sudden death may occur in tumors of the posterior fossa as a direct result of lumbar puncture.

Diagnosis.—Apart from the consideration of the topographical diagnosis, certain essential points must be emphasized. Tubercle is probable if there is a coexisting tuberculosis of lymph nodes or of the bones. The use of the von Pirquet test is important for its exclusion. In children tubercles are always multiple.

The question of syphilis must always be fully considered and the Wassermann reaction should be made. Roentgenography may be employed, especially when tumor at the base or of the hypophysis is suspected. Lumbar puncture always requires extreme care in the suspected presence of brain tumor, but it may be necessary for the exclusion of meningitis. Given the former, the pressure of the cerebrospinal fluid is increased, but it is in other respects normal. Brain tumor is often only to be differentiated from some forms of encephalitis and from chronic internal hydrocephalus by the course of the disease. Acute onset and a history of pauses or retrogressions are evidence against brain tumor. The diagnosis of brain abscess must be considered whenever the symptoms point to the cerebellum or to the temporal lobe and particularly when an otitis media has preceded them within a certain period of time.

Treatment.—In most cases treatment must be symptomatic. Antipyrin or morphin to control pain; chloral hydrate for protracted convulsions;

lumbar puncture, if necessary, to relieve a congestive hydrocephalus and its resulting symptoms. The latter procedure is useful only when the ventricles are not occluded. Operative removal of the tumor is a possibility only in exceptional cases. Generally speaking, only the cerebrum, the cerebellar portion of the pons, and the hypophysis can be laid open for operation. Surgical interference is always dangerous. Simple trephining and cerebral puncture to relieve pressure is a palliative measure to be considered.

Large doses of potassium iodide should always be tried even though there be no suspicion of syphilis. It has often had a favorable influence upon other forms of brain tumor. Of course, in every case in which lues is suspected strenuous antisyphilitic treatment must be instituted at once.

IX. CEREBRAL PARALYSIS OF CHILDREN: INFANTILE CEREBRAL PALSY

Spastic Infantile Hemiplegia and Diplegia

The term cerebral paralysis of children covers a clinical group. It is understood to represent the results of various non-progressive injuries which affect the brain in earliest childhood or before birth. In the study of the given clinical picture the localization is more important than the nature of the injury. It is usually impossible, even at autopsy, to get definite knowledge of the nature of the original disease, of usual occurrence many years before. But even while it is impossible to establish definite criteria of general application, individual clinical types may be very easily selected from the chaos of cases. Indeed, some special consideration of hemiplegic and diplegic forms is quite opportune, because etiologically they have a distinct viewpoint from which their prognosis may be determined. Nevertheless, it must not be forgotten that a continuing series of transitional cases relates the individual types.

Etiology.—A number of predisposing factors enter into the origin of the cerebral paralyses of childhood. These are often operative even when the direct cause of the disease is known, the development of which depends upon a certain lack of resistance of the brain tissue to injury. Thus the disease occurs in children who are members of a family in which a succession of nervous and mental disorders has been observed. It is often possible to show that the parents were tuberculous or luetic or that the father was an alcoholic. Either the first-born or the youngest of a family is affected with relative frequency.

The direct cause is either of a traumatic or infective nature. Birth traumata are responsible for many cases, particularly of the diplegic type. Severe and protracted, or violent and rapid labor, inducing a serious degree of asphyxia in the child, is believed to play some causative part, but probably a less frequent one than is commonly supposed. Meningeal hemorrhages resulting, as a rule, from a tearing of the veins at their entry into

the sinuses, are doubtless an actual, but not as exclusive cause of injury to the brain.

Premature birth is another important etiologic factor. Many children who suffer with spastic paraplegia were prematurely born. Nevertheless, it is to be remembered that prematurity and difficult labor are not infrequently recorded in the history of cases in which the origin of the disease doubtless dates back a long time before birth. Such instances afford an opportunity for error in determining the actual causes of the condition. It is quite possible that some of these prenatal cases may have arisen in part from traumata received during pregnancy and perhaps even from psychic traumata. A certain percentage of them, whether of prenatal or postnatal development, may be traced to syphilitic or so-called parasyphilitic disease.

Brain embolism or encephalitis may occur in the course of infectious disease. This is particularly true of pertussis, diphtheria, scarlet fever, measles, influenza, and occasionally of almost any infectious disease. Certain of these cases present a definite brain disease in themselves—the acute polioencephalitis of Strümpell, a form already discussed under the head of acute encephalitis.

In general the rule of the so-called Little's etiology may be accepted, *viz.*, that difficult labor, premature birth, and asphyxia of the new-born cause most of the diplegic cerebral paralyses, while the majority of the hemiplegic paralyses appear only after birth and are of infective origin.

Pathologic Anatomy.—In the greater number of cases the initial lesion is of vascular type, in the way of hemorrhage, embolism or thrombosis. In a minor number it is due to some inflammatory process. Pure degenerative changes in the cerebral cortex are probably very rare. It has been said already that cases are seen, as a rule, only in their later stages when any conclusion as to the nature of the original lesion is no longer possible. The brain by that time shows more or less localized destruction, foci of softening, scars, cysts, and particularly structural defects of the cortex, by way of excavated funnel-shaped depressions which may extend into the ventricles, the so-called porencephaly (Heschl). Sometimes the finely graven convolutions of the cortex described as microgyria are seen. Instead of these localized foci, a more diffuse injury is often observed in the form of a sclerosis, or hardening, and contraction of large areas or entire divisions of the brain (atrophia cerebri). The tuberous scleroses present a peculiar type, in which single, hard, nodular areas are scattered throughout the brain. Cases in which no gross lesions of the central nervous system are discoverable macroscopically, but in which microscopic examination reveals a diffuse proliferation of the neuroglia and a destruction of nerve cells, are not uncommon. The meninges often show the results of old inflammatory processes, in the way of cloudiness, adhesions and thickening, the results of which, like those of many cases of recovered meningitis or hydrocephalus, cannot be distinguished clinically from true cerebral paralyses.

Frequently pathologic conditions are found in the pyramidal tracts.

Spastic Infantile Hemiplegia; Unilateral Cerebral Paralysis of Children

Hemiplegias are distinguished in a general way from diplegias by the facts that they are usually of postnatal origin, that the arm is more commonly and more severely affected than the leg, that they result more frequently in disturbances of growth, and oftener lead on to epilepsy.

Typical cases take something of an acute course, after the manner described in encephalitis. The hemiplegia often coincidently affects the face. The paralysis is at first flaccid, but soon takes on a spastic type. The actual paralysis of motion is often less prominent than are the muscular spasms. Aphasia, commonly present in the beginning, soon disappears, and in time the paralysis itself partially clears up, especially in the leg, while in the arm contractures are apt to follow. As the paralysis improves, choreic and athetotic movements often develop on the paralyzed side of the body. The growth of the affected parts is retarded. The mental faculties are at times slightly, or may be severely affected. After weeks, months, or even years, typical epilepsy, often of progressively increasing gravity, may appear.

Special Symptoms.—The distribution of the paralysis may be very regular. The palsy of the facial nerve, in its later stages, may be discernible only by careful observation, as when the patient begins to smile or to cry. Of other cranial nerves, the hypoglossal is not infrequently affected, while strabismus or involvement of the motor oculi is very uncommon. Pupillary fixation may be significant of a luetic cause.

As to the extremities, the arm and especially the hand is, as a rule, more severely affected than the leg. There are many cases in which the leg recovers so largely as to give the casual observer the impression that only a monoplegic paralysis of the arm has occurred. Only by careful examination, by noting the increase of the patellar reflex, and by the discovery of slight differences in growth, as between the well and the affected sides, is it possible to arrive at a correct conclusion. The exaggeration of the deep reflexes is the most constant and persistent symptom. Of course, the patellar reflex may often be exaggerated upon the normal side, but the unilateral difference will be distinct nevertheless. Babinski's sign, the stretching of the great toe when the sole of the foot is scratched, is frequently found on the affected side. Coincidently with the increase of the reflexes, the pathognomonic rigidity characteristic of the disease develops. This is usually most masked in individual groups of muscles and particularly in the flexors and pronators of the arms and in the flexors of the legs. Resulting contractures soon appear. The leg is slightly flexed and rotated inward; the foot is drawn downward and inward; the arm is flexed to a right angle; the forearm is drawn up and pronated, and the hand may be fixed in varied positions. In cases of relatively long standing the contracture cannot be overcome even when considerable force is employed. To these continuing contractions, sometimes scarcely noticeable, are added spasms occurring upon voluntary motion. These are usually very distinct and serve as a serious embarrassment to the use of the limbs. The patient

constantly struggles against intrinsic obstacles and his movements become exaggerated and slow. From the functional point of view these spasms are often much more annoying than the paralysis. Indeed, true paralysis may be found upon careful examination to be entirely absent. The intensity of the one is quite independent of that of the other. The paralysis may be most marked in the hand; the rigidity may be most definite in the leg or in the musculature of the shoulder. Ataxia and intention tremor are not infrequently coincident with the paresis.

On account of the spastic fixation of the lower extremity the gait is often very characteristic. The foot is not carried directly forward, but is advanced by a circular swing operating from the hip; it drags a little and the toes only are planted on the ground. In milder cases, the awkwardness of action may appear only when the child attempts to stand on one foot or tried to hop or to stand on his toes.

Fig. 127.—Right sided spastic infantile hemiplegia. Spastic paresis of right arm and leg with atrophy of musculature. Delayed growth of right side of body. Fixed talipes equinus. Wing-like elevation of right arm in walking.

In walking, a peculiar movement, quite typical of cerebral paralysis in children, is observed. The faster the child walks the higher is the affected arm raised, wing-fashion, and waved to and fro in the air (see Fig. 127). These associated movements may be imitative of those on the normal side in every detail, as is very strikingly shown if the child is allowed to roll a bread pill between its fingers. The involuntary movements of the affected limbs are very noticeable and very annoying. They appear in the later stages of the disease in about one-third of the cases the onset of which occurs in advanced childhood. The quick, jerking movements, usually affecting an entire extremity, or the shoulder girdle, or the facial muscles, constitute chorea; while the slower, rhythmic, flexing, or extending, stretching motions of the fingers and, at times, of the toes, are termed athetosis. Frequently, grimaces are of similar quality.

Chorea and athetosis are in a degree antitheses of the paralyses. The more completely the paralysis and the spasms disappear, the more varied do the involuntary movements become. In extreme cases, they make the limbs almost impossible of control. As an intermediate phase between the spasmic and athetosic conditions one often sees an athetoid spreading of the fingers, with an excessive degree of passive mobility which tends to their over-extension.

The paretic limbs commonly suffer from trophic changes. These usually

take the form of a delayed growth or hypotrophy of the affected parts, in the matter both of length and mass, which may involve a high grade of atrophy in their musculature.

Epilepsy and idiocy, as complications of the disease, are of grave significance in considering the ultimate fate of the patient. More than half of these sufferers become epileptics. The convulsions which accompany the initial stage of the disease are usually followed by an interval of weeks, months, or even years, during which the patient remains free from attack. Subsequently epileptic seizures set in, increase in frequency and may continue throughout life. Even the mildest cases, in which recovery from the paralysis is almost complete, are not secure from this serious complication.

Disturbances of the intelligence, like epilepsy, are the sad sequelæ of many cases of infantile hemiplegia. In very few cases do the mental faculties remain wholly unimpaired and even in these exceptions a distinct change in character is often observed. The patient grows irritable and shows a tendency to fits of temper and acts of violence. All degrees of mental degeneration, from slight imbecility to most complete idiocy, are recorded.

Choreic Paresis.—This term is used, according to Freud and Rie, to designate those clinical forms in which the hemichorea has not been preceded by the stage of spastic paralysis. As a rule, it is of insidious development in older children who do not suffer from epilepsy, from marked disorders of intelligence, or errors of growth.

Spastic Infantile Diplegia; Cerebral Diplegia of Children

The most serious forms of cerebral diplegia are grouped, in the literature of these cases, under the name of Little's disease and, according to Freud, are termed general spastic disease whenever the entire body is involved, or paraplegic spastic disease when the lower limbs alone are affected. These forms are distinguished by the fact that spasticity of the limbs is the dominant feature of the disease-picture and that the legs are the more seriously paralyzed or are alone affected. They are further characterized by a distinctly regressive tendency, and by the evidence of their prenatal origin or of an etiology in birth traumata in the great majority of cases. In instances of general spasticity it is very often possible to obtain a history of asphyxia neonatorum and of difficult labor; while premature birth is more frequently relational to paraplegic spasticity (Feer, Freud).

Severe cases of general spasticity appear even in early infancy. The child seems to be as stiff and rigid as a stick of wood and is difficult to dress or to change. It is impossible to make him sit up. Such an one, however, marks an extreme grade of the disease, usually provoked by intra-uterine injury. Milder forms are discovered by the parent or even by the physician only when the child is old enough to begin to learn to walk, when it is found that the spasticity of the legs makes this impossible or extremely difficult. Many a patient so afflicted never learns to walk or does so only in later years. Placed upon the feet they present a very characteristic picture.

The thighs are rotated inward and the knees are pressed firmly together in consequence of spasm of the adductors. The tips of the toes only touch the floor. If the attempt to walk is made, the knees are pushed past each other with great exertion, the legs tend to cross (Fig. 130), and the child advances only by rotating upon the axis of its body. When the spasms are less severe, and they sometimes improve slightly with increasing years, the patient may be able to walk, but only by constantly overcoming the nervous tension. The slow and strained gait gives one the impression of a person wading in deep mud. The toe step remains throughout. Upon lying down or rising up, the legs are often moved completely in unison, as though they are bound together. In sitting they are held horizontally, on account of the extensor spasms.

FIG. 128.—Cerebral diplegia (Little's disease). Premature birth at seven months. Asphyxia. Crossing of legs on attempting to walk, strabismus, dysarthria, easily frightened.

The spasticity is usually less marked in the arms and often does not involve the trunk at all. Chorea, involuntary movements, and typical athetoses are much less common in the diplegias than in the hemiplegias, but an athetoid spreading of the fingers and toes is often met with. Tremor and ataxia are not at all rare. The deep reflexes, especially in the lower extremities, are always exaggerated, but the degree of spasticity may hide the patellar reflexes entirely. At rest, the spasms are often less marked, but reappear upon voluntary action or brisk passive motion. As a rule, they are especially marked when the attempt is made to stand the child upon its feet.

In the paraplegic forms, the nerves supplying the ocular muscles are frequently involved, producing a degree of strabismus. Optic atrophy, inequality of the pupils and nystagmus, are not of exceptional occurrence. If the facial nerves are affected the spasticity gives to the face a mask-like expression, which is in sharp contrast to the forced mimicry it displays upon the exercise of emotion, as when the child cries or is frightened. Dysarthria and bradylalia are often noted and, combined with the difficult operation of the muscles of expression, give one the suggestion, at times, of a high degree of imbecility, even though the intelligence may not be very seriously affected. Spasm of the pharyngeal muscles may occasion difficulty in swallowing. Retardation of growth and actual hypoplasia of the limbs

take a less important place in this form of paralysis than in the hemiplegic type. The musculature may show some atrophy, but occasionally is hypertrophic. The constant finding of the high position of the patella (Schulthess), which becomes a most prominent symptom when the knee is sharply flexed, is significant.

Attacks of spasms or convulsions are quite frequent shortly after birth in all forms of diplegia. Later convulsions, of the epileptic type, are decidedly less frequent than in hemiplegic paralysis. Indeed, in paraplegic spasticity they are of quite uncommon occurrence.

In the cerebral diplegias, failures of intelligence and even idiocy of serious degree, are common and naturally so, since a certain percentage of cases result from grave malformations of the brain. A small head, the so-called pseudo-microcephaly, may be noticeable even at birth. Nevertheless, there are cases, especially of the paraplegic type, in which the mental faculties are partially or well developed.

It has been noted already that Little's disease shows a tendency to improvement. After a number of years only the stiffness of the legs may remain of the general spastic disorder. Paraplegic spasticity may disappear completely in the course of time.

Fig 129.—Four-year-old boy. Microcephaly. General muscular rigidity, atheosis, idiocy.

Peculiar Types of the Disease

Those forms of the disease in which the manifestations of spasticity are distinctly confined to the lower extremities are called paraplegic paralyses.

Pronounced cases, associated with a high degree of idiocy and with well developed epilepsy, and in which the arms are more or less seriously affected, even to the point of contractures, indicate a doubling of the hemiplegic conditions and are designated as bilateral spastic hemiplegia.

Pseudobulbar paralysis (Oppenheim, Peritz), is a term applied to cases of the latter type to which is added a bilateral involvement of the cranial nerves. The title suggests a symptom-complex which results from bilateral disturbances in the brain areas controlling the muscles of expression, deglutition and speech, and resembles very closely the disease-pictures

produced by injuries to the gray nuclei of the cranial nerves in the medulla. Pseudobulbar paralysis may also be seen in the congenital form of general spastic disease. Facial expression is wholly wanting in these bilateral spastic types: the face appears vacant as though carved in stone. The control of facial movement may be so lacking that in place of a smile the face is distorted into the risus sardonicus. In the more markedly paretic forms the patient cannot pucker the lips, distend the cheeks, or protrude the tongue. The control of suckling and swallowing is usually less disturbed and the movements of the facial muscles in laughing or crying may be well regulated. At times, the child will use the fingers to push the food into the grasp of the more automatic muscles of the pharynx by which the normal act of deglutition is performed involuntarily.

General Chorea and Bilateral Athetosis.—These terms relate to those severe forms of disease in which local voluntary choreic movements, or simultaneous, generalized, associated movements dominate the clinical picture. Constantly recurring, jerky motions of the entire body and often repeated grimacing are observed. The functions of speech and of voluntary movement are seriously impaired, particularly in the more severe pareses. Frequently an abnormal flaccidity of the muscles replaces the hypertonia.

Cerebellar Forms.—There are forms of the disease in which impairment of cerebellar coördination is a prominent feature. The disability is seen both in standing and sitting and in an abnormal flaccidity of the muscles in general.

An atonic-astatic type of infantile cerebral paralysis resembles, in a degree, the cerebellar forms. This disease-picture, so well outlined by O. Foerster, is featured by a generalized atony, by the absence of all involuntary resistance to passive motion in the muscles, which results in a marked overextension of the joints, and by a loss of control of static function. Each individual muscle may be made to contract by stimulation of its nerve supply, but the patient is unable to stand or to sit. He simply collapses. The head cannot be held erect but falls to one side. As in all the cerebral diplegias of children, the disease shows a distinct tendency to improvement continuing over a period of many years. It would seem that this type, which in its primary stages occasionally appears in the common forms of general spasticity, is dependent upon lesions in the frontal lobes. According to Clark, who has proposed the name of cerebro-cerebellar diplegia, it is a condition in which the functions of the cerebral and cerebellar hemispheres are coincidently affected.

Infantile Spastic Spinal Paralysis or Spasmatic Tabes.—This term was formerly employed to designate the pure spastic pareses of the legs or of all the extremities which were believed to be localized in the cord. Their cerebral origin is now clear in those forms which exhibit strabismus, mental disturbances, speech disorders, and epilepsy. To-day there is a general tendency to consider them all as of cerebral localization. Since, however, these diseases frequently affect the new-born, it is generally believed that they may lead to a delay in the development of the cortico-spinal bundles and, particularly, of the pyramidal tracts. A spasticity

without paralysis, the more severe involvement of the legs, and a gradual spontaneous improvement during life may find a very satisfactory explanation in this manner.

Familial forms of cerebral diplegia and of amaurotic idiocy belong to the hereditary degenerative diseases and are discussed under that head.

Diagnosis.—The several clinical forms are not always sharply distinguished and mixed forms are often seen. The pathologic conditions in the brain cannot be determined during life. According to Vogt, the coincident development of sebaceous adenomata of the skin is characteristic of a tuberous sclerosis of the brain. High degrees of microcephaly are indicative of a prenatal origin.

Fig 130.—Cerebral diplegia (Little's disease), so-called tabes spasmodique. Tetra spasm, indistinct in the arms. Intelligence good. Premature birth at seven months.

During infancy it is often impossible to recognize the milder cases, since hypertonia is physiologic and is emphasized in many of the chronic disturbances of nutrition. A marked exaggeration of the reflexes and especially the evidence of idiocy may establish diagnosis, while abnormally early closure of the fontanelle may afford the first suspicion.

In fully developed cases, the differentiation of the disease from forms of paralyses of peripheral origin (poliomyelitis, birth palsies, etc.), which may present a similar picture, particularly when they have led to well-marked contractures, is of the greatest importance. The spastic character of the paralysis, the increase of the reflexes, the less pronounced atrophy, the mental defects, the development of epilepsy, or of choreic and athetoid movements, all testify to cerebral paralysis. When doubt prevails, an electrical examination gives definite distinctions. In the cerebral paralyses, the affected muscles give normal reactions to the galvanic and faradic currents. By this means, also, the atonic-astatic forms may probably be distinguished from congenital muscular atony which affects the muscles of the trunk and neck less severely and shows a reduced irritability to electrical tests.

In every case the question of brain syphilis must be given careful consideration. Doubtful subjects demand the Wassermann reaction. Inequality of the pupils, and especially their failure to react to light, must be considered as strong indications of syphilis.

Brain tumor, in many instances, may be distinguished from the disease

under discussion only by its progressive course or by the presence of choked disc. This is measurably true of hydrocephalus in older children which may simulate spastic diplegia.

If nystagmus, intention tremor, and bradylalia occur under paraspastic conditions the picture may resemble that of multiple sclerosis very closely. But, on the one hand, it is quite certain that the latter disease hardly ever occurs in childhood and, on the other hand, that atypical cases of cerebral diplegia frequently belong to the familial forms which are usually characterized by a progressive course. The disease is differentiated from Friedreich's ataxia by the symptoms of spasticity and by the exaggerated reflexes.

Amaurotic idiocy and diffuse sclerosis can be mistaken for cerebral diplegia only if the progressive nature of the disease is ignored.

Prognosis.—Many hemiplegias and most cases of general and paraplegic tetany have a tendency to spontaneous recovery. Children affected learn to walk at the age of eight or ten years. The prognosis is governed chiefly by the state of the intellect and by the absence or presence of epilepsy. The appearance of the latter can never be foretold in hemiplegia even of mild form.

Treatment.—The treatment of meningeal hemorrhages of the new-born and of acute encephalitis has been already discussed. When syphilis is an etiologic factor an energetic antisyphilitic treatment is indicated. For the rest the aim of treatment must be to develop the best degree of motor function.

For this purpose electricity may be used. The paretic muscles are stimulated with the induced current and the anode of the galvanic current is passed over the spastically contracted ones. Protracted warm baths also aid in relaxing the hypertonicity and may be given frequently, for weeks at a time, especially in the diplegias.

Properly conducted and long continued active exercises are of the greatest importance. Their practice requires a certain measure of intelligence and good-will on the part of the patient. Advantage may be taken of the tendency of the affected member to move in unison with the well limb, so that it is well to permit the latter to participate in the training (H. Curshmann). Exercise, supported by massage and passive motions, is possible and useful only after the contractures have been relieved by tenotomy and readjustment. Thus the treatment of these cases has been rightly relegated more and more to the domain of orthopedics which has an opportunity for great results.

In hemiplegia the deformities of the foot are readily corrected by the plastic lengthening of the tendo Achillis. In the arm, a lessening of the hypertonus and a strengthening of the paretic muscle groups can be achieved by proper tendon transplantation, transferring parts of the hypertonic muscles into functionally inactive muscles. The observation that chorea and athetosis do not occur in a member upon which tendon operations have been performed is especially interesting. This is an all the more

important fact because these function-disturbing involuntary movements are but little influenced by other methods of treatment.

In diplegia very beautiful results have been obtained by putting the legs in plaster and keeping them in the Gratsch position for a considerable period after the bilateral tenotomy of the tensor fascia lata in the popliteal space, of the adductors and the flexors, and of the tendo Achillis (Fig. 131). Careful after-treatment with massage and with active and passive exercise enables the patient to walk within a few months. The crooked back is usually a very difficult problem in correction. As already noted, treatment is effectual only if the intelligence is fairly well preserved.

Very recently, good results have been obtained in several cases of Little's disease by Foerster's operation. The principle of this operation lies in relieving excessive irritability of sensory nerves leading to the spastic muscle groups, which serves to excite the spasms. To accomplish this

FIG. 131.—Cerebral diplegia (Little's disease). Mask-like facies. Legs held in the Gratsch position by plaster dressing after preceding tenotomy of the tendon Achillis, the flexors of the leg, the adductors and of the the tensor fascia lata.

several of the posterior nerve roots are laid bare in the spinal canal and are cut. This operation may prove to have a great future. The selection of the roots to be cut is, of course, a very important matter. It will suffice to call attention to Foerster's original reports.

Stöffels' operation follows a different therapeutic principle. It is directed to the weakening of the spastic muscle groups by the section of a part of the motor nerve supply in the peripheral nerve trunks. Thus in hemiplegic paralyses of the arm the functional relations may be materially improved by the weakening of the pronators and flexors, by a resection of the median nerve in the bicipital groove. Similarly, the spastic toe step is corrected by resection of a part of the tibial nerve.

Both the Foerster and the Stöffel operations demand careful orthopedic after-treatment, with massage and with active and passive exercise, in order to insure permanent results.

The treatment of epilepsy in this disease is identical with that of its true forms. In a few cases trephining and the excision of the primary

disease focus in the cortex, or the removal of offending cysts or scar-tissue has proved useful. Disturbances of speech and intelligence require pedagogic treatment. Clearly idiotic patients should be placed in suitable institutions.

X. SCLEROSIS OF THE CENTRAL NERVOUS SYSTEM

Partial secondary scleroses of entire divisions of the brain and tuberous sclerosis have been discussed in an earlier chapter (page 500).

Diffuse sclerosis of the brain is a rare disease. It may be a sequel of traumata and usually begins insidiously during the first year of life. In the course of months, or even years, it eventually terminates fatally. It is occasionally interrupted by epileptiform or apoplectic attacks which are associated with aggravations of the primary disease.

Its symptoms consist in a progressive spastic paralysis of the entire musculature, together with a loss of speech and intelligence going on to complete unconsciousness. The optic nerves become atrophic. The hardening of the brain and spinal cord, and especially of the white matter, is dependent chiefly upon a proliferation of the neuroglia and is to be looked upon as the result of an interstitial inflammation.

Multiple focal sclerosis is extremely uncommon in childhood, probably occurring only as the terminal stage of multiple encephalomyelitis. It is usually sequent to acute febrile diseases. It is hardly necessary to discuss the malady further since its course and symptomatology are identical with those witnessed in the adult. Briefly, the symptoms include spastic paresis of the legs, intention tremor, optic atrophy, nystagmus, sensory disturbances, bradylalia, paralysis of the sphincters, etc. The majority of the children who present a corresponding symptom-complex are suffering with some atypical form of cerebral diplegia or with some type of hereditary degenerative disease, as familial diplegia, hereditary ataxia, etc. Multiple sclerosis may be simulated by progressive paralysis, brain syphilis, and hysteria.

XI. ACUTE POLIOMYELITIS; SPINAL PARALYSIS OF CHILDREN

(Heine-Medin's Disease; Acute Epidemic Infantile Paralysis)

Acute infantile paralysis is an infectious disease of the central nervous system, occurring in widespread epidemics or in sporadic form, and most common in early childhood. The term acute anterior poliomyelitis applies to the type of greatest clinical importance. The name signifies the localization of the disease processes in the anterior cornua of the spinal cord. It causes flaccid paralyses, affecting individual muscle groups, which may persist throughout life.

Etiology and Epidemiology.—The greatest epidemics of recent years in the Scandinavian countries, in North America, Germany and Austria have greatly enlarged our knowledge of the disease.

Cases of seemingly sporadic occurrence must be looked upon as the residuum of lingering epidemics which doubtless present more such single

cases than we ordinarily suspect. This is the more true because, up to the present time, abortive cases in which no paralysis develops are diagnosed only during epidemics and even then with relative infrequence.

Children in the first three years of life are by far the most definitely prediposed to the disease, although adults are also attacked. In the epidemics recorded, the largest number of cases have occurred during the summer months, particularly in July and August, but continuing to October.

Landsteiner and Popper have succeeded in inoculating monkeys with the disease. From the recent researches of Flexner, Roemer and others, in experimental poliomyelitis in monkeys, we have learned that the incubation period is from eight to nine days, that the virus passes through the Berkfeld filter, and has the consistency of glycerin. Further, it reaches the central nervous system by way of the lymph passages and, outside of the nervous system, it may also be found in the nasal and pharyngeal mucus and in the saliva. Apparently it is not affected by the gastric juice and has been found in the feces. This virus is destroyed quickly by high temperatures, but resists cold and drying. One attack confers immunity. Immune bodies which, being mixed with the virulent infectious material, destroy its virulence, are found in the blood alike of human being and monkey, convalescent from the disease.

The method of spread is probably by contagion from person to person, in which event, as in epidemic meningitis, healthy or but mildly affected carriers play the most active part (Wickman). In convalescents, the virus is evidently present in a virulent form for many weeks in the secretions of the mucous membranes and in the membranes themselves (Kling, Peterson and Wernstedt). It has been found also in the dust of the sick-room. It has been proved beyond contradiction that stinging flies (Stomoxys Calcitrans), may transmit the disease. No other insects and neither food material nor drinking water enter into its etiologic consideration.

The causative organism has recently been obtained in pure culture by Flexner and Noguchi. It is an extremely small globular cell which in view of its cultural characteristics must be classed, in all probability, as a bacterium.

The port of entry of the organism seems to be both in the digestive and the respiratory tracts, but particularly in the pharyngeal ring. Numerous cases have been found among the children of families in which the severity of the disease has varied widely, abortive forms being seen in some of the members.

Pathologic Anatomy.—In children dying in the acute stages of the disease, focal hemorrhagic changes in the anterior horns, and most commonly in the cervical and lumbar enlargements, may be seen macroscopically. Microscopic examination shows that the inflammatory process, which apparently spreads in the vascular sheaths (Wickman), may not only extend to the posterior horns, but even to the white matter, and is always more diffuse than would be imagined from the clinical findings. The meninges always show inflammatory changes under the microscope (Warbitz and Schell). As a consequence of the inflammatory process in the

region of the gray matter, extensive injuries of the ganglion cells and often neuronophagia are found.

In older cases the disease foci are sclerotic and atrophic. The ganglion cells have disappeared or degenerated; the vessel walls are thickened, the glia proliferated, and the entire half of the cord may appear contracted even to the naked eye. The anterior horn is narrowed and the boundary between the posterior horn and the white matter is blurred. The affected muscles are more or less degenerated and are salmon-colored, bright pink, gray, or even yellowish in tint and at times broadly striated.

Symptoms and Course.—The incubation period varies from five to ten days, and occasionally may be shorter. It is followed by a febrile initial stage, which gradually passes into the period of beginning paralysis. The latter, in turn, goes on to permanent paralyses and contractures.

The initial stage is characterized by pyrexia, frequently exceeding 39°-40° C. (102°-104° F.). There is marked disturbance of the general health. The pulse is usually very rapid; the sensorium is affected slightly if at all; there is a great desire for sleep which, however, is restless and disturbed by dreams and delirium. Locally, it may be possible in some cases to find an angina present; in others there may be bronchitis, and, again, such digestive disturbances as stomatitis, vomiting and diarrhœa, or a severe degree of obstipation. There is but little headache and typical general epileptiform convulsions are rare. Herpes may be present, but is extremely uncommon. The temperature usually falls to normal after the first few days, but occasionally runs a typical course for a week or two. This early stage, which presents great difficulties of diagnosis, is further marked by several significant symptoms. The most important of these, and the one which first invites the parents' attention, is an extreme sensitiveness of the skin to touch, and the expression of pain on passive motion. The child screams as soon as an attempt is made to lift him, or, indeed, when the mother or the physician even approaches the bed. He refuses to be moved from the bed and begs to be left alone. If he is raised he stiffens the spine, which does not otherwise indicate hypertonia. He makes frequent complaints of spontaneous pains in the back and in the muscles; and an extreme sensitiveness to pressure over the nerve roots may be shown for a long time after the early stage has passed. A second important symptom is the great tendency to perspiration. To these must be added a third finding, established by E. Müller, a distinct leucopenia (3,000-5,000 leucocytes). Not one of these symptoms is constant, and even leucocytoses have been reported several times. At this period, lumbar puncture usually reveals a cerebrospinal fluid under increased pressure and commonly clear, although it is, now and then, opalescent. Its protein content is increased and a fibrin clot may develop upon standing. Sediment obtained from it by centrifuging shows a lymphocytosis. Cultures from it are negative, and after the recrudescence of the initial stage these pathologic findings disappear.

This stage rarely lasts longer than two or three days. Doubtless there are cases in which it passes in several hours and is entirely unnoticed, so that the patient who went to bed seemingly well wakes up paralyzed in the

morning. The early paralyses are usually distributed over the greater part of the body. Paralysis may be preceded by spasms and jerking of the affected parts. It is of a flaccid type, involving, most frequently, the legs and trunk and, less often, the arms and the areas supplied by the cranial nerves.

It reaches its maximum spread in from a few hours to a few days and after this period of initial development it shows only retrogressions. Successive attacks, affecting new parts, are very exceptional.

The paralysis is not always easily recognized at first; but upon careful examination a hypotonicity, or at least an absence of patellar reflexes is always discovered. Very often the muscles of the trunk and abdomen are first affected and then the seemingly meteoric distension of the abdomen, contrasting with the flaccidity of the abdominal wall, attracts attention. Then, too, the patient cannot sit up or maintain a sitting posture. Disturbances of control of the urinary and anal sphincters are common in the initial stage, but are always transient. Retention of urine, requiring catheterization, occurs only when the lumbar segments of the cord are involved, and it is then coincident with paraplegia of the legs.

Fig. 132.—Acute anterior poliomyelitis. Paralytic club-foot on right due to paralysis of the extensors of the toes and of the muscles of the calves. The tibialis anticus and the flexors of the toes are normal. Atrophy of the thigh and leg.

The muscular paralysis is often relieved, to a considerable extent in the course of succeeding days or weeks. Entire members, completely paralyzed, may fully recover the power of motion. A considerable number of cases, indeed, are restored without any apparent defects, but these are, of course, the exceptions. Some part of the muscle groups originally paralyzed usually suffers permanent injury.

The paralyses are flaccid; they are peripheral and produce atrophy. If the affected limbs are raised they fall back inanimately upon the bed. The deep reflexes are lost. The electrical examination reveals reactions of degeneration which are soon followed by atrophy of the muscles. While the nerves and muscles are often excessively irritable at first, both to mechanical and electrical stimulation, they soon fail to respond to excitation with the induced current and only respond to the direct current by slow wave-like contractions in which the anodal phase is the greater.

Muscles in which the reactions of degeneration are fully established

recover incompletely or not at all. In the course of epidemics, however, exceptions to this rule have been observed.

The disappearance of the deep reflexes and especially of the patellar reflex, again, is the rule; but in the event of paralyses of the arms alone, exaggerated patellar reflexes have been observed several times. This is readily explained in view of the coincident affection of the white matter of the cord which may lead to an injury of the pyramidal tracts. The tendo Achillis reflex, or ankle clonus, often proves to be exaggerated. The skin reflexes may be intact or may be wanting, when the underlying muscles are affected. The Babinski phenomenon may be positive.

Fig. 133.—Paralysis of the abdominal muscles and paralyses affecting both legs following epidemic infantile paralysis.

The atrophy of the muscles often causes marked loss of size and form in the limbs. The natural modelling is lost and the entire member may seem to consist merely of skin and bone. In very young children the loss of muscle mass is sometimes masked by the development of fatty tissue.

The various types of paralysis may present many combinations but, in actual experience, they take certain prevailing forms. Most frequently only one leg remains paralyzed; more rarely a single arm or both legs; occasionally paraplegia of the arms, crossed hemiplegias, and even hemiplegic varieties are seen. It is characteristic of acute poliomyelitis that the paralysis never affects all the muscles of the entire limb equally. Individual muscle groups are involved, while others remain free. Very often muscles which are associated functionally, even when supplied by entirely distinct nerves, are simultaneously affected. In the leg the peronei are most commonly paralyzed. The participation of the quadriceps and the tibialis anticus in the paralysis, while the sartorius remains free, is a frequent picture. In the arm the deltoid is most frequently affected, while the smaller muscles of the hand generally escape. The muscles of the neck and trunk very often suffer at the outset, but they usually recover. If this is not the case, serious scolioses and lordoses ensue. Paralyses of the abdominal muscles may leave localized atrophied areas in the abdominal wall which result in hernia upon deep respiration or muscular strain (Fig. 133).

Paralyses of the bulbar or pontine forms, involving the cranial nerves,

very rarely observed among sporadic cases, occur quite frequently in the course of epidemics. The facial nerve is the most frequently affected, while the abducens, the hypoglossal, and the oculomotor are seldom attacked. If the nucleus of the vagus is invaded, dyspnœa results and in most cases causes death.

These paralyses show the most rapid improvement during the first week, although gradual improvement may take place during the ensuing half-year and even later. The possibilities of improvement should be limited perhaps to one year from the invasion of the disease. After the disappearance of the initial symptoms, the general condition is usually excellent. A certain psychical irritability and peevishness, and a tendency

FIG. 134.—Acute poliomyelitis. Paralysis of the musculature of the trunk and legs.

to pain in the limbs may be noticeable for a time. The absence of sensory disturbances is especially characteristic of the later stages of the disease and has a diagnostic value.

The stage of permanent paralysis is further distinguished by secondary results in the way of contractures and deformities which cripple the unfortunate child for life. If all or nearly all the muscles of an extremity are paralyzed, contractures are not formed, but the limb hangs limply from the body like that of a doll.

If, however, only individual muscle groups are affected, a more usual result, the action of antagonistic muscles causes, within a few weeks, the development of contractures, which by the shortening of tendons and the fibrous degeneration of the muscular tissue itself, acquire a fixed abnormal position. The pressure even of the bed-clothes and of the body, when resting upon the limb, increase the tendency to the development of such deform-

ities. These contractures are harmful, in themselves, in that they cause an overstrain and thus do further injury to partially paralyzed, weakened, but not always entirely useless muscles. Serious functional disorder is also involved in the formation of flail-joints, which are especially to be dreaded in the shoulder and hip.

Disturbances of growth are not uncommon. There is always the question of shortening of an affected limb. The skin of the paralyzed extremities often appears pale or cyanotic and markedly colder to the touch than that on the well side. The severe lordoses and scolioses which may result from paralysis of the muscles of the trunk have been previously mentioned.

Paralytic contractures of the leg and foot are very common and very troublesome. According to the nature of the paralysis, talipes planus, equinus, or cavus results. An extreme degree of contraction may make the patient walk on the dorsum of the foot. Genu incurvatum and recurvatum are also sequent to poliomyelitic paralyses and are not infrequently seen.

Fig. 135.—Acute poliomyelitis, hand walker.

With the loss of the quadriceps alone, or in combination with severe paralyses of the muscles of the back, the child is often able to move about only on all fours or to drag himself along by his hands.

Forms pursuing a peculiar course have been brought into recognition by the brilliant studies of Wickman, who is to be credited with gathering together the various clinical varieties under the name of Heine-Medin's disease.

1. Abortive forms are apparently very common in the course of epidemics and doubtless escape diagnosis when they appear sporadically. Typical initial symptoms, of even intense degree, appear, with which disturbances of digestion, anginas, or meningeal manifestations may be associated. The marked depression, the pain in the limbs, and the fever often give the impression of an influenza. A correct diagnosis is made only upon the appearance of paralyses in other members of the family who are similarly affected. In cases of this abortive type recovery is rapid and complete, even when the diminution of the patellar reflex and the hypotonicity of various muscle groups leave no doubt that the disease is acute poliomyelitis. The survivors of these abortive forms are, moreover, immune.

2. Cases with fatal course. Death is caused far less frequently by the severity of the infection than by the localization of the process in the vital centres in the medulla. These centres, as a general thing, are not primarily affected. The disease process may be initially developed, for instance, in the nuclei of the cranial nerves situated in the pons and the

interbrain, whence it causes paralyses of the ocular and facial muscles. Descending, it reaches the region of the vagus and the respiratory centres. Yet more frequently, the path taken in these fatal cases resembles that of the ascending spinal, or Landry's paralysis. The disease passes from the centres governing the leg muscles to those of the trunk, thence to those of the arms and, finally, to the respiratory centres, in rapid succession. This form of the disease usually terminates fatally within a very few days and rarely lasts as long as two weeks. It is preferentially occurrent among older children. The fever is not especially high and the temperature may even reach a normal point during the closing days. A fatal outcome is not invariable. The disease process may be arrested even after the respiratory centres are affected.

3. Pontine and bulbar forms have been already described. Paralyses of the ocular and facial muscles, of the soft palate, and of the tongue occur not only as initial symptoms, associated with widely distributed paralyses of the extremities, but in occasionally isolated types, and produce clinical pictures which simulate superior or inferior polioencephalitis. When such acute peripheral paralyses as, for instance, in the distribution of the facial nerve, appear sporadically, it is always permissible to entertain the suspicion of acute poliomyelitis.

4. Encephalitic or cerebral forms, leading to spastic hemiplegia, or to cerebral infantile palsy are known, but are of quite rare occurrence in the course of epidemics (see Chapter on Encephalitis, page 491), Wickman further describes yet more rare clinical types as atactic meningitis and polyneuritic forms.

Diagnosis.—The diagnosis is difficult in the initial stages, but may often be made successfully if proper weight is given to the characteristic sensitiveness to touch, to the pain upon motion, accompanied by an unclouded sensorium which distinguishes it from epidemic meningitis, to the profuse perspiration, and to the leucopenia. When there is danger of its confusion with meningitis, a lumbar puncture, and the demonstration of disturbed electrical reactions in the various muscle groups should decide the question. At the onset, some cases are mistaken for muscular or articular rheumatism, influenza, sciatica, polyneuritis or tuberculous meningitis. The rapid development of poliomyelitic paralyses, reaching their maximum spread within a few days, the flaccid, atrophic quality of these paralyses, the absence of sensory disturbances, and of interference with urinary or rectal control after the subsidence of the acute symptoms are characteristic and of distinct diagnostic value.

Differential Diagnosis.—Multiple neuritis is very uncommon in young children. It goes on to complete paralysis very gradually and is marked by long continued fever. Sensory disturbances persist even in its late stages. The implication of cranial nerves and the fact of pain upon pressure over the nerves and muscles involved can hardly be utilized in this differentiation. Ataxia is rather indicative of polyneuritis, but it may also occur in poliomyelitis. When the muscles affected are strictly limited to the peripheral innervation, the condition is very probably polyneuritis. Bilateral sym-

metrical symptoms, associated with the early appearance of edema, are similarly indicative of neuritis. The polyneuritic form of infantile paralysis is apt to be recognized only in epidemic invasions.

Post-diphtheritic paralyses also develop gradually. Furthermore, in this condition sensory and ataxic disturbances affect the soft palate in particular, which poliomyelitis, on the contrary, is only exceptionally involved.

Cerebral paralysis of children may now and then occasion difficulty in its differentiation from the more circumscribed processes. The contractures of poliomyelitis, for instance, may simulate spasms. The reactions of degeneration, as well as the diminution or loss of the deep reflexes, are indicative of infantile paralysis. It must not be forgotten, however, that exaggeration of the patellar reflex and of the ankle clonus may occur in poliomyelitis, and especially, in those forms in which the arms are paralyzed. There can be no doubt of cerebral paralysis when athetosis, chorea, idiocy, or epilepsy develop.

Birth paralyses of one or both arms, when they are first observed in childhood, cannot be distinguished from poliomyelitic paralyses saving by aid of the history. Inward rotation of the arm, as a result of paralysis of the infraspinatus, is common in birth palsies. Myatonia congenita may be recognized by the widespread, symmetrical flaccidity of the musculature and by the absence of the reactions of degeneration.

Progressive muscular dystrophy is a disease the differentiation of which, in difficult cases, can only be made from the history and by careful observation of its course. The dystrophies are bilaterally symmetrical and are gradually progressive. Paralysis of the legs in spina bifida occulta is often combined with paralyses of the sphincters and with usually symmetrical sensory disturbances.

Pseudoparalyses, due to delayed growth in rickitic patients, or in congenital lues, may be mistaken for the paralyses of poliomyelitis. Careful observation, however, shows their pseudonymic quality. They do not involve changes in electrical reactions.

Paralysie douloureuse, so-called, produced by jerking the arm in small children is also recognized as a pseudoparalysis and recovers promptly when proper treatment is applied.

Hysterical monoplegias, uncommon in childhood, may occur with distinct atrophy, but give normal electrical reactions.

Paralysis of the abdominal muscles, due to poliomyelitis, have been repeatedly mistaken for true abdominal hernia. The demonstration of coincident results of infantile paralysis and the location of the protruding tumor should protect one from this error.

Prognosis.—The older the patient, the greater the danger of a fatal termination. No prognostic conclusions can be drawn from the height of the fever. In epidemics the mortality from the disease varies between ten and twenty per cent.

Complete recovery is not very common. The severity of the epidemic must be considered in answering all questions of prognosis. Even widely distributed paralyses may disappear, but the resultant functional disturb-

ances are usually less severe if only a small area has been involved in the paralysis at the outset. The electrical reactions will assist the prognosis of individual muscle groups, since those that remain responsive to the induced current and do not give degenerative reactions will undoubtedly recover, while those with complete reactions of degeneration permit but a poor outlook for their full restoration. The reappearance of spontaneous voluntary movements in paralytic areas is particularly important. The return of function often precedes the reëstablishment of normal electrical reactions by a long period. Of the permanent paralyses, those seriously affecting the trunk muscles, as well as paraplegias of the legs, particularly when they involve the quadriceps and the muscles of the hip, compel a very grave prognosis. If but one leg is paralyzed, it is usually possible, by the pursuit of modern orthopedic methods, to enable the patient to walk.

Treatment.—During the first few days, absolute rest and quiet are essential. Very few but very fortunate experiences have been reported with the plaster of Paris bed designed for the entire body by the orthopedists. A bland diet, the absolute avoidance of alcoholic and even minor stimulants, and the effective care of the bowels are demanded. Frequently acetylsalicylic acid or sodium salicylate are given, and by rectum if necessary, during the initial stage. Hexamethylenamine may be tried. If graver symptoms appear, and particularly the indications of ascending paralysis, bloodletting over the spine, at the supposed level of the lesion, or a therapeutic lumbar puncture, should be employed.

Even in mild cases rest in bed must be strictly enforced for two or three weeks. After the initial stage, physical treatment of the muscles should be undertaken and should be continued uninterruptedly so long as there is any remaining hope of thereby improving function. This treatment, consisting of massage and electricity, should be continued for months. It makes great demands upon the endurance of physician and patient alike, but it is quite certain that improvement and often recovery of the muscles are achieved much more rapidly and completely under such treatment than is had if the paralyzed limbs are let alone.

Properly directed massage takes the most important place among physical methods of treatment. It should be employed once or twice a day for short sessions and in widely distributed paralyses and in bed-ridden cases it should extend to the exercise of the normal but inactive musculature also. The development of permanent contractures must be counteracted by active and passive movements. In fact, this tendency should be preguarded by the position which the child occupies in the bed. He should not be permitted to lie with the legs drawn up or curled up in a ball. In paralyses of the legs the feet must be protected from the pressure of the bedclothes by suitable wire frames. In some cases it will be found necessary to apply proper splints very early in the course of the disease in order to prevent contractures and overextension of the weakened muscles.

With the massage, warm baths 35°-38° C. (95°-100° F.) have been usefully combined. The addition to the bath of aromatics or of salt may be made.

The curative value of electricity is not so well established. The strength and quality of the currents employed must be so regulated, with reference to each stage of the paralysis, as to barely excite contractions. Usually, therefore, the anode is passed slowly over the affected muscle, while the strength of the current is carefully graded. The induced current is useful only in slightly impaired or convalescing muscles. Care must be taken to avoid injurious effects of the electricity. In very young children an indirect injury may be very easily done by the undue moistening and cooling of large areas of the body-surface in the application of wet electrodes.

Electric treatment should be given daily, at first, in five or ten minute sittings; and later every other day. After a period of treatment covering four to eight weeks, it is well to discontinue it for a week or two. Medicinally, strychnia or the iodides may be added.

A little later it becomes very important, particularly in extensive paralyses, to improve the general condition, so far as possible, by a nutritive dietary and by the opportunity of country or mountain air.

If the paralyzed limb but partially recovers, the normal muscles must be safeguarded from the evils of inactivity by exercise and resisted movements.

Permanent deformities very often show wonderful improvement under orthopedic treatment. Even children who are reduced to moving about on all fours, may be enabled to stand by protracted corrective measures. Operative interference is indicated only when spontaneous repair has ceased which, in general, will be from nine months to a year from the onset of the disease. Premature operative attempts are apt to fail.

The proper selection of available measures for relief must be made, in each individual case, in the light of a careful study of the functions which have been lost, of those which are absolutely requisite, and of those, again, which may most readily be dispensed with—an exercise of art, in the true sense of the word, which demands complete knowledge and control of the technic.

Ambulatory splints are never curative, but of merely palliative effect. For the purpose of fixing flail-joints, of regulating the movements of partially paralyzed muscles, and of correcting abnormal joint positions, they may be indispensable. In arthrodesis of the shoulder, knee, or ankle joint, when all or a majority of the muscles controlling the joint have been destroyed, artificial ankylosis may be necessary. Thus, for instance, a paralyzed arm which hangs helpless, but in which the musculature of the forearm and hand are preserved, may regain a large measure of its normal usefulness if it is ankylosed in its proper position at the shoulder.

A third procedure is the method of tendon transplantation after Niccoladoni. Healthy muscles, or portions of them are used for the reproduction of lost function by uniting them with the peripheral tendons of paralyzed muscles or by transferring their muscular power through artificial silk tendons (Linge), to the required point of insertion on the bone. These admirable methods have been greatly elaborated and have proved extremely

useful in many cases. For further particulars the reader is referred to the orthopedic text-books and, particularly, to the exhaustive monographs of Vulpius.

XII. DISEASES OF THE SPINAL CORD

1. Myelitis

The most important member of this group, acute poliomyelitis, has been discussed under the synonym of Heine-Medin's disease or acute infantile paralysis. All other forms of myelitis, and transverse myelitis in particular, are comparatively rare in childhood. As in adult life, they exhibit symptoms of pain, sensory disturbance and paraplegia, the functions of bladder and rectum being involved. It should be remembered, always, that these conditions may develop in children as a result of syphilis and that, in that event, they have a relatively good prognosis. Compression myelitis, a paralysis of the cord due to pressure and usually dependent upon tuberculous disease of the vertebral column, is more common. In mild cases it may cause pain in the trunk and back, weakness of the legs, and exaggerated patellar reflexes. In more severe cases the legs and the sphincters are paralyzed, the sensory functions are impaired, and there is a tendency to decubitus. Where a convexity of the spine indicates spondylitis the disease is readily recognized. The nervous symptoms and particularly the pain may be the only early indications of the disease, and, then, a careful examination of the vertebral column, with the aid of the Roentgen ray and with due consideration of the rigidity of the neck and back and the local tenderness upon pressure, etc., is essential. The treatment is surgical.

Landry's paralysis, in so far as it is dependent upon disease of the spinal cord, is an acute ascending poliomyelitis.

2. Tabes Dorsalis

Tabes dorsalis, the typical gray degeneration of the posterior roots, not infrequently has its beginning in the later years of childhood and is, in all probability, invariably due to hereditary syphilis. Its onset and course are insidious, resembling in a general way its progress in the adult. The ataxia and the consequent disturbances of locomotion are less prominent and the patellar reflexes are not always lost in childhood. A cardinal symptom is the failure of the pupil to react to light and, associated with this, lancinating pains, headache, optic atrophy, and urinary incontinence are observed. Headache and enuresis are especially early symptoms. Gastric crises and arthropathies sometimes occur. One sex is affected as frequently as the other.

The development of progressive paralysis is comparatively rare. The disease usually terminates fatally after puberty is established. Mercurial treatment is of no avail. If the optic atrophy appears early, as it commonly does, the patient should be placed in an institution for the blind.

3. Tumors of the Cord

Tubercles and gliomata of the cord are found in children. Sarcomata of the spinal meninges are more important, because they are often

amenable to surgical treatment. Their symptoms are frequently ushered in by pain and even by unilateral paralyses of the Brown-Sequard type. In the differential diagnosis, spondylitis and syphilis of the cord should always be considered.

XIII. ENDOGENOUS OR HEREDITO-FAMILIAL DISEASES OF THE NERVOUS AND MUSCULAR SYSTEMS

The heredito-familial or heredito-degenerative diseases of the nervous system, in the strict sense of these terms, give a characteristic history. They usually affect several members of a single generation; they may be transmitted through successive generations; they may arise without apparently external cause; and, as a rule, they progress without interruption. The identical type of disease is usually repeated in the given family and even its peculiarities and the age at which its first manifestations appear in the members of a given generation are practically the same. External injuries or infectious diseases may occasionally cause a premature development. In each succeeding generation the onset of the disease is apt to occur at an earlier age. Not all the members of a family are necessarily affected by the disease, but even the healthy may transmit the idiosyncrasy to their progeny. In all this group of diseases, sporadic or so-called erratic cases occasionally appear outside of the distinctly familial line. This possibility is always to be considered if a clinical picture presents features which isolate it from the symptom-complexes of non-hereditary disease.

The basic etiology of these diseases is shrouded in obscurity. Possibly alcoholism, conception during intoxication, great differences in the age of the parents, their consanguinity, or their advanced age may play, each of them, a causal part. When the disease has once made its appearance in a family there can be no doubt that a germ-injury results which is transmitted to the offspring, who are born with a nervous system in which some of the tracts or nuclei are deficient or deteriorate after a certain length of time; they succumb, in fact, to a premature senility (Jendrassik) used up by functional demands upon them and lacking the capacity for the normal repair or regeneration which occurs in healthy organisms.

Pathologic Anatomy.—In the pathologic anatomy of these diseases no inflammatory processes are discoverable. On the contrary, aplasia, atrophy, degeneration, affecting certain nerve paths and neuron systems electively, are the characteristic features.

The clinical picture in these cases presents an almost indescribable variation. Briefly, in the following paragraphs, a few only of the principal types are presented. So many variations of these are possible that the exceptions, it may almost be said, are more common than the rule.

Prognosis.—In the individual case, the prognosis is often too well known from the family history. Arrest or improvement is extremely rare. No prediction may be safely made that any one member of a family will be spared by the affliction. Not until the critical age, at which other members of the family have succumbed, has been passed, may any hope be entertained.

Prophylaxis.—As a matter of prevention, the question of weaning an infant from a mother who comes of an affected family and substituting a healthy wet-nurse may be considered. Any measures which tend to spare the nervous system from strain and to avoid all excessive muscular exertion may be considered prophylactic and are probably more useful, when the disease is fully developed, than the forced demands of gymnastic exercise swimming, sports, etc.

Treatment.—The treatment of these disorders fills an unsatisfactory chapter in medicine. Recourse can only be had to symptomatic measures and to the attempt at the psychic influence of both parents and children. If for no other reason, school attendance and intercourse with other children are to be recommended. For the rest, intelligent resort may be had to such physical methods of treatment as massage, hydrotherapy, baths, electricity, etc.

1. Amaurotic Familial Idiocy (Tay-Sachs' Idiocy)

This interesting condition affects exclusively children of Jewish descent. The onset of the disease is usually observed in the second year. The child, hitherto healthy, cheerful, and well-developed, becomes quiet and sleepy while the attentive parent notices the beginning of an impairment of vision. The patient no longer follows bright objects with the eyes. An ophthalmoscopic examination of the fundus reveals the pathognomonic feature of the disease, a grayish-white discoloration in the region of the macula lutea, larger than the papilla, and at its centre, in place of the fovea centralis, a cherry-red or rust-colored spot. The optic nerve shows more or less atrophy and becomes increasingly atrophic with the course of the disease. Nystagmus, inequality of the pupils and strabismus may also appear

With the rapidly increasing loss of sight, to which deafness is usually added, a rapid deterioration of all the mental functions, approaching complete idiocy, makes its appearance. Simultaneously, a progressive muscular weakness develops. The head is bowed flaccidly upon the breast, the limbs become more and more powerless, until they present the picture of complete bilateral paralysis. Usually they are atonic, but occasionally become spastic. The child dies by the end of the second or third year of life.

Pathologic Anatomy.—No marked anomalies are demonstrable macroscopically, but the microscopic findings are typical of the disease (Schaffer, Vogt). The entire central gray matter of the nervous system shows degenerative changes. The ganglion cells are distended and present characteristic structural alterations, revealed by staining methods, into which we cannot enter here. The cells of the nuclear layer of the retina show similar changes.

Diagnosis.—The findings in the macula lutea are pathognomonic, but have been absent in several cases.

Juvenile Amaurotic Familial Idiocy

At a later age, a familial form of the disease appears which presents so many analogies to the infantile type that, with Vogt, we may look upon it as a juvenile phase of the same malady. This conclusion is confirmed by the

microscopic pathological findings in the central nervous system, which consist in a diffuse degenerative process in the ganglion cells and bears a strong resemblance to that which is found in the Tay-Sachs' type. The onset of the disease is postponed until the fourth to the sixteenth year; it is of longer duration; but it similarly results in blindness, diplegic paralysis, progressive mental decline, and death. The main clinical difference is in the abscence of typical findings in the macula lutea and the development of a simple optic atrophy, even with a normal papilla. The blindness in these cases is probably entirely central, a fact which may obtain in the infantile Tay-Sachs' form. The Hebrew race furnishes a majority of these later cases but not to so exclusive a degree as in the infantile type.

2. Familial Cerebral Diplegias and Forms of Cerebrospinal Disease

The familial cerebral diplegias present clinical pictures which may resemble very closely simple cerebral diplegia, or Little's disease. The usual symptom-complex of spasticity, with ultimate involvement of the cranial nerves, and with mental disturbance, generally presents itself. To this relatively simple picture, however, is added a most variable combination of symptoms, absent or at least uncommon in the true cerebral diplegias. These include tremor, ataxia, optic atrophy, bulbar disturbances, bradylalia, idiotic laughter, loss of sphincteric control, and muscular atrophy or pseudohypertrophy. It is by this combination of symptoms, by its gradual and late onset, now in early childhood and again in established puberty, and by its slow but surely aggravated progress, that the disease should be distinguished, even in isolated cases, from the congenital or postnatal, exogenous, cerebral diplegias of infancy. Its familial occurrence alone is not sufficient argument for a diagnosis, since true Little's disease, caused by birth traumata, has been repeatedly recorded of successive members of a family.

The history of familial diplegia in a given family, despite of numerous individual variations, shows that the disease remains true to type and presents fairly similar pictures in each group. It extends commonly over years and even decades of time.

3. Diseases of the Myostatic System

1. Pseudosclerosis and progressive lenticular degeneration (Wilson's disease). The onset of this group of extremely interesting diseases usually dates back to late childhood. The group includes isolated or familial cases of diseases due to a pathologic condition of the extra-pyramidal tracts and, in true Wilson's disease, of the lenticular nucleus. This consists of proliferation of the glia with atrophy of the nerve cells and fibres and later, even cavity formation. Coincidently there is found a cirrhosis-like hepatic disease, which, however, is without symptoms during life. The symptoms include a peculiar mask-like rigidity and hypertony of the facial muscles and extremities. Babinski's phenomenon is negative and the deep reflexes not exaggerated. There is dysphagia, dysarthria, marked slowness of motion without paralyses, however, but moderate or severe tremor and distinct

psychic changes. Eye symptoms such as nystagmus and optic atrophy are absent, as are also evidences of cerebellar symptoms and disturbances. In acute cases, death may occur after a few months but the more chronic continue for several years.

2. Progressive torsion spasm, torsion neurosis or progressive lordotic dysbasia, is the term applied to a disease-picture but recently described. The condition probably depends upon some organic lesion of the central nervous system. Apparently it occurs chiefly among children of the Jewish race and after the period of infancy. It develops insidiously and progressively, is marked by pulling and twitching spasms, especially noticeable in walking. This gives the patient a fantastic clown-like appearance. The proximal muscles of the extremities and trunk are chiefly involved. The face is not affected and the intelligence not impaired. In the matter of differential diagnosis bilateral athetosis must be considered. Treatment, so far, has proved fruitless.

4. Hereditary Ataxia (Friedreich's Ataxia)

The classical type of this spinal disease is characterized by static and locomotor ataxia, the absence of the patellar reflexes, the development of nystagmus, and the formation of a peculiar form of club-foot with retraction of the great toe. To these usual symptoms, curvature of the spine (*kyphoscoliosis*), and mental disturbances are often added. The disease commonly begins between the fourth and the seventh year; it is gradually progressive and lasts for several decades

Another type of hereditary disease is presented in the *heredito-ataxie cerebelleuse of Marie.* In this form, the disturbance of coördination has the characters of cerebellar ataxia. The gait is staggering, rather than stamping or dragging, and the muscles of the trunk share in the disturbance of balance. The patellar reflexes are present and even exaggerated. The limbs may be slightly spastic. Nystagmus is rare; but other disturbances of the ocular muscles, producing strabismus and ptosis, are common. Optic atrophy is observed. Club-foot does not occur. The disease is of late development and often after puberty is established.

Between these two definite types there are all sorts of transitional forms, so that no strict lines of separation are possible. Symptoms of other heredito-degenerative nervous disorders, including auditory impairment, idiotic mirth, and muscular atrophy may be associated, in individual cases, with either type.

Static ataxia not infrequently manifests itself in constant motor restlessness, or in uninterrupted balancing and oscillatory movements of the head and limbs which resemble tremor, chorea or athetosis. These involuntary motions may become very annoying to the patient in walking. The speech is usually slow, scanning and indistinct. The pupillary reaction, as a rule, is normal.

The pathologic substratum of the disease is an aplasia or degeneration of varying distribution in the posterior columns of the cord or in the cere-

bellum. An atrophy of the lateral cerebellar tracts, the columns of Clark, and Gower's tract may ultimately ensue.

Diagnosis.—At its onset, and especially in those solitary cases which are common in this disease, its differentiation from cerebellar tumor, brain syphilis, and even infantile tabes dorsalis may need consideration. Normal pupillary reactions with distinctly atactic symptoms contraindicate tabes.

Treatment.—Exercises (Frenkel), recommended for tabes are said to give good results also in this condition. Considering the long duration of the disease the method is not without good purpose.

5. Muscular Atrophies

Early Infantile Progressive Spinal Muscular Atrophy (Werding-Hoffmann).—This disease begins in the first year of life and first shows itself in weakness of the legs. In succession, the muscles of the back, neck, shoulders, and arms are involved, until finally the entire body, with the exception of the facial muscles, is paralyzed. The extension of the paralysis, after some years, to the respiratory muscles ushers in the end. The paralyzed muscles may exhibit fibrillary twitching. Excessive development of the adipose layer may hide the extent of the atrophy. The deep reflexes gradually disappear; the electric excitability is reduced and, later, the reactions of degeneration appear. Speech, general sensation, and the control of the sphincters remain intact.

The disease is due to degeneration of the cells of the anterior horn and the consequent degeneration of the motor roots and of the muscles they innervate.

Diagnosis.—The distinction of this disease from congenital myatonia may be difficult. Its progressive course, the presence of fibrillary twitching, and the development of the reactions of degeneration are significant of muscular atrophy. The disease may be distinguished from acute poliomyelitis, occurring in early infancy, by its insidious approach, its progressive quality and its familial history.

Progressive Neurotic Muscular Atrophy, Peroneal Type (Hoffmann.)—In this disease, appearing commonly in later childhood, the peroneal muscles and other muscular groups of the legs are first affected and show a symmetrical atrophy. The gait suffers peculiarly, the entire limb is lifted high. The toe drops and is the first part of the foot to touch the ground. This is the so-called steppage gait. Sensory and vasomotor disturbances are often associated with these motor phenomena. The electrical reactions may be reduced or altered even in apparently normal muscles. The deep reflexes gradually disappear and fibrillary twitching in the atrophic areas is common. The muscles of the forearm and hand may become affected in precisely the same manner as those of the leg, leading to the deformity of "claw-hands."

The course of the disease, broken by pauses and remissions, may extend over a number of years. Its structural basis is a degeneration of the peripheral nerves supplying the affected areas, although changes are also found in the spinal cord, particularly in the column of Goll. Cases have been

recorded in which the nerves were palpable as thick strands, incident to an hypertrophy of their connective tissue (Dejerine-Sottas.) Since the disease is very chronic, orthopedic methods, by way of tendon transplantation, etc., may sometimes prove beneficial.

Progressive Muscular Dystrophy.—This is a purely muscular disease. In many individual cases a number of muscle groups atrophy and degenerate in rather characteristic grouping and succession. The atrophy may be preceded by a seeming hypertrophy of the muscles. But these excessively large muscles are weak and flabby. Their apparent increase in size is the result of a proliferation of the fatty and connective tissue elements, a pseudohypertrophy, or lipomatous pseudohypertrophy. Electrical examination shows no reaction of degeneration; as a rule fibrillary twitching is also absent, the reflexes are, at most, reduced. Disturbances of sensation and loss of sphincteric control do not occur. Deformities of the feet develop occasionally. The weakness of the gluteal lumbar and dorsal muscles, frequently involved, gives a characteristic clinical picture, the features of which are a peculiar waddling gait, a more or less high-grade lordosis, and a peculiar difficulty in stooping or rising from a recumbent position. In fact, children accomplish these movements only by the aid of their hands with which they climb up, as it were, on themselves.

FIG. 136.—Progressive muscular dystrophy, eleven-year-old boy. Pseudohypertrophy of the muscles of the nates and calves. Difficulty in rising to an upright position on account of the weakness of the muscles of the back.

The major number of cases of muscular dystrophy begin in childhood and not infrequently as early as the first year. The disease continues over many years and death is often due to tuberculosis. A more minute description of the various types of this malady is to be found in the text-books on medicine or neurology and, therefore, may be omitted here.

The demonstration of hypertrophy, the absence of fibrillary twitching, of the reactions of degeneration and of sensory disturbances, are important in the diagnosis, as is also the freedom from disease of the hand and the forearm and the normality of the patellar reflex. With these means of identification, its distinction from acute poliomyelitis, neurotic muscular atrophy, and myatonia should be a ready one. The possibility of its confusion with congenital dislocation of the hip and coxa vara, which may induce similar perversions of gait, should be noted. In this event the Roentgen picture is of value. The muscular hypertrophy of Thomsen's disease may be definitely distinguished by careful observation of the functional disorder of the muscles and by the evidence of the myotonic reaction. Combinations of the two diseases, however, have been described.

6. Uncommon Forms

Besides the forms of disease briefly described, there are other hereditary familial conditions which occasionally occur in children. Hereditary spastic spinal paralysis, amyotrophic lateral sclerosis, progressive bulbar paralysis, progressive ophthalmoplegia, Huntington's chorea or chronic progressive chorea, essential hereditary tremor, familial paralysis agitans, hereditary optic atrophy, general neurofibromatosis or von Recklinghausen's disease, progressive degeneration of the lenticular nucleus, among others, may be mentioned. Congenital myotonia or Thomsen's disease may also appear during childhood or even in infancy.

XIV. DISEASES OF THE PERIPHERAL NERVOUS SYSTEM

1. Paralyses

The peripheral paralyses show, in a general way, the same relations and demand the same treatment as in the adult. But a brief description, therefore, of their special features will be necessary.

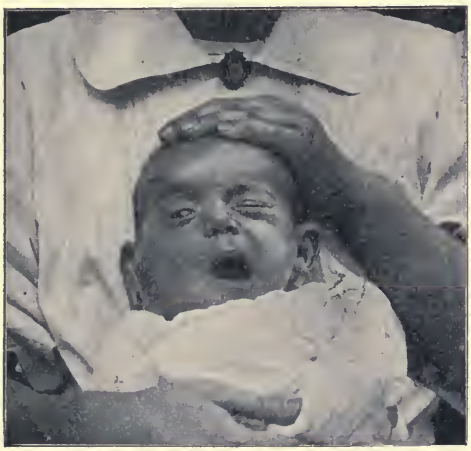


Fig. 137.—Right sided peripheral paralysis of the facial nerve as a result of tuberculosis of the right mastoid, six-month-old child.

Facial paralysis is very common among children and particularly so in infancy. It may be of congenital origin and due to a congenital absence of the nucleus (see page 488), but not infrequently it is the result of trauma from the use of the forceps in delivery or from extracranial pressure during passage through the pelvic outlet. Most frequently of all it results from ear infections and especially from tuberculous disease of the mastoid. (Fig. 137.) It may also be a manifestation of acute poliomyelitis localized in the pons, an origin always probable in the so-called rheumatic forms. It occurs, too, as a post-diphtheritic symptom.

Paralyses of the serratus, the radial, the median, the peroneal nerves, etc., are occasionally seen as the results of traumata, infective diseases and certain forms of poisoning. Birth palsies are discussed elsewhere in this volume.

A rather common event in childhood, up to the fifth year, is the painful paralysis of the arm—the *paralysie douloureuse* of Chassaignac. When the arm is wrenched, as in lifting a child by the hand or sustaining him when he stumbles, the member may be seized with intense pain and drop in pronation as though paralyzed. All motion is shunned, although careful observation reveals no paralysis whatever. All passive movements can be performed,

but the child objects and cries out, particularly when any attempt at supination of the forearm is made. This is not a neurosis, but a *derangement interne*, a disturbance of the joint, a subluxation of the head of the radius.

Treatment consists in forcible stretching, supination, and complete flexion of the forearm. This relieves the condition at once. In some cases, psychic limitations must be overcome by encouraging the child to reach for desired articles, the well arm being bandaged, if necessary, to restrain its preferential use.

2. Neuralgia

Occipital and trifacial neuralgias are practically the only forms which occur in children of school age. Usually these appear in children with neuropathic stigmata. Nerve pressure points may be very distinct. Etiologically, coryza, influenza and, in rare instances, malaria are in relation to the disorder. Errors in refraction must sometimes be causally considered.

Therapeutically much may be accomplished by regulation of the mode of living, avoidance of eye-strain, especially by lamplight, exercise in the open, attention to the action of the bowels, etc. The fitting of eyeglasses, the removal of adenoids and the treatment of chronic nasal disorder's are frequently remedial. Internally, a course of treatment with arsenic is often useful. During the acute attack, quinine, antipyrin, and other similar remedies may be tried, but care must be exercised to prevent their abuse. The daily application of weak anodal currents, for two or three weeks, may give permanent results.

3. Polyneuritis

The most frequent form of this malady, post-diphtheritic paralysis, is discussed under the head of the primary disease. Other forms of polyneuritis are extremely rare in children. It may develop in relation to any of the infectious diseases or to certain forms of chemical poisoning. Lead, arsenic, sometimes in its therapeutic use, and alcohol must be considered in this connection.

The course, prognosis and treatment are the same as in the adult. The differentiation from acute poliomyelitis is discussed briefly on page 517.

XV. DISEASES OF THE MUSCLES

Congenital muscular defects, congenital muscular atony, and muscular dystrophy have been already discussed. Other diseases of this class are so uncommon that the mere mention that they occur in childhood must suffice. Occasionally we meet with myoplegia periodica, myasthenia pseudoparalytica, polymyositis which may be primary or may result from an invasion of the Trichina, and finally with progressive myositis ossificans, a rare condition, but one which almost always begins in childhood.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

I. CONVULSIVE DISEASES

1. Spasmophilia or the Spasmophilic Diathesis

Laryngospasm, Tetany and Eclampsia.

Spasmophilia is a condition of hyper-irritability of the nervous system peculiar to early life, indicated by excessive electrical or mechanical reactions of the peripheral nerves and by the tendency to tonic and clonic convulsions.

Spasmophilia is an extraordinarily common disease in early childhood. It represents the major part of the convulsive disorders of children, including laryngospasm. It may exist for weeks and months in the apparently healthy without arousing the attention of parents or physician by any threatening manifestations. At certain seasons of the year the disturbing evidences of the spasmophilic diathesis may be demonstrated in a large percentage (thirty, or more per cent.), of all infants. Timely attention to this latent condition is of great importance, since therapeutic measures are possible, in many a case, which will suffice to avert the paroxysmal manifestations threatening the child.

The discussion, therefore, should be confined initially to *the distinctive characteristics of the spasmophilic state,* sometimes described as latent tetany or the tetanoid condition. Its most certain and constant sign, which may, indeed, be considered pathognomonic, is the fact of electrical hyper-irritability, the so-called Erb's phenomenon. This sign was discovered by Escherich and has been especially studied by Mann and Thiemich. The most valuable results of these important researches is the demonstration that in spasmophilia the kathode opening contraction, ordinarily produced in infants only with currents in excess of five milliamperes, appears with weaker currents, ranging down from four or three to even one milliampere, or less.

For practical purposes this one estimation is generally sufficient. In most spasmophilic infants, even when they are very small, the reaction may be obtained without an anesthetic. During a convulsion, however, particularly in children of three months or less, difficulties are frequently encountered. The hand is often closed so firmly that the contractions cannot be determined.

The test is made by placing the negative electrode on the breast or abdomen, while the positive, in the form of Stintzing's normal electrode, three centimeters square, is placed over the median nerve in the elbow. If necessary, the results may be obtained over the ulnar or the peroneus. In determining the minimal contraction it is customary to begin with the weakest current, for under conditions of hyper-irritability stronger currents will often produce a kathodal closing tetanus, which usually obscures the recognition of the opening contraction. With a little practice the voluntary movements of the child may be easily distinguished from the reaction to the opening and closing of the circuit. Every kathodal opening contraction

determined in this manner, with a current of less than five milliamperes indicates spasmophilia.

The determination of mechanical hyper-irritability is an easier matter, if less reliable. It is manifested by lightening-like contractions, resembling those produced by stimulating the nerve electrically, whenever the peripheral nerve trunks are tapped with the pleximeter. This occurs most readily in the case of the facial nerve. Tapping the cheek even with the finger, brings out this lightning-like contraction of all the facial muscles, the facialis phenomenon, or Chvostek's sign. When the child laughs or cries, in a word whenever the facial muscles are under nerve control, the sign cannot be developed. Direct idiomuscular contractions, especially in very young infants, may simulate the phenomenon. A contraction which can be produced by the nerve alone is conclusive, as for instance the contraction at the outer canthus of the eye when the masseter is sharply tapped. In infancy the facialis phenomenon is of great value and may save all the trouble of electrical examination. In older children it is hardly to be considered a certain indication of a spasmophilic diathesis. The facialis phenomenon, however, may be indefinite or altogether absent. It is often possible, in this event, to produce a very distinct radial or peroneal response by tapping the stimulation points of these nerves in the arm and at the head of the fibula. These tests are recommended in all doubtful cases. They are particularly successful in slightly emaciated infants. In any case, they are reliable only when voluntary motion has ceased.

Another pathognomonic sign is the Trousseau phenomenon, a test, however, which is frequently lacking. At times, its application is very painful and hence it is dangerous in children who are disposed to laryngospasm, on account of the resultant excitement. The Trousseau phenomenon consists in the artificial production of a tetanic spasm of the hand by compression of the vascular and nerve structures in the bicipital groove. It is best done by the use of an elastic band placed around the arm (Fig. 138), which must be left in place for two or three minutes. In infants the classical "obstetric position" of the hand does not always appear. The test is positive only when the hand becomes firmly fixed in a spasmic position, similar to this, from which it can be released, even by passive efforts, with difficulty.

Etiology and Pathogenesis.—Several etiologic factors are recognized which play a part in the development of spasmophilia. Heredity deserves first mention. Convulsions and particularly laryngospasm, are often familial afflictions. The history frequently shows its appearance in the parents and brothers and sisters of the patient. The facialis phenomenon may often be demonstrated in the mother. The view that spasmophilia may develop upon the basis of a neuropathic constitution may be established in many cases.

The disorder has a very obvious dependence upon seasonal change. The number of manifest and even of latent cases rises in the winter and spring months, and reaches its maximal point from March to May. The respiratory injury which results from continuous living in poorly venti-

lated quarters is generally held to be responsible for this prevalence, which is similarly shown in rickets (Kassowitz).

A third and very remarkable factor is the dietary. Breast-fed children almost invariably escape this disease. Children fed upon cow's milk recover from the disorder when they are placed on human milk. Discontinuance of cow's milk often results in abatement of the electrical hyperexcitability; which immediately reappears when cow's milk is again given. Other dietetic variants of irritability may be demonstrated, but there is no doubt that in many children a very special influence attaches to cow's milk. Further, it has been shown, in a number of instances, that neither the casein nor the fat, but rather the whey, is responsible for this untoward action (Finkelstein).

FIG. 138.—Tetany in late rickets (seven-year-old boy). Trousseau's phenomenon persisting on the left after removing the constricting band. Rapid recovery under treatment with phosphorous and cod-liver oil.

The age of the child has a bearing, since spasmophilia rarely makes its appearance earlier than the fourth month. Its manifestations are most frequent from the sixth to the fourteenth month. They are relatively common after the second year. Premature infants show a special predisposition to the diathesis.

Infectious diseases of all kinds may not only aggravate preëxisting tendencies and in paroxysmal form, but they may excite renewed manifestations in cases in which the hyper-irritability had subsided. It is a well-known fact that disturbances of digestion and nutrition may play a part in provoking spasmophilia. Indeed, the disease was formerly regarded as an auto-intoxication arising from the stomach or intestinal tract.

That, in a majority of cases, spasmophilia, is associated with rickets is well established; nor can there be any doubt that a close relationship between them exists. The greater frequency of both of these diseases in the spring, the rarity of spasmophilia in countries which are free from rickets (Japan), the similarity of the metabolic disturbances each presents, the combination of tetany with late rickets, and the beneficial therapeutic effects of phosphorus and cod-liver oil in both conditions, all tend to emphasize this relationship.

No characteristic structural changes have been discovered in post-mortem examinations of the central nervous system in cases of spasmophilia

or tetany. Attempts have been made to reach a better understanding of the nature of the disease by the study of its metabolism. The toxin suspected and sought for has not been found, but the more recent researches have discovered anomalies in the metabolism of the minerals. Quest has shown that the brain of the spasmophilic child contains less calcium than the brain of the normal infant and in metabolic experiments spasmophilic children show a negative calcium balance (v. Czybulsky, Schabad). It is by no means certain that these anomalies of the calcium metabolism are in the nature of the cause of tetany; according to Pexa's very recent reports, indeed, it seems improbable. That the metabolism of the alkalies and especially of potassium, deserves special consideration is suggested by other researches (Aschenheim, Lust). The theory of the parathyroid epithelial bodies, supported mainly by Escherich, is very interesting. This theory places the responsibility for the entire disease upon structural (hemorrhagic), or functional injuries of the epithelial bodies lying in the tissues surrounding the thyroid. It is known that the extirpation of these bodies may produce true tetany and may affect the calcium metabolism similarly with the alteration it shows in children suffering with tetany. Nevertheless, it appears that numerous histologic examinations of these epithelial bodies, of very recent date, do not support this theory. Still more lately the cause of the disease has been attributed to functional disturbances of the thymus (Lust).

Clinical Manifestations.—Three principal types of convulsions may develop upon the basis of the spasmophilic diathesis. (1) Laryngospasm or respiratory spasm; (2) eclamptic convulsions; and (3) tonic convulsions of a manifestly tetanic type.

Why in one child a given type should develop and, in a second case, another form cannot be explained. Neither can one phase be considered as an aggravation of another. Many infants suffer from laryngospasm alone. In others it is combined with eclampsia; and while there are children who suffer from tonic convulsions alone, there are cases in which these several forms may appear interchangeably. A persistent tetany, of demonstrable degree, is doubtless the most uncommon type in early childhood.

Severe attacks of spasmophilia are often preceded by psychic alterations, clearly evident to the careful observer. The child cries much, scares easily, is peevish, permits the approach only of certain persons, is unusually restless, watches everything that happens in its vicinity with apprehensive manner.

Spasm of the vocal cords; laryngospasm; spasm of the glottis. Mild spasm of the glottis, manifested merely as an audible whooping inspiration, may often be heard as the accompaniment of the least excitement, as in laughing or crying, without producing any further trouble. Every sound of this kind will be heard with suspicion by the experienced observer and will lead him to make the proper tests for the characteristic evidences of spasmophilia. Such mild degrees of laryngospasm may unexpectedly change into serious and even fatal forms if not carefully safeguarded. Severe attacks are very exciting occurrences. The face suddenly grows pale,

the head falls back and respiration ceases. The child makes several strenuous, but unsuccessful attempts to get air. The eyes protrude, the lips become purple, and the face, covered with a cold sweat, gradually takes on a leaden pallor. Consciousness is lost and a few twitching movements around the mouth and eyelids are the only signs of life. Finally, the limbs relax, the urine and stools are passed, and death seems inevitable. Suddenly, the spasm relaxes and a few crowing respirations show that the air is passing the still narrowed glottis, A deeper inspiration follows, after a few minutes respiration is gradually reëstablished, and the seriously endangered patient sits up—frightened and exhausted, but otherwise quite as well as he was before the attack. But laryngospasm does not always terminate in this way. Life may end in the midst of it. The patient is not asphyxiated, but the fatal result is due to the stoppage of the heart. In such a case artificial respiration is hopeless.

Again, the spasm of the glottis may pass into an attack of general convulsions. In severe cases, attacks of laryngospasm may follow each

Fig. 139.—Persisting tetany. Cardiac death. Carpopedal spasm persisting after death.

other rapidly. Twenty or more such attacks may occur in twenty-four hours. Milder and more severe attacks may alternate with each other, just as the most variable gradations between abortive and really dangerous spasms are possible. Attacks are generally somewhat less frequent at night. The exciting causes of an attack consist chiefly in psychic excitement, fright, crying, screaming, sudden awakening, and the rapid ingestion of a large meal. A very dangerous form of laryngospasm is the so-called expiratory apnœa, a spasmic arrest of respiration in the expiratory phase, which may cause death very suddenly and sometimes without attracting attention. Frequently it will develop and pass by entirely unobserved, since the crowing inspiration which ordinarily warns the attendant is lacking. Not uncommonly a rigidity of all the respiratory muscles, including the diaphragm, and, at times, extending to the entire body may be observed at the onset of an attack of severe laryngospasm.

These laryngeal and respiratory spasms, as manifestations of spasmophilia, occur almost without exception in rickitic infants. After the second year they are practically unknown and in this respect they differ from tetany and eclamptic convulsions.

Eclamptic convulsions, commonly known to the laity as fits, spasms or cramps, consist in attacks of localized or general muscular contraction, attended by loss of consciousness and quite resembling epileptic seizures. Slight convulsions often manifest themselves in mere vacancy or pallor of the face, combined with a twitching of the eyes and eyelids, the so-called "quiet fits." Usually, however, more extensive muscle groups are involved in these convulsions. The face is commonly affected and often all the limbs. A unilateral onset is not infrequently seen. Consciousness is lost from the beginning, the pupils do not react, and there is no response to skin stimulation. These symptoms are rarely preceded by a tonic spastic condition, usually combined with spasm of the glottis. If the attacks are severe, they present a very impressive picture. For a moment, the face is mask-like and vacant; the next, it is distorted by intense twitchings. The eyes move constantly and rapidly, or roll upward so that the sclera is alone visible. The tongue wags to and fro. The lips often froth, and in older children who have teeth, they may be blood-stained. The child lies on the bed, the entire body shaken by rhythmic shocks, giving vent to groans or short sharp sounds produced by corresponding expiratory spasms. The fontanelle is usually firmly tense during an attack. The pulse is rapid and irregular. During the attack, or immediately following it, the child often passes flatus, feces, or urine. The manifestations of motor irritability gradually disappear and the child falls into a sleep from which it awakens after a time very much exhausted. As a rule the duration of these eclamptic attacks is not very great, generally occupying from one-half to two minutes and but rarely extending to three or even five minutes. The convulsions, however, may follow one another so rapidly that a true status epilepticus results. In such cases fever, and at times, very high temperature 41° C. (106° F.), or more, may develop, probably as the result of irritation of the thermic centres. Fever is not found in any other form of spasmophilia.

Convulsions may appear once and never return; but of course this is not common. Usually a small series of attacks is observed. The individual events may be repeated very irregularly. One or two attacks a day, or as many as twenty or thirty in the twenty-four hours, may appear. In themselves, they are much less dangerous than the spasms of the larynx. In infancy they are apt to be especially frequent in the course of acute or subacute disturbances of digestion, particularly when there is much gaseous distention of the abdomen. They often attend the onset of febrile diseases. Among older children, they occur conspicuously, as so-called occasional spasms, at the beginning of gastric disturbances or of such infections as measles, varicella, etc. With other manifestations of spasmophilia, they are uncommon after the second or third year and in later childhood are rare. It is possible that some of the so-called reflex spasms of older children, supposedly caused by ascarides, obstipation, the presence of foreign bodies, etc., belong in this category. This is certainly true of the teething spasms which formerly played so distinctive a rôle. Many a case, believed to be epileptic, but recovering toward puberty, is proved by more careful study of its nature and history to be a late eclampsia (Thiemich).

Some of the repetitional absent fits of the run-about-age are associated with electrical hyper-irritability and relational to spasmophilia (see page 548).

Tetanic carpopedal spasms, or arthrogryposis, consist in peculiar tonic spasms of the hand resulting in the well known "obstetrician's hand." The phalanges are extended at an angle to the palm, with extreme apposition of the thumbs, as shown in Figures 139 and 140. If, at the same time, the forearm is flexed and pressed against the body the so-called "paw position" results. The feet not infrequently maintain a similarly spasmic posture, as seen in Figure 140. These tonic contractures usually persist for hours and doubtless, in many cases, are painful. The cause of the individual attacks is not known. If the condition has persisted for some time a pillow-like edematous swelling is often seen on the backs of the hands and feet.

FIG. 140.—Tetany. Obstetrician hand. Edema of the dorsal surface of hands and feet.

In infancy this tetanic position of the hands is often not so typical; the fingers are often flexed and, at times, abducted, but the thumb is always bent inward and sometimes wedged between the other fingers. The differentiation between this spasm and a simple closure of the hand, fist-like, with the thumb adducted, is always possible by a little careful comparison of the voluntary with the involuntary action. The tetany of the foot cannot be mistaken.

Other muscle groups may take a part in the disease-picture of these tonic convulsive conditions. The face is most commonly affected and, on account of the tensity of the muscles of expression, the patient wears a troubled, thoughtful, or downcast, pinched expression (Uffenheimer's "tetanic facies"). In severe cases, particularly in older children, the lips are drawn and slightly puckered (the fishmouth). Spastic strabismus is far from rare. To all these symptoms may be added spasm of the muscles of the neck, resulting in a fixed opisthotonos, and giving a picture which, with possible inequality and delayed reactions of the pupils, due to involvement of the unstriated musculature of the iris, may resemble that of meningitis.

Tonic spasms of the muscles of the trunk, arms and legs have also been observed. These are usually symmetrical. Not infrequently, at the height of the disease, micturition is embarrassed by spasm of the musculature of the bladder. *Ischuria paradoxa*, with dribbling of small quantities of urine and coincident retention, may result, which causes the progressive disten-

tion of the bladder until it reaches to the level of the umbilicus. While such symptoms of manifest tetany, lasting for hours or even days, may be intercurrent with every attack of laryngospasm and eclampsia, continuous attacks of so-called persistent tetany may last for many days or weeks. These are seen most frequently in cachectic children with chronic disturbances of nutrition. In this form of persistent contractions there is neither pain nor edema. There is a tendency among clinicians to consider this persistent tetany as relational to a loss of water or to molecular changes in the tissues. Others regard them as due to particularly severe injuries of the epithelial bodies.

Cardiac Death.—In all forms of spasmophilia sudden death, due to stoppage of the heart, is not uncommon. It happens most frequently during an attack among children who suffer with laryngospasm or expiratory apnœa, but occasionally it occurs independently of any acute spasms. The child, who has just taken his food, may be found dead in his crib. In such cases the ingestion of a large quantity of food seems to be the exciting cause of the sudden failure. Children in the status lymphaticus or, more particularly, those of the status thymico-lymphaticus, stand in special danger of this cardiac death. The author has recently expressed the opinion that these are cases of tetany of the heart, as spasmic phase of the spasmophilic diathesis affecting the paths of the cardiac nerve supply, on either the vagus or the sympathetic side.

Under the name broncho-tetany Lederer describes a condition of dyspnœa and consequent cyanosis, supposed to be due to spasm of the bronchial musculature, persisting for days or weeks. This leads to atelectasis and edema of the involved portions of the lungs and often ends fatally. The clinical symptoms simulate those of broncho-pneumonia. The condition is said to be differentiated by the Roentgen picture, the broncho-tetany showing a floating cloudiness, and mottled shadows indicating pneumonia.

Course, Duration, Complications and Termination.—The spasmophilic diathesis never disappears rapidly, but usually its individual manifestations may be relieved, or in a measure curbed, quite quickly. Even the electrical hyper-irritability may be reduced sometimes to a normal point within a few days or weeks. This abatement, however, is not synonymous with recovery. One may be assured of the latter only when the provocative injuries enumerated no longer call forth this hyper-irritability and its symptomatic expressions. Frequently has it been shown that a return to the use of the customary cow's milk mixtures will redevelop the entire symptom-complex. Then again it happens that the disease which, without the physician's aid, has wholly disappeared during the summer, will make its reappearance in winter without the intercurrency of any infection to excite the laryngospasm or the eclamptic convulsions.

The course in each case is distinctly individual. Not infrequently, the entire history is of a succession of either laryngospasms or eclamptic attacks. When the disease is long continued, as a result of persistently improper feeding, we generally find that first one and then another form of

tetany will prevail. In such cases carpopedal spasms are much more frequently found, with careful observation, than is generally supposed.

It is perhaps hardly permissible to speak of the complications of spasmophilia, but the condition itself often appears to complicate other diseases and then requires very special attention. In this event, the observation that spasmophilia frequently causes hyperpyretic temperatures (Finkelstein), is of the utmost consequence. This bears upon influenza, pertussis, serous meningitis and other diseases, and admonishes one that, for this reason, perspiration producing packs can be given, in the treatment of such children, only with extreme care.

There are several conditions that are supposably related to spasmophilia and are believed to be due to the same anomalies of metabolism as are the convulsions. These are opacities of the crystalline lens (cataract) and symmetrical hypoplasia or erosion of the enamel of the permanent teeth. These conclusions, however, rest upon incomplete data.

Systematic inquiries into the later fate of eclamptic children are very important in the correct prognostic evaluation of the disease (Thiemich and Birk, Potpeschnigg). It has been found that a large number of these children later show signs of neuropathy or mental defect and that only about one-third of them continue to develop with entire normality. In only exceptional cases does true spasmophilia eventually pass into true epilepsy.

Diagnosis.—The more important diagnostic points have been fully discussed. That definite electrical hyperexcitability, supported, in early infancy, by mechanical hyper-irritability; that the Trousseau phenomenon and the occurrence of true tetanic convulsions are pathognomonic will bear repetition. Laryngospasm, on the other hand, may appear as a symptom of other diseases, as in meningitis, cerebral sclerosis, etc.

It is well to note that conditions very similar to laryngospasm have been recognized in children who have passed the period of infancy and who exhibit the phenomena of so-called "absences." These disturbances do not stand in direct relationship to spasmophilia (see page 557). Occasionally cases are seen which are indubitably spasmophilic, but in which the kathodal opening contraction does not occur with currents of less than five milliamperes. This is especially true in children of less than three months of age. In later childhood the pathognomonic value of the K. O. C. is not absolute, while the predominance of the A. O. C. over the A. C. C. probably is. A number of points in the differential diagnosis of spasmophilia from other eclamptic conditions are discussed on page 542.

Treatment.—Breast-milk feeding, sunlight and fresh air are the most certain prophylactics, as they are the most effective remedies, against spasmophilia. They must be secured, if possible, in all cases in which the facialis phenomenon or other signs of latent tetany are discovered in infancy. If artificial feeding is unavoidable, the most important rules are to diminish the total dietary and especially to reduce the cow's milk to the lowest possible measure. This may range between one-third and one-half litre a day. It should be replaced by carbohydrates in the form of gruels, flour-pap, or barley water and, eventually, in the exclusive form of malt-

soup. Older infants may be transferred to a mixed dietary, including vegetables, stewed apples, etc., sooner than usual. Still older children may be fed adequately upon a milk-free diet for a long time, large quantities of food at any one feeding should be avoided under all circumstances, because of the danger that excess may precipitate a severe attack of laryngospasm and consequent cardiac death. This is particularly true when paroxysmal manifestations have already appeared. The internal administration of phosphorus and cod-liver oil, as directed on page 207, is always indicated. If dyspepsia or other disturbances of nutrition appear they must be treated, but without overlooking the necessary prohibition of large quantities of cow's milk.

In addition to, or in combination with the phosphorus and cod-liver oil, large doses of calcium salts should be ordered. The calcium lactate or the calcium chloride, ten grams (℥iii), to 200 c.c. (℥viii), of water may be given in doses of one tablespoonful, five times a day, or better still, according to S. F. Meyer, the calcium bromide, twenty grams (℥vi), to 300 c.c. (℥x), of water to be given in similar doses.

This treatment should be continued throughout convalescence from the various forms of tetany. It may be added that, at times, raw milk given, of course, under the most careful precautions, is preferable to sterilized milk.

Laryngospasm and eclampsia, however, require much more strenuous measures. The invariable rule may be laid down that the child who is suffering from these dangerous symptoms should be given a cathartic immediately. A teaspoonful of castor oil may be repeated several times. If meteorism exists, lavage of the bowel should be given at once, and again in two hours if indicated. Milk should be discontinued at the same time. Only boiled water, sweetened if necessary, should be given for twenty-four hours. Salt solutions are contraindicated. If mother's milk is obtainable the day is won; otherwise it is well to give rice or oatmeal gruels, to which thirty or forty grams (℥i-iss), of dextrin and maltose a day may be added. This diet is to be continued for several days. Phosphorus and cod-liver oil, in combination with calcium salts, should be given from the beginning. After this initial period cow's milk may be added gradually and carefully watched, commencing with only 50 c.c. (℥i), each day and increasing it slowly if the symptoms do not reappear. Frequently a transition to malt-soup feeding is to be recommended. Weeks may elapse before increases of weight are observed.

In atrophic infants, especially if the atrophy is due to flour-feeding injury, the feeding method requires grave consideration. Not infrequently it fails to influence the manifestations of spasmophilia. If no wet-nurse can be procured, one may have to be content with giving phosphorus and cod-liver oil and treating the disturbance of nutrition according to the usual methods. It is apparent that the persistent forms of tetany, as a rule, do not yield very readily to dietetic influences. Doubtless protein-milk will be found especially useful in this connection. Finkelstein recommends that large quantities of fluid be given by enteroclysis and that moist packs be employed for their sedative influence.

Severe eclamptic attacks, particularly when they occur in rapid succession, require special treatment. The tepid bath, an enema, cooling applications to the forehead, and moist packs for the reduction of the high fever are measures which can be employed even when the physician is not at hand. If the convulsions persist, or should they occur very frequently, chloral hydrate, to be given per rectum, is indicated. To infants, a dose of 0.5 gram (grs. viiss), may be given and may be repeated, if necessary, in a short time. By mouth smaller doses should be given. This remedy may be employed also when severe and threatening laryngospasms occur. Urethane, in one or two gram doses (grs. xv-xxx), by mouth, or in 1.5-3.0 gram doses (grs. xxv-xlv), per rectum, is also useful. Chloroform inhalations as a means of relieving the status eclampticus are to be considered only in extreme necessity. It is possible, at times, to stop repeated convulsions by lumbar puncture. Several good results have been reported in the spasms attendant upon pertussis (Eckert).

Where there is a tendency to single spasms, and especially to spasm of the glottis, it is permissible to continue for a few days the internal administration of sodium bromide or calcium bromide.

℞ Sodii bromidi, grams 3.0 (Grs. xlv).
Aquæ destillatæ, c.c. 100 (℥iii).
M. Sig.—One to two teaspoonfuls, three times a day.

The writer has had but little experience with the subcutaneous injection of magnesium sulphate so highly recommended by Berend. The solution is prepared by adding 8.0 grams (℥ii), of magnesium sulphate to 100 c.c. of water; from 15 to 20 c.c. being used for the injection, which may be repeated, if necessary, on the following day. The injection is painless and is said to affect the spasmophilic symptoms rapidly and favorably. The presence of cystitis, nephritis, or meningitis are contraindications to its use. Special dietetic treatment is not required, but the avoidance of food containing large quantities of salt is essential. Phosphorus and cod-liver oil may be given in combination.

In closing, a few suggestions may be offered for the treatment of severe laryngospasm. Everything that may frighten or excite the child, such as throat inspection, the test of the Trousseau phenomenon, and even the unnecessary presence of persons in the room, should be carefully avoided. The child's head should rest upon a horse-hair pillow or upon an air cushion, in preference to a feather cushion. A vessel of cold water and a hypodermic syringe, containing camphor in oil, should be kept beside the bed. During the periods of most frequent attack the child must be under direct supervision by day and by night. At the onset of a severe attack, the attendant should watch quietly at first, but if the child does not get air after a very short time some cold water should be dashed over it. If the attack still continues the back of the tongue is to be depressed with the finger and, if necessary, an injection of camphor in oil is to be given. Attempts should be made to practice artificial respiration. The effort often meets with an insuperable obstacle in the fact that the spasm firmly closes

the opening between the vocal cords, which will not permit the passage of air. As soon as the child begins to breath of its own accord, the well-known crowing sound being heard, it is best to desist from any attempt at further aid in order to avoid exciting a new convulsion. If, however, the breathing does not become regular artificial respiration should be continued. Intubation or tracheotomy never can be done in time, because death is due to stoppage of the heart and not to asphyxiation. The practice of prior and continued intubation to tide the child over days of greatest danger may be considered.

Finally, it may not be superfluous to add, that comparatively to the frequency of laryngospasm, deaths arising from it are quite rare. It is usually possible to master the several manifestations of spasmophilia.

2. Eclampsia Due To Non-spasmophilic Causes

Not all eclamptic attacks occurring in infancy or among older children develop upon the basis of a spasmophilic diathesis. A certain proportion of them are caused by organic diseases of the brain, by way of malformations, porencephalus, intracranial hemorrhage, sclerosis, lues of the brain, and hydrocephalus. A certain number are due to meningitis. Of those not traceable to organic causes, the so-called idiopathic convulsions of early childhood, a majority come under the head of spasmophilia. A goodly percentage, however, ranging from 7 to 20 per cent. of all eclampsias, are nothing less than true epilepsy of infancy which, in later life, develops into the typical form. It is also true that certain poisons occasionally cause convulsions in children. Among these are alcohol, opium, santonin, mushrooms and other vegetable poisons. To complete the etiologic tale, it must be noted that convulsions are a phenomenon of uremia. Urinalysis and the presence of edema may suggest this origin in individual cases.

There remains a considerable number of convulsive disorders, the nature of which is not yet known. A part of these are of reflex quality and some appear to be the result of intestinal auto-intoxication. Presumably many a grave eclamptic picture depends upon serous meningitis, of the relations of which but little has been known until recently. It seems to be true, also, that in children beyond the period of infancy, who show a certain neuropathic tendency, convulsive attacks are occasionally caused by extrinsic influences, such as infectious diseases, disorders of digestion, and notably profound psychical disturbance. The psychasthenic convulsions of Oppenheim, the nervous convulsions of Bendix and the functional epileptic convulsions of Bratz are in this category. These functional epileptic convulsions, however, do not occur, as might be imagined, in direct consequence of psychic traumata, but these traumata, rather, are followed by days or weeks of nervousness, during which period the convulsions may develop. A favorable prognosis distinguishes these conditions from true epilepsy.

The so-called terminal convulsions seen in extremely cachectic infants, after rapid losses of weight and particularly of water, in pylorospasm, or in the course of chronic disturbances of nutrition and always in the stage of

extreme exhaustion, have nothing to do with spasmophilia. Most probably they are to be regarded as the results of the action of bacterial poisons, or of poisons developed in the intermediate metabolism of the child, or of serious molecular changes incident, in part at least, to the loss of water in the nerve cells (Thiemich).

The differential diagnosis of these several forms of convulsions is often very difficult. The attacks may be identical in character. The demonstration of mechanical and electrical hyper-irritability shows, in most instances, that the eclampsia is of tetanoid origin. It should be remembered however, that the spasmophilic child may be affected, like any other, with meningitis, encephalitis, etc. Emphasis must be laid upon the fact that spasmophilic convulsions are almost unknown before the third month; that

Fig. 141.—Unilateral convulsion in a four-day-old child with porencephaly.

individual attacks are usually of short duration, and that the child hardly ever makes any outcry during the attack. In the recognition of meningitis, the tensity of the fontanelle and the general hyperesthesia are particularly important points. Idiopathic convulsions cause a bulging of the fontanelle only during the attack unless, indeed, one convulsion follows another without intermission.

Lumbar puncture is to be urgently advocated in all doubtful cases. Convulsions occurring during the first three months, and particularly during the first few weeks of life, are commonly due to organic brain lesions, and most frequently to meningeal hemorrhage resulting from difficult labor. Cases have been seen, however, in which the convulsions were associated with nutritional disturbances and disappeared speedily when the diet was regulated.

Paralyses or pareses remaining after a seizure, even though they are

transient, testify to the organic cause of the convulsions. A unilateral type of convulsions, on the other hand, is quite often seen in purely functional eclampsia. Cases to which a parental history of epilepsy attaches always arouse the suspicion of that disease. This is true, also, of those instances in which single or group attacks occur at longer or shorter intervals without provocative causes. Such children may be naturally fed; they give no evidence of febrile disease or of nutritive disturbances; and the electrical reactions of their peripheral nerves are constantly normal. It must not be forgotten that an interval of several years may fall between these first convulsions of infancy and the later manifestations of epilepsy. In older children, in whom convulsions develop with accompanying high fever, it is well to remember that one may have to deal with the onset of an infectious disease.

Treatment.—The symptomatic treatment above described may be found equally useful in most of the non-spasmophilic convulsions. Thorough evacuation of the bowels is almost always indicated, together with hydrotherapeutic antipyresis. In the terminal convulsions of digestive or nutritional disease in infancy, neither cathartics nor chloral hydrate should be used. In these cases the subcutaneous infusion of a physiologic saline solution or its use by enteroclysis is indicated. For the rest, treatment must depend upon the making of an accurate diagnosis, since meningitis, brain syphilis, early hydrocephalus, exogenous poisoning, uremia, etc., will each suggest its particular therapeutic indications. Incision of the gums, circumcision, and other similar resources of perplexity cannot be countenanced.

GENERAL MUSCULAR HYPERTONIA, WITHOUT SPASMOPHILIA

Briefly, attention should be called to the fact that frequently infants who are not sphasmophilic and do not evidence organic brain lesions, exhibit continuous muscular spasms, especially of the lower extremities. These are definitely related to acute or chronic disturbances of nutrition and are particularly common in children who have been injured by excessive flour feeding. They persist for weeks, or even months, with but slight variations of intensity, until they disappear gradually with a general improvement of nutrition. These hypertonias quite commonly affect the muscles of the back and neck to the point of developing a well-nigh constant opisthotonos which, combined with other spasms, simulates the picture of cerebral disease. It does not show any exaggeration of the reflexes. In the early months of infancy such spasms, constituting the myatonia of Hochsinger, are observed in the course of such serious general disease as sepsis or congenital syphilis, or following such severe injuries as extensive burns.

3. Nutant and Rotatory Spasms

This rather uncommon disorder consists in the development of turning, twisting and, more rarely, of nodding movements of the head which the affected child exhibits upon lying down. While very evidently the movements are involuntary, the child remains entirely conscious. The disease is seen, at the earliest, in the fourth month and disappears after the third year. It is usually continuous for weeks or months, varying in its intensity from time to time. Upon close observation it will be noted that the peculiar

short, but fairly rapid movements of the head are increased whenever the eyes are fixed upon any object, and are usually accompanied by a distinct nystagmus. This symptom is noticed, however, at other times. It is exaggerated if the head be held still and may appear independently of the head movements. All manifestations of the disease cease during sleep. The nutant spasm appears chiefly in rickitic children. A neuropathic taint may play some part in its development. Its exciting cause seems to be found in the relative darkness, or the poor lighting of the room in which the child is housed (Raudnitz), necessitating the turning of the eyes upward and sidewise when the child seeks to look at the light.

Diagnosis.—Nystagmus due to amblyopia, with clouding of the cornea, etc., must be excluded. The twisting of the back of the head in rickitic children who suffer with hyperhydrosis, eczema, etc., may be readily recognized as a voluntary thing. Complex movements resembling nutant

FIG. 142.—Pseudotetanus.

spasm are not uncommon in idiocy. The so-called stereotypies may be cause for confusion.

Treatment.—Placing the child in well-lighted rooms, the improvement of its general health, and the treatment of existing rickets usually result in the disappearance of this form of spasm.

4. PSEUDOTETANUS

Pseudotetanus (Escherich) is a rare disease, occasionally seen in infancy but generally in children from four to six years of age. The clinical picture commonly develops, in full, without fever. It simulates traumatic tetanus in all particulars, excepting that no wound is present and no tetanic bacilli are demonstrable. Taking an ascending path from the legs, a tetanic rigidity of all the muscles of the trunk, back, neck, and face, with resulting trismus or lockjaw (Fig. 142) gradually develops. Only the hands and arms and the ocular muscles remain free. As in true tetanus, excitement, noise, etc., call forth convulsive paroxysms. During sleep the rigidity relaxes, but does not entirely disappear. The characteristic signs of spasmophilia are lacking.

Complete recovery usually occurs in the course of several weeks. The nature of the disease is unexplained. It may have some relation to true tetanus (Pfaundler). The treatment is symptomatic. Chloral and bromides

and, if necessary, morphin, with feeding by stomach tube are indicated. Whenever true tetanus is even suspected the specific serum treatment should not be neglected.

5. Epilepsy

Epilepsy is a very common disease of childhood. Nevertheless it will be but briefly discussed, since in a general way it presents practically the same manifestations as in the adult.

Symptomatic and typical forms of the disease are recognized. The first of these is etiologically related to organic lesions of the brain, involving inflammatory and degenerative processes and resulting in scars, cysts, etc. It may be due to traumata and is often a consequence of the cerebral palsy of children (*q. v.*). In this section only the true form of epilepsy will be discussed.

True epilepsy is a disease in the etiology of which hereditary factors play an important part. Among these, parental epilepsy, alcoholism, neuropathies, nervous diseases of various forms, and congenital lues are numbered. Its well-known but variant manifestations will be briefly described. Three principal types are recognized; the major epilepsy or *grand mal*; the minor epilepsy or *petit mal*; and the psychic equivalents. Certain paralytic equivalents, by way of paroxysmal and at times painful paralyses of definite parts of the body, without spastic symptoms and without loss of consciousness, are also distinguished.

Attacks of grand mal occur in childhood as they do in adult life. An aura, not always constant, is succeeded by sudden pallor, falling, and loss of consciousness. In rapid succession follow a stage of tonic general spasms, associated with cyanosis, and a stage of clonic convulsions in which the tongue is bitten and other injuries may be sustained. In this phase, foaming at the mouth, fixation of the pupils, evacuation of urine and feces, etc., occur. Finally the patient falls asleep and upon awakening is completely amnesic (see Figures 143-144). Any characteristic aura and the initial outcry noticed in adults are often wanting in children.

The minor and simpler attacks of petit mal are more frequent in childhood than in later years. They are manifested by sudden and very brief fainting spells, by attacks of dizziness, or by so-called absences. The child pales for a moment, the expression grows blank and distant, the sentence or the play is interrupted, and any object in the patient's hand is dropped. Then it all passes and the child resumes his play, as though nothing had happened, or occasionally he falls asleep. In other instances, rudimentary manifestations of motor irritation appear; a few lightning-like twitchings, sometimes recurring in an identical muscle group, are observed.

Still another epileptic manifestation which may be seen in children is the salaam spasm or nodding epilepsy, a succession of rapidly repeated bowings of the head or body, as if in greeting. In this form consciousness is not necessarily lost, but amnesia may follow. Yet another type is the *epilepsia cursiva* marked by a sudden run forward, which terminates either in a

fall or an encounter with some obstacle, when unconsciousness or a convulsive attack ensues.

The psychic equivalents of the epileptic state do not play so important a

FIG. 143.—Epileptic attack. Beginning of the clonic stage.

FIG. 144.—Epileptic attack. Last stage. Foaming at the mouth, sleep.

rôle in childhood as in adult life, in that they rarely tend to the commission of criminal acts. Still they are not entirely unknown among children and manifest themselves in fits of unreasonable ill-temper or excitement, marked by disobedience, stubbornness, senseless demands upon

their companions, and the exhibition of intense anger. The more characteristic type of dream-like abstraction, associated with apparently well-considered action, with the desire to roam and to force the way into strange places, etc., have been observed. Subsequent amnesia is not absolutely essential to this type.

The number, frequency and periodicity of the seizures differ in each case. The status epilepticus, leading to continuous attacks, which may seriously threaten life is not of unusual occurrence even in childhood. The beginnings of epilepsy in children have been carefully studied by Birk. Three forms of onset may be distinguished.

1. Epilepsy taking an intermittent course. One attack or a succession of seizures, occurring in infancy and often failing of correct diagnosis, is followed by a long interval of freedom, after which attacks of grand mal or petit mal develop. The period of the reappearance of the disease is commonly that of school age or of puberty. In the interim, the child often presents symptoms of nervous disturbance, alterations of voice, irritability, pavor nocturnus, fits of anger, and often diminished intelligence.

2. An uninterrupted course of epilepsy persisting from earliest childhood. This form is the most common one, although the disease usually takes on a more moderate degree for a certain time after the initial attacks in infancy. In this intermediate period, in fact, the seizures are sometimes so mild as to escape recognition and, if observed, their true interpretation awaits the reappearance of the *grand mal.*

3. Forms of epilepsy which appear in later childhood. In these types, again the favorite period of onset is either at school age or at puberty.

Course and Termination.—Very few epileptics escape some measure of mental impairment. Generally speaking, a gradual change of character and a progressive mental decay, which varies from the mildest degree of defect up to complete idiocy, sets in. Easily angered, irritable, not infrequently given to acts of violence, temperamentally brutal, these children, even though their mental faculties are fairly well preserved, present difficult problems in training with respect to their familial, social and communal relations. As a rule, the psychic deterioration is in direct proportion to the number of attacks the child suffers, so that the frequent recurrence even of *petit mal* must be considered serious. Recoveries or improvements so great as to approach very closely to recovery are uncommon, but are not unknown. The prognosis is always more favorable if the child is treated early and if the initial attacks appear late.

Diagnosis.—Attacks of grand mal may be confused with other types of convulsions. Its differentiation from infantile eclampsia has been previously discussed (page 542). Late eclampsias may be distinguished by their history and by the discovery of the pathognomonic signs of spasmophilia, a not unimportant distinction to make because of the favorable prognosis which, under suitable treatment with phosphorus and cod-liver oil, the latter affords. The symmetrical defects of the enamel of the teeth as a symptom of tetanoid disturbances of metabolism, in early childhood, are of some significance. The hyperpyrexic, reflex, and sporadic convulsions of later childhood are

assignable, in part, to eclampsia and, in part, to true epilepsy, but probably relate to the latter as a type of metabolic epilepsy to be discussed presently. Some of these cases, exhibiting frequently repeated attacks of convulsions due to psychic trauma ("affekt-epilepsie"), in children of emotional quality and suggestive of a neuropathic type, must be distinguished from true epilepsy because of their much more favorable prognosis. Nodding spasms, *Maladie des Tics*, simple fainting, may be readily excluded by careful observation. A differentiation from hysteria may be very difficult both in the major and in the minor form. At times it even happens that hysteria may be associated with epilepsy. Severe injuries, scars, teeth marks in the tongue, foaming at the mouth, absence of the pupillary reflex, are pathognomonic signs of an epileptic attack. The readiness with which they are excited, the retention of consciousness, the success of treatment by cold water and by psychic influence, are indications of hysteria. No diagnostic dependence can be placed upon the presence or absence of amnesia. Success or failure in diminishing the severity of the disease by a change of scene is not conclusive evidence for or against hysteria, since like indeterminate results have been frequently observed in epilepsy. Other psychic or physical signs usually permit a differentiation even when the physician does not see the attacks himself. We may generally hold fast to the fact that deterioration of the mental faculties by repeated epileptic attacks is typical, and where this is not clear, room may be left for a more favorable prognosis.

It should be noted that some children suffer slight, repetitional lapses of consciousness which cannot be laid at the door of either epilepsy or hysteria. They are sometimes called narcolepsy and deserve mention on account of their relatively favorable prognosis.

Children, between four and eight years, are often affected with occasional "absences" of brief duration, without actual loss of consciousness. These may appear from ten to forty times a day for years, without interfering with the patient's development. They do not respond to treatment with the bromides, but they do react to psychic stimulation. Mental influences may play some part in the causation of the disorder. Long continued rest in bed, or a change to country life may temporarily arrest its course. Since its relationship to spasmophilia has been shown, it may be expected that spontaneous recovery in individual cases will be hastened by dietetic treatment, together with the use of phosphorus and cod-liver oil.

Treatment.—First of all it is to be said that certain cases of epilepsy, and not of the symptomatic type alone, may be cured or benefited by operative interference. For further light on this subject, the reader is referred to textbooks of neurology.

The correction of anomalies of refraction is not to be overlooked, since it may do much to diminish the frequency of attacks. A positive Wassermann reaction demands energetic antiluetic treatment. A number of cases of metabolic epilepsy show a certain dependence upon disturbances of nutrition, especially in the field of digestive function. The careful study and suitable management of these conditions may reduce materially the number

of seizures. These dietetic corrections should include the avoidance of food rich in purins and in sodium chloride, and the adoption of a non-irritating and largely salt-free vegetable diet. Not only the abandonment of gross dietetic errors is implied, but the treatment also of gastric disorders and particularly of hyperacidity, and the relief of obstipation. Complete abstinence from the use of alcohol is essential.

Medicinally the bromides are the sovereign remedy. The sodium bromide alone, or in combination with the ammonium salt, is to be preferred. Enough of the drug should be given to arrest, if possible, the attacks. For very young children the total daily dose may run as high as six grams (ninety grains). Older children may receive as much as eight grams (two drams). Under such dosage, the clinician must be prepared, of course, for the appearance of signs of bromidism. The quantity may be decreased after a while without diminishing the results, but its administration in adequate doses must be continued not only for many weeks, but in some cases for years. Usually through such long courses of treatment, the doses of the bromide may be greatly and gradually reduced. Smaller doses of bromine are commonly satisfactory if the patient is kept on a salt-free diet after the method of Toulouse-Richet. This is a rather difficult form of treatment for long continuance and is dangerous in a degree, so that it is best employed periodically or when rapid and intense action is required. Excellent results are reported in some cases with the use of pheno-barbital (luminal) which may be given in doses of three to five grains three times daily. Thyroidin has proved of occasional benefit. Combinations of the bromides with opium, according to Flechsig, have been used successfully in several cases when bromine alone was ineffective.

In the treatment of the epileptic attack itself we are limited to the appropriate protection of the patient against injury. The sleep following the attack should not be disturbed. The status epilepticus may be combated by an enema of chloral hydrate, containing 0.5 to 2.0 grams (8-30 grs.) or with amyl hydrate, in solution of 3 to 4 grams (45-60 grs.), to 100 c.c., (3 ounces), of warm water to which is added five to eight drops of tincture of strophanthus or opium if necessary. According to Vogt, a high enema is effective during or immediately after a severe seizure. In mild cases attendance at school, and preferably at a special school, is possible and advisable. At a more advanced period it may become necessary to place the patient in a private sanatorium or in a public institution devoted to the care of epileptics. In the choice of occupation only light tasks about the house or garden should be considered.

II. CHOREA MINOR (St. Vitus Dance)

Chorea minor is a neurosis of subacute course, probably of infectious nature, chiefly observed in childhood, and marked by choreic disturbances of motion and associated psychic changes.

The motor disturbances in chorea consist of a series of lightning-like contractions of individual muscles combined with certain peculiar forms of

incoördination (Foerster). The faults of coördination are seen in the hesitation over voluntary acts and their complication with involuntary movements; in the failure to maintain attempted positions; in the lack of that coöperative work of accessory muscles and of the opposing muscular groups upon which the development of equilibrial motor complexes depends. The impulses to spontaneous movement excite the marked contractions of widely separated muscles, which become annoyingly prominent in every act and occasion a constant restlessness of the entire body.

The fully developed clinical picture of chorea cannot be mistaken. The child is in constant action, now jerking the shoulder, now hoisting the arm in a peculiar manner, now again throwing the head to one or the other side. The face is in continual muscular play; the most variable expressions appear in kaleidoscopic array; at one moment brimming with merriment, the next changing to embarrassment, restlessness, fright, or modesty. The muscles of the face, shoulders and hands are always affected most severely; but, in many cases, those of the trunk and the legs also participate in the muscular unrest. In the latter event, the gait is very much disturbed and standing and sitting may become impossible. The patient falls helplessly when placed upon his feet. In these severe cases all voluntary action is thwarted. Such a child is unable to dress or undress or to feed himself. Speech is similarly disordered and often fails entirely; the little sufferer, with much grimace, whispering and stuttering but a few words. Very gradual is the approach to this fully developed phase of the disease. At the onset, the motor disturbances are less marked and may even be interpreted as intentionally erratic. The affected child is often punished at school for his inability to write, and at home when he drops objects on every occasion. His school companions quickly notice the muscle twitchings and, mocking and imitating them, tend to induce their rapid aggravation. It is always true that the involuntary movements are more marked when attention is directed to them and the child attempts to control them. This motor restlessness subsides in sleep, except in the rare form of chorea nocturna in which it may be even more intense during the night.

Fig. 145.—Chorea minor. Seven and one-half-year-old girl. Told to sit quiet, involuntary motions of the muscles of the face and neck, and the right arm and leg.

Physical changes in chorea minor are usually discernible from the very beginning of the disease. They are evidenced by irritability, peevishness,

sudden fright and failure of mental concentration. Serious psychoses hardly ever develop in children.

In addition to the characteristic symptoms described, a flaccidity of the limbs or hypotonia is usually demonstrable (Bonhoeffer), in fully developed cases. It is usually recognized, in lifting the child, by the familiar discovery of the definite looseness of the shoulder. Commonly the reflexes are not decreased. Gordon has called attention to a quite peculiar reaction of the patellar reflex, determined in a certain number of cases. The lower leg, thrust forward by the reflex, is fixed for a moment in this position and sinks back slowly to the point of rest. Muscular power is affected only in exceptional instances and sensation is always intact. Pupillary variations, such as inequality, abnormal dilation, and hippus are seen occasionally. Fever does not belong to the clinical picture, but may develop with a complicating rheumatism.

Cardiac phenomena appear in almost every case. These may consist only in irregularities of the pulse or arythmia; but very frequently sighing or blowing murmurs are heard. They may wholly disappear, with recovery from the chorea, or may remain permanently present. These heart murmurs must be regarded as evidences of a verruciform endocarditis, which may lead to permanent cardiac lesions. Pericarditis is much less common.

Duration, Course and Termination.—As a rule, chorea shows a fairly rapid development of the disease to its full height; and, after a variably long persistence, a gradual subsidence to its final disappearance within three or four months. Cases continuing, however, six to twelve months are observed. Exacerbations are not unusual and recrudescence of the disease is quite common. Recovery is the rule, but fatal results have been reported in 2 or 3 per cent. of the cases. In these fatalities we generally trace the results of a septic endocarditis, or unexpectedly sometimes, hyperpyrexia and coma develop in connection with a complicating serous meningitis.

Uncommon Cases.—Hemichorea is unusual. As its name suggests, its symptoms are confined to one side of the body.

Chorea mollis, or paralytic chorea, runs a course marked by pareses and muscular weakness. Its motor disturbances may play a very secondary part, but a flaccid monoplegia or paraplegia and a loss of reflexes ensue, which may be correctly interpreted only by the antecedence of the choreic movements or by the subsequent return of motor power. The prognosis in these pareses is favorable.

Etiology.—Age and sex play a causative part. The disease is very uncommon before the fourth year and is probably seen most frequently between the seventh and the twelfth year. Girls are peculiarly predisposed. Neuropathic stigmata must be counted among the favoring factors. Psychic trauma, sudden fright, etc., may excite chorea. Syphilis but rarely enters into consideration as an etiologic factor.

A relationship to articular rheumatism in its several forms is so frequently demonstrated in choreics that there is growing tendency to regard chorea as a form of rheumatic disease, coördinal with the joint inflam-

mations and with endocarditis. In this sense we have come to speak of an infectious chorea despite its afebrile course.

In actual experience not only is an endocarditis often seen in the course of chorea, but the heart disease may antedate the appearance of the chorea as a consequence of a rheumatic attack, or an articular rheumatism may supervene and, at times, alternate with exacerbations of chorea. Indeed, the development of an acute chorea has been seen during a recovery from an attack of articular rheumatism. The rarer complications also of articular rheumatism, such as erythema nodosum or multiforme, have been observed in connection with chorea.

Pathogenesis.—No definite infective organism is yet known. The disease is generally supposed to be due to toxic or infective injuries of large brain areas. According to Bönhoeffer, a disease of the cerebellar peduncles and of the cerebellum itself may be held responsible for the choreic disturbances of motion.

Diagnosis.—Simple irregular movements, due to embarrassment, are frequently seen in young children, which may suggest the picture of an early chorea. A flaccidity of the limbs and the progressive course of the disease indicate chorea. Diadococinesis, or the impossibility of rapidly alternated pronation and supination of the hand, is usually observed even in the early stages of chorea. It is quite valuable as a diagnostic measure in older children. It is important to note that a symptomatic chorea is observed in certain brain affections, such as cerebral infantile palsy. Particularly in the event of a hemichorea care must be taken, by testing the reflexes, etc., to exclude organic brain disease. Congenital chorea or choreic symptoms which develop during the first year of life are always so associated. Tuberculous meningitis sometimes begins with hemichorea.

Chorea electrica will be discussed later. Hysterical chorea usually appears as an imitative disease and may spread, epidemic-like, through a school as a result of the admission of a case of true chorea. Superficially, it presents a closely similar picture. Close examination and due consideration of the attendant circumstances will point to a correct diagnosis. In such cases the presence or absence of the peculiar type of patellar reflex, already described, is of probable diagnostic value.

Treatment.—The most important factor in the treatment of chorea is rest. The child should be kept out of school and placed in a suitable environment. The members of the immediate family may not exercise a desirable influence over him and it may become necessary to remove him to entirely new surroundings. Absolute rest in bed is urgently recommended until the severer symptoms have disappeared. The disease may affect the general nutrition of the child and care must be taken that he receives sufficient nourishment. Sometimes this is far from an easy matter on account of the interference with the act of deglutition and the consequent necessity of careful feeding. Cold water treatments are harmful; while protracted tepid baths, and especially long-continued moist packs over the entire body surface, often have a very favorable effect. A combination of change of

scene, of rest in bed, and of such methods of bathing may make all other treatment unnecessary.

Medicinally, nothing is to be expected from the salicylates and other antirheumatic remedies. Antipyrin seems to be more useful. The disease is possibly shortened by a course of treatment with arsenic. The dose should be gradually increased in the usual manner. It must not be carried too far if good results are to be expected. From six to eight drops of the Fowler's solution, three times a day, should be the maximal limit. Injections of sodium cacodylate may also be employed. Salvarsan, or neosalvarsan, given intravenously or intramuscularly, has acted favorably in several refractory cases.

In severe forms of the disease, chloral hydrate may be necessary to secure for the patient the necessary sleep. If the heart is affected, amylene hydrate, in doses of 3.0 grams (45 grs.), per rectum, or bromide may be preferred. Thiemich recommends the trial of scopolamin hydrobromide, in doses of one-half to one milligram ($\frac{1}{60}$-$\frac{1}{20}$ grain), once a day.

PARAMYOCLONUS MULTIPLEX; CHOREA ELECTRICA

Paramyoclonus is characterized by symmetrical convulsive contractions of single muscles or groups of muscles, those of the shoulder girdle, the arms and the face being chiefly involved. They may be very frequent and do not respond to treatment. Psychic trauma and hereditary neuropathies play an important part in their etiology.

Chorea electrica is a collective term covering various convulsive diseases which are not proper to chorea minor. Some of these cases are probably related to paramyoclonus; others, perhaps, to *Maladie des Tics*, to epilepsy or to hysteria. These latter types show a marked tractability under proper suggestive treatment. In the epidemics of so-called chorea electrica, observed by Dubini in upper Italy, in which a large mortality was noted, there was undoubtedly a form of organic central or meningeal disease.

III. NEUROPATHIC AND PSYCHOPATHIC CONSTITUTION

(Hereditary Neuropathy)

The neuropathic or psychopathic constitution plays a large rôle in childhood. Its full significance has been realized but recently. Under these terms are grouped those abnormal hereditary tendencies of the nervous system which tend to develop in the direction of a number of nervous or psychic disturbances. Primarily, these abnormal tendencies manifest themselves in reactions to physical, mental, or psycho-emotional stimuli, which in their intensity, duration and effect put the personal life of their subjects into sharp and distinct contrast with that of normal individuals. Such neuropathic or psychopathic children can hardly be considered as mentally deficient. In fact, the greater impressionability of their nervous system not infrequently gives them an intellectual, artistic, or esthetic quality of rather unusual order. In them, indeed, we have to deal with developmental conditions, the correct appreciation and proper pedagogic training of which may be of the highest importance in the evolution of character.

The explanation of the condition is to be found in two chief factors, not infrequently coöperative. The first of these is the inheritance of certain

stigmata which may be traced back to generations of varied nervous and mental disease in the family of one or both of the parents. Neurasthenia and hysteria in the immediate ancestry are of essential significance.

The second factor is seen in the injurious influence of a mentally unhealthy environment in which the child is reared and from which he receives his first impressions and experiences in the relation of his ego to the world at large. These injurious influences are apt to be exaggerated in the case of an only child of the family.

Other extrinsic conditions, in the burdens of school life, the awakening of the sexual powers, the effects of traumata or of infectious diseases and the like, have a secondary but an important influence in such lives. They do not, as a rule, affect the normal child, but in the nervously or psychically abnormal they often excite the most variable afflictions which must come up for later discussion. Among the far-reaching exogenous injuries from which such young nervous subjects suffer is that of the habitual use of alcohol. It should be said that an exudative diathesis is often combined with the symptoms of neuropathy.

Symptoms.—The clinical pictures which the neuropathic constitution presents are as varied as life itself. In early infancy such children are usually distinguished by their abnormal tendency to fright and by the fitfulness of their sleep. As their intellectual faculties develop, various psychic peculiarities appear, conditioned upon the type of their hereditary stigmata and the influence of their particular environment. They are chiefly manifest in their emotional qualities. Extreme and intense excitability, outbursts of violent temper or emotion, not due to any physical condition, may make their appearance even in late infancy. The child soon holds the entire family under his tyrannic sway and does not yield the sceptre it holds without a struggle. He compels the mother to sit beside his bed until he falls asleep, or keeps her rocking him half the night. He will eat only certain foods and accepts them only from certain dishes. The most ordinary tasks of every-day life are subject to his expressed desires. Parent or would-be teacher is punished for every supposed slight to the small egotistical personality by hours of pouting and sullenness, by the obstinate refusal of food, or even by actual attack, or by an outburst of anger in which the child rolls about on the floor and acts as though insane.

In another type, an abnormal timidity appears to govern the entire nature of the child. He is afraid to be alone or to stay in the dark, afraid of even harmless domestic animals, etc. Or, again, he exhibits an extreme variability of temperament. The most insignificant occurrence throws the child out of his customary routine and changes his happy demeanor to one of sadness. Unlike the grief of the heathy child, this is not of momentary duration and easily consoled. The mood persists for hours, perhaps, or even days and is maintained against all consciously pleasant impressions. Still another type of child is extremely prone to complain; exquisitely hypochondriacal thoughts employ his waking moments or he constantly overflows with sickly sentimentality. Yet, again, the child develops into a precocious wiseacre in whom every trace of childish innocence is soon lost.

In brief, it is not hard to see in the individual case the mirrored picture of its environment or the product of an inherent tendency, fostered by its surroundings, in the easily influenced character of the child.

Elaborations of idiosyncrasy are encountered in the imaginative realm which are described as pathologic dreaming. The active phantasy of the child not only translates him into the successive rôles that his play-dreams make for him, but it becomes difficult for him to distinguish between imagination and reality. Upon this phantasiastic tendency the child constructs long stories as though they were tales of actual experience, all the incidents of which may be shown later to be of pure invention (*pseudologia phantastica*). Not infrequently these tales are found to serve the momentary advantage of the child. Lying accusations of adults by such children are not uncommon and the stories they may tell of sexual attack are often "made of whole cloth."

As physiologic stigmata, unusual activity and fidgeting are observed in many cases. These peculiarities may be noted during the first year. There is often times a markedly labile quality of the vasomotor mechanism, which manifests itself in rapid changes of color in the face, in easy variations of the pulse-rate, and in dermatographia. The deep reflexes are often increased, while the conjunctival and swallowing reflexes, on the contrary, may be diminished. Not uncommonly the facialis phenomenon is encountered in children of school age who show no other signs of the spasmophilic diathesis. Intercurrent affections of the skin and the air passages cause an abnormal degree of irritation and coughing in such children (Czerny). The so-called stigmata of degeneration, such as cranial and facial asymmetry, malformations of the ears or the genitalia, cryptorchidism, strabismus, left-handedness, etc., are to be interpreted as in the adult. They are worthy of note, especially when several of them are present, but they have no pathognomonic significance.

Peculiarities of habit are common. Such children suck their thumbs or their lips, bite their finger-nails, pick the nose, pluck at the hair, or shake the head incessantly. Such constantly repeated motions, which may be continued even in sleep, the *jactatis capitis nocturna* or the bowing spasm of the trunk, etc., are called stereotypies.

The more moderate disturbances of physical and psychical equilibrium, so far discussed, may be kept within due bounds or speedily improved or even cured by suitable training and by the hygienic influence of a wholesome environment. Tact and persistence, the very qualities apt to be wanting in the home of such a child, are of, course, indispensable. Submission to the will of his elders, truthfulness, habits of cleanliness, the suppression of unfit desires—all of these essential qualities are best acquired from the example of the teacher. The neuropathic child will never acquire them in the constant presence of the living example of their opposites. Intercourse with normal children is greatly to be desired and is often the most direct remedy for egotism and timidity. The school is a preparation for the future life of the child and is greatly to be preferred to individual tutor-

ing at home. When the tendency to indulgence in phantasy and pathologic dreams is especially marked, the mind must be directed to real values by practical occupations around the house or in the garden. Modelling, manual training, the collection of natural objects and the observation of nature, are means to this end. The pleasure of production inherent in every child must be aroused. Fairy stories, tales of horror, and lessons unadapted to its age, may injure the child materially. It is important that such children should be sent to bed at a suitable hour with unalterable regularity. Sexual impressions should be guarded against. To allow the child to sleep with adults, to witness the careless personal exposure which some parents permit themselves, or the exercise of their more intimate relations, is to incite a probably serious disturbance of his psychic balance.

All of these measures and precautions, but superficially touched upon, are of greater importance than disciplinary attempts which, as a rule, accomplish little when the training has once been started in the wrong direction. Likewise, they are far more important than the prescription of "nerve-building foods," or of hydrotherapeutic methods which have proved absolutely useless and suffice only to promote the acquirement of a full-fledged hypochondriasis.

To these milder manifestations of the neuropathic constitution may be added symptoms which have a distinct pathologic quality and which periodically overshadow the general status and seriously affect the life of the child. Certain symptom-complexes commonly attach to the conception of neurasthenia and still others to that of hysteria. Apart from these quite a number of isolated expressions of disease, such as migraine, tic malady, etc., may appear. The true psychoses may develop upon this constitutional basis, but they are uncommon in childhood. Some epilepsies, a large percentage of cases of enuresis, and certain disorders of speech may also relate themselves to the neuropathic group.

The psychopathic constitution, in so far as it may be congenitally traced is often in close heredity to more or less serious degrees of feeble-mindedness, a fact which makes the prognosis in the given case more grave.

1. Migraine; Hemicrania

Migraine is a distinctly hereditary neurosis. It occurs in childhood, but in less acute form than in later years. Its periodic attacks of severe headache, associated with the vomiting of bile, resemble those of the adult in every other respect, making any special discussion of the disorder superfluous.

2. Maladie des tics Convulsifs

Under this term we group certain peculiar convulsive movements of the muscles of the face, especially those concerned in winking, scowling, grinding of the teeth, etc., or of the muscles of the trunk, neck or limbs, producing such acts as nodding, jerking of the shoulders, grasping with the hands, and even dancing and jumping. These movements monotonously repeated and without variation, are distinguished from the stereotypies by their convulsive character. As a rule, they increase very markedly when the

patient believes himself under observation or when he becomes excited. The movements are to a certain extent controllable by the will. In connection with them some children utter a cough or bark or even indecent words.

There are cases, mistaken for tic, which are, in reality, manifestations of hysteria, as, for instance, the saltatory reflex spasm and other similarly complex motions, which are readily amenable to treatment. In other cases, these convulsive contractions are associated with voluntary or primarily intentional movements; they are quite analogous to the stereotypies in their mode of origin, but are not infrequently curable. The best success is generally áttained by properly individualized gymnastic treatment. The patient being placed before a mirror, the attempt is made to keep the affected muscles absolutely at rest by will control alone, while the abnormal innervation of these muscles is suppressed by means of slow, irregular, voluntary movements. These exercises must be carefully supervised and directed by the physician.

3. Emotional Respiratory Convulsions; Absences

Under this caption are included convulsive respiratory pauses, occurring during emotional excitement and particularly in exhibitions of anger. Consequently they are sometimes called convulsive rage. They simulate very closely the true laryngeal and respiratory convulsions of spasmophilia. They do not produce, however, a true laryngospasm. Respiration, as a rule, stops in the midst of a crying-fit and following either a deep unobstructed inspiration or a profound expiration. In the course of the crying, the child suddenly rolls its eyes, grows rigid and cyanotic, throws himself about, and finally falls unconscious. Usually he recovers completely within a few seconds. The signs of the spasmophilic diathesis, a heightened electrical excitability, the Trousseau phenomenon, etc., are wanting.

These convulsions occur chiefly during the run-about-age, from two to five years, although they are met with in infancy. In some instances the child, doubtless, is volitionally concerned, to a degree, in the initiation of these attacks; learning to incite them when, in the face of parental opposition, he desires to get his own way. When, however, the mechanism of the attack is once set in motion, it passes beyond the patient's control. The spasms may appear spontaneously as a result of fear or other psychic excitement. In some cases this condition is preceded in infancy by a true spasmophilic laryngospasm.

Prognosis.—The prognosis is supposed to be entirely favorable. When one has seen some very severe cases, however, he will accept this view cautiously, although no fatal outcome has been reported.

Treatment.—The treatment should be essentially prophylactic, but not to the point of giving way, as is too often done, to the child's every whim and fancy. The child should be given, rather, to understand that he must expect severe punishment if he holds his breath. It is often possible to avert an attack by threat or blow or by the sudden distraction of some unexpected occurrence.

The more anxious and disturbed is the attendant, the more readily do the "absences" occur. On this account a change of environment or even a short stay in hospital may prove as desirable in severe cases of this sort as it is in other forms of neuropathic disturbance, where the object is to eliminate the element of sympathy.

A combination of the bromides with a course in psychotherapy is to be recommended. In the presence of the attack itself the treatment appropriate to true laryngospasm is indicated. Cold water should be dashed in the patient's face and, if necessary, artificial respiration is to be employed.

PATHOLOGIC REFLEXES

The emotional respiratory spasms are actually the prototype of a number of nervous disturbances which may be regarded as pathologic phases of the normal reflexes. They are usually classed as monosymptomatic hysteria. They involve the reflex perpetuation of abnormal motor phenomena which originally had been caused by local pathologic conditions. After the disappearance of the original pathologic stimulus they persist, in a degree, as a functional habit, responsive to physiologic stimuli of ordinary character, but in the exaggerated degree which depends upon the emotional quality of the subject. Thus habitual winking or spasm of the eyelids may persist after conjunctivitis; polyuria may continue after irritation of the bladder; nervous cough after laryngitis or bronchitis. Seemingly typical attacks of whooping-cough may occur for weeks or months after recovery from true pertussis or may be reproduced by a slight bronchitis, etc. Some forms of infantile vomiting, rumination, etc., are to be better understood, perhaps in the same light.

Therapeutically it is possible sometimes to break up the abnormal symptom-complex by the introduction of some new and unusual factor in the reflex event. Thus, in rumination or habitual vomiting, the feeding of gruel often serves a good purpose. With older children, suggestive treatment, together with use of the faradic current is often useful in particular with nervous cough, polyuria, and the like. It may be necessary to confine oneself to the treatment of the general nervous condition of these patients.

4. Pavor Nocturnus

This very common malady consists in a sudden awakening from sleep in a state of fear. Generally, it occurs but once in the course of a night, but it may be frequently repeated at irregular intervals. Children suffering with this abnormal condition are usually between four and eight years of age. The attack occurs during the early sleeping hours. The child rouses suddenly with an expression of deadly fear or intense fright; screams, defends himself from imaginary objects, clings to his mother, and is quieted only by lighting the room and reassuring him for several moments. Half an hour may elapse before the child drops to sleep again. His confused speech often indicates the nature of the alarming dream of ghosts, robbers, animals, etc., he has experienced. Often it seems that the child does not really emerge from its dream into full consciousness. The remainder of the night passes undisturbed and in the morning the child remembers neither the dream nor the physician who sat by his bed.

These attacks, often described as nightmare, may be traced sometimes to such physical causes as tight clothing, an overloaded stomach, or a distended bladder. Intestinal parasites may be occasionally responsible; but the cause is more often attributable to respiratory obstructions, chronic

coryza, hypertrophy of the tonsils, and adenoids. The treatment, if all these points are taken into consideration, will not infrequently give prompt results. It is desirable, however, to study the often highly developed imaginative quality of children so affected and to prohibit everything by which the imagination may be excited. Fairy tales and other fantastic stories, theatrical performances, moving pictures, the influence of sexual impressions, and the use of alcoholics, should be avoided. As in all the functional neuroses of childhood, the element of fear should be especially considered. Sometimes the pavor will disappear at once if the child is removed from the room in which the parent or the nurse sleeps.

In the matter of the differential diagnosis, it must not be forgotten that true epilepsy may hide behind an apparent pavor nocturnus. The occurrence of irregular exhibitions of enuresis is especially important in this connection.

5. Neurasthenia

Neurasthenia is characterized by rapidly increasing exhaustion of pathologic degree, with an abnormal irritability. Neurasthenia of extrinsic origin, the nervous collapse which comes as a result of excessive demands upon physical and mental energy, the acute nervous exhaustion, is very uncommon in childhood. It occurs, probably, only in those who attempt wage-earning with school attendance, even at the expense of the night hours. The neurasthenia of childhood which develops upon the basis of a neuropathic diathesis is quite common and is a many-sided thing. The nervous system of those who, whether constitutionally or as a result of improper training, are unprepared for the experiences and unfit for the ordinary exertions of life, proves incapable of meeting the small daily tests and demands which the average school makes. This is particularly true when the readily exhausted child is spurred on, by his own ambition or that of his parents—and ambition is apt to be boundless in the neuropathic family—to persistent efforts during his school years which simply serve to destroy the capacity of his nervous system. Repeated disappointments, following upon strenuous endeavor, are the hardest physical blows which such a child has to sustain. In fact some of the suicides of children are traceable to this fact.

The more aggravated forms of masturbation, so commonly observed, are not a cause of neurasthenia but the result rather of psychopathic stigmata. Nevertheless, the habit may contribute measurably to the increase of neurasthenic symptoms.

The objective symptoms of neurasthenia are identical with those of the neuropathic constitution. To repeat them in brief: increase of the deep reflexes, diminution of the conjunctival and swallowing reflexes, rapidly alternating pallor and flushing, a variable pulse-rate, dermatographia, and the facialis phenomenon. Added to these are often noted an increase of blood-pressure, at times amounting to a pulsating rigidity of the arterial walls; variations in the vigor of the nerve supply to the facial muscles and the pupils; restless movements; spasm or tremor of the eyelids when closed

(Rosenbach's phenomenon), hyperhydrosis, stuttering, etc. As a rule, varying combinations of a number of these symptoms appear. A slight degree of anemia, may be coincident and is often, but erroneously, considered causal of the neurasthenia.

Physically, neurasthenia may lead to a variety of complaints which can be merely mentioned. The most important of these are headache and head pressure. These occur chiefly among school children and their exciting causes are to be found in anemia and in the unhygienic conditions of the schoolroom. Headache usually increases during school hours and may eventually become continuous, so as to be present even on rising. It may diminish if the child is allowed to sleep late on Sunday. Anorexia, gastric discomfort, obstipation and vomiting, the latter often occurring on the way to school or after forcing down an unwelcome breakfast, are not uncommon.

Nervous asthenopia (Wilbrand and Sänger), deserves particular mention. It is marked by the sense of flickering before the eyes, by the escape of tears, by headache and eyeache upon reading. It shows no organic lesion. Acute diseases have a much more severe course in neurasthenic children than in others. Pertussis (Czerny), and bronchitis, for instance, often taken on an asthmatic character. It has already been noted that itching and coughing are exaggerated symptoms in these patients.

Disturbances of sleep are common with them. It is not unusual to find an infant who lies wide awake for half of the night. Falling asleep is difficult and often long delayed. In the morning the child dislikes to wake up and leaves his bed but poorly rested and refreshed. Troublesome dreams, pavor nocturnus, tics, and even true somnambulism may further disturb the patient's rest.

In the psychical sphere, the element of fear and the tendency to hypochondria demand particular emphasis. For the latter, the adults immediately associated with the child are commonly responsible, but it deserves special consideration because it is often accentuated by the pernicious activity of the physician.

In children of advanced school age, mental exhaustion, resulting in feeble memory, inattention and want of concentration may play an important part. The child loses all zest for work; an indefinable fear of every school-task to which he is set possesses him; failure of accomplishment causes further deficiency, and a consequent apathy and dulness results which strangles all endeavor. Should the parents attempt to relieve the situation by providing private instruction for the child, making still greater demands upon his already flagging energies, the result is a further injury to his nervous system rather than an improvement in his school work. A child in this stage of exhaustion not infrequently commits moral wrongs, as lying, stealing and even offering violence to the parents, acts which are wholly at variance with his true character. In the differential diagnosis of these conditions, dementia præcox or hebephrenia, to the primary stages of which they bear some resemblance, requires careful consideration.

Treatment.—The treatment of neurasthenia in children must combine psychical and physical measures in a suitable manner. Hydrotherapeutic procedures, abundance of fresh air, moderate and healthy play, together with the remedy of the existing anemia, are the best means to be employed. It should be remembered that heroic attempts to harden the child by cold water bathing often do harm and serve to increase the neurasthenic symptoms (Hecker). In severe cases a period of complete rest is essential and removal from school for several weeks may be absolutely necessary. Equally essential is it to obtain an accurate knowledge of the home environment and of the psychical influences bearing upon the child. Since in only exceptional cases may the child's whole environment be changed, an individual plan of treatment, which takes account of all the factors determinable and which, too frequently, must content itself with compromises, must be worked out in each case. Institutional treatment, climatic cures, sea-baths, etc., have the advantage that they introduce the child to new surroundings for a time, at least. Suggestive treatment is of small value for the relief of neurasthenia. The personality of the physician may accomplish much, provided he can master the effects of gross deficiencies of training. Thus it may be possible, at least, to eliminate the constant discussion of the child's affliction as a topic of conversation. He may even give the parents to understand that the subject should not be mentioned in the presence of the patient. Further, they should be impressed with the fact that punishment aggravates the condition; that regularity in the entire routine of living, and especially of meal hours and bedtime, must be rigidly enforced; and that all pathologic ambitions must be curbed.

Special attention should be directed to the diet. Czerny and Siegert have called attention to the fact that a diet consisting largely of milk, meat and eggs often gives the nervous disorder a strong stimulus. Protein is to be minimized. In fact, a diet consisting largely of vegetables and fruits, without eggs and with but little milk, is most appropriate. The possible use of alcohol and, by older patients, of tobacco must be ascertained and discontinued. Masturbation must be stopped. Organic disorders, such as oxyuris, tend to increase the nervousness and should be carefully remedied.

In certain cases, the use of bromides may be unavoidable; at least temporarily, in order to combat possible sleeplessness and sexual irritability alike. As little as possible should be used. Moist packs sometimes serve the same purpose. The anorexia, obstipation, headache and mental irritability will never be conquered by drugs if one does not succeed in providing fresh air, and light, healthful exercise for the body, with rest, self-reliance and relief from all excessive demands for the mind, thus bringing the elements which make for happiness into the young life.

6. Phobias; Uncontrollable Ideas and Acts

Phobias, or conditions of fear of certain places or persons, of articles of clothing, etc., are comparatively uncommon in childhood. The author has had the opportunity to observe one young child who has suffered for

years with an intense fear of time-pieces and especially of clocks. Much more common are certain uncontrollable ideas or conceptions, in close relation to uncontrollable acts. They offer a difficult puzzle when the child keeps the causative motive a secret. Several examples may be taken from Strohmeyer's excellent text. For instance, one child habitually thinks of a coffin; another is haunted by the idea that there is verdigris on its fingers; and still another, that it must stab its mother, or that it has committed some sin. A child is sometimes possessed of a doubting mania and must constantly reassure himself that he has carried out a most indifferent action correctly.

Exaggerated pedantry, excessive scrupulousness, compulsory counting, and the like, are occasionally observed as they are in the uncontrollable conception neuroses of adults. The author knows of a case in which every accidental bump or touch upon one side of the body caused the patient to give a similar bump or touch to the symmetrical point on the other side. If he did not succeed in directing the bump to the identical corresponding spot, the result was a repeated bumping to and fro in the attempt at compensation.

These phobias, or uncontrollable ideas, are not always of serious prognostic significance, even though they stand very near, at times, to the border-line of the psychoses or may even be regarded as within the pale. It is possible that suitable pedagogic training and, in older children, a conscientious self-training may bring about a recovery. In the phobias the possibility of a sexual relationship must always be considered.

7. Disturbances of Psychical Impulses

Poriomania.—The habit of running away, or poriomania causes children, and particularly boys, to leave home or school and to wander about without plan or aim. They may be found far from home, half-starved and exhausted. In some cases, the fear of punishment seems to be an exciting cause; in others a deep depression rules the event, which cannot be traced to any external factor at all. Others, again, exhibit an uncontrollable longing for the open; while occasionally it is a matter of hyperphantasy and again of mere feeble-mindedness. It is always important to decide whether the basis of the habit does not lie in an epileptic or hysterical subconsciousness. In the treatment of the condition a long continued sojourn in a suitable institution can hardly be avoided.

Masturbation.—This affliction is found in the majority of weak-minded and neuropathic children and at times to an excessive degree. It is never the cause, but always the result of the neurosis, whether it be dependent upon weakness of will or upon an excessive and premature irritability of the sexual apparatus. It is more fully discussed under Diseases of the Genito-urinary Organs.

8. Hysteria

Hysteria in children does not differ in its nature or manifestations from hysteria in the adult. Therefore the reader's attention will be called only to the few peculiarities it presents in childhood.

Manifestations of hysteria are seen very often in children and may be seen the more often if the physician schools himself to study, not only the immediate disease that he is called upon to combat, but the child, sick with the disease, as an entity. The frequency of the condition is not surprising when one considers the sensitiveness of the childish mind to suggestive impressions. The age at which hysteria is first observed corresponds with that at which the child becomes aware of his surroundings and of his own personality, which is between the second and third year. More commonly, of course, the disturbance is first noted at school age. As the patient grows older it approaches more and more closely to the qualities seen in the adult. In later years, girls are affected a little more frequently than boys. Among causal relations we very often find a distinct taint of hereditary neuropathy, while injuries of environment not infrequently have a potent influence. The directly exciting cause may be of an apparently physical quality, such as a blow, a fall, etc. In all probability there is always a physical trauma associated with the psychical injury. Very commonly the hysterical phenomena follow directly upon purely psychical traumata, such as sudden fright, fear, domestic grief, etc. Sometimes the condition may be imposed upon an actual organic disease; as, for instance, when abasia develops in connection with an angina that has confined the patient to bed; or an hysterical aphonia remains after laryngitis; or paralysis and contractures follow a slight contusion; or a spasm of the lids persists after a conjunctivitis has subsided. Some of the phenomena in this group may be mistaken for pathologic reflexes (see page 558). The fixation of disease symptoms in the subconscious memory of such children is readily understood. The excessive anxiety and the liberal attention lavished upon them by parents and attendants, in the event of illness, offers to their minds distinct advantages in contrast to the treatment they receive when they are well. Indeed there are hysterical mothers to whom the sick child, with his peculiar sort of suffering and with the general interest that he awakens, appeals more strongly than does the child who is well and who fairly sun themselves in the glory reflected from the poor little martyr in whose service they are tied hand and foot.

By way of example: A five-year-old boy was brought to the hospital suffering with abasia and a peculiar form of tonic spasm, resembling in many respects a pseudotetanus, which followed upon a febrile angina. The manner of the parents suggested a suspicion of hysteria. By the next day it was already possible, with the aid of slight faradization and the suggestion that the boy "march like a soldier," to get him on his feet; the following day he learned to climb the stairs, and then rapidly to walk and turn somersaults—when the mother promptly removed him from the hospital, very much put out to think that her child had been so harshly treated.

With the usual character of the hysterical child, prone to deceit and lying, conscious simulation may contribute occasionally to the development of the disease-picture. Self-injuries have been recorded even in childhood.

A very wide-awake hysterical boy of twelve years had vomited ascarides. Ever since this event he had from time to time produced worms which he

claimed had crawled out of his mouth or nose. His parents travelled about with him, going from physician to physician, spreading his fame, and always very much offended if anyone ventured to doubt the alleged facts. With the aid of an apothecary they had found medieval literature in which similar cases were described. The boy was brought to the hospital where he was kept under careful surveillance. Nothing happened for many days, until the child was allowed to play in the hospital garden. The following day he appeared and triumphantly produced a worm which had come out, he said, of his nose—but it happened to be an ordinary angleworm.

Not infrequently hysterical manifestations are the result of imitation. This is the history of the well-known school epidemics of chorea, tremor and similar complexes, starting from a single actual case in the small community. The patient may be even self-imitative, so that an attack of true chorea may recur as a manifestation of hysteria.

The recognition of hysteria in children may be a very difficult matter on account of the monosymptomatic type of the disease. The generally recognized stigmata of hysteria, such as pressure points, anesthetic zones, limitation of the visual field, etc., are frequently absent in children and a diagnosis must be made rather upon the strength of the general impression. The child's environment must be considered and the contrast between the objective findings and the subjective symptoms may forecast or fully determine the diagnosis. Certain forms of paralysis, as astasia and abasia, in which the motility of all the limbs and even of all the muscles is intact, arouse a suspicion of hysteria. Similarly, cuff-like circumscribed bands of anesthesia or analgesia, which, incidentally, are uncommon in childhood, are scarcely compatible with organic disease and suggest, at once, the thought of hysteria. On the other hand, a coxitis or a contracture of the elbow may successfully simulate an organic condition.

A few of the more common manifestations may be cited. Hysterical vomiting, even in small children, may make the question of adequate feeding a difficult one or may compel the parents to conform the dietary to the notions of the small tyrant. Severe scolding, or the single use of the stomach-tube, may cure such cases even in late infancy. In older children a persistent anorexia and hysterical vomiting may affect seriously the nutrition of the child. It is not always easy to exclude organic disease and the question is very often determined only by the employment of strong suggestions or sharp authority.

Attacks of abdominal pain, meteorism, pseudotumors, tachypnœa, bizarre types of respiration, stuttering, asthma, hiccough, spasms of sneezing, pollakiuria, diurnal or nocturnal enuresis, incontinence of the bowels, may all appear as manifestations of hysteria. Mutism, the complete inability to utter sounds, in which no attempt to speak is made, an inability which is sometimes the result of fright, is not uncommon. Hysterical headache may be a very obstinate symptom and is hard to distinguish from the neurasthenic form.

A diagnosis may be possible only by the experience of the prompt results of antihysteric measures and by the sudden disappearance of symp-

toms when the patient's attention is suddenly distracted by some pleasant impression.

The major convulsions of hysteria, with their well-known quality of clownism and their pronounced "attitudes passionèes," may be seen in children of ten years or even less. Generally speaking, they are of uncommon occurrence, but having once occurred they will be easily repeated during consultation; in fact, in the wards they may be provoked by suggestion. As a rule, but not invariably, they may be readily distinguished from epileptic seizures. Their differentiation has been discussed under epilepsy. In some instances, they are associated with fainting spells or cataleptic attacks. A classical case of so-called chorea magna has been observed in a girl less than six years of age. In this disorder the child stages a complete performance with theatrical precision, tears about the room, shouts in confused speeches, and afterwards remembers nothing of the event.

As additional phenomena of hysteria are to be mentioned hysterical insomnia; paroxysms of fear, which subside only if the light is left burning in the room or when the mother or nurse remains beside the bed; somnambulism, daydreaming or diurnal automatism and well-developed twilight states. Pseudologia phantastica (see page 555), often blossoms riotously in hysterical girls and not infrequently leads to false accusations of attendants, against which even the physician must be upon his guard.

The intelligence of hysterical children is usually good; not infrequently they give the impression of precociousness and their conversation turns upon subjects better suited to older persons. They observe their elders closely, even while their interest is largely or wholly centred in their own personality. The desire to attract attention and to arouse the admiration of others is always in the foreground. A shrewd hysterical girl in the Heidelberger Children's Hospital, thinking she was not receiving sufficient attention and seeking to draw observation to herself, was asked the motive of her acts and replied uniquely that she was "luring people." This desire is undoubtedly the main spring which subconsciously actuates many of the described symptoms and even inspires deeds, otherwise inexplicable, which seem to be the fruit of an evil, ugly, or unethical spirit and can hardly be associated with ordinary childish hysteria.

Diagnosis.—Without recounting details, it should be emphasized that a tentative diagnosis is extremely important to the treatment of hysteria. If the physician does not promptly establish his authority over the child in the treatment of the hysterical symptoms, the prospect of success through his efforts becomes very doubtful, and the chances of ultimate recovery rest with the next consultant, or pass to the quack, or to one of the many exploited "cures." Nevertheless a careful examination is required in every case, for the hysterical child may be subject to organic disease and error in this direction is unpardonable. In fact, it is hardly ever safe to attach the name hysteria to the diagnosis since the laity, and especially the neuropathic laity, is resentful of the term.

Treatment.—The treatment of hysteria is psychical and offers a favorable outlook. Serious cases do not recover readily in the environment of

the home. Remissions when the child is returned to it are frequent. The so-called miracle cures usually succeed best during a consultation, when the child finds himself alone with the physician. If it can be avoided, the parents should not be permitted to witness the first attempts at treatment. Still greater success is attainable if the patient is placed promptly in an institution, a move which, in itself, often suffices for the disappearance of the symptoms. At times, the very fear of being placed in an institution is enough to affect a cure. Yet even in such a place the treatment may be unsuccessful if the parents accompany the child. It is often difficult to gain this point, but the least leniency often ruins all the chances of recovery.

The actual psychical treatment employs two principal methods: (1) The method of intentional neglect. Care should be taken that the child does not injure himself, beyond which the attacks are to be entirely disregarded. The entire institutional staff, including physicians and nurses, must be fully instructed. When the child no longer has spectators for his exhibitions, they usually disappear spontaneously in a very short time.

(2) The second method of treatment is that which consists in the element of surprise (Bruns). The results must be achieved before the child has time to fit himself into his new surroundings or to take a stand against the strangers dealing with him. In fact, the entire treatment is dependent upon a single play; for if it is not possible to overawe the child or to make him conform at once to the suggestion of the physician, he will immediately realize that he has not met his master and the outlook for recovery is greatly lessened. Of course the psychology of the individual patient must be considered. A harsh command or a word may be sufficient. For instance, the seemingly paralyzed arm is raised and at the point of releasing it, the patient is told to "hold it there" (Bruns). Better results, still, may be gained with the support of some slightly painful or mystifying method of treatment. For this purpose, faradization often serves very well. If it has been already tried on the child without result, some other device, such as Bier's stasis, perspiration-producing bitter medicines, subcutaneous injection and narcosis in pseudo-surgical affections etc., give better results. In all this, less depends upon what is done than upon how it is done. The verbal suggestion connected with the treatment must be so definite that the child has no room for doubts. It is generally good policy not to laud the remedy in the presence of the child, but rather to dilate upon its good points to the parents, so that the child may receive the suggestion more or less indirectly without being able to discover the immediate intention. If it is possible to make the recovery by means of psychical influence—a more welcome result—by reference, for instance, to the mother's anticipated pleasure, to the child's early return to the home, etc., the soil for success is all the better prepared. The recovery may be reached by a suggestive therapy, operating progressively, as in the example of abasia given above. In hysterical aphonia it may be necessary, to begin with, to produce only the vowel sounds and the rest of the vocalization gradually later on.

In still other cases, influence operates but very slowly and gradual improvement is secured by the suggestive method from day to day. Where

hysterical attacks do not disappear under intentional neglect alone, it may be well to give a cold bath, an electrical treatment or some other unpleasant measure. These measures should not impress the child as punishments; they must always be suggested to him as treatment. If this is not assured, they will lose all therapeutic affect. Quite similarly, temporary isolation, the screening of the bed and the like, may be so employed. Hypnosis must be very carefully considered before resort to it is had. It should be employed only in very exceptional cases. The possibility of psychic injury lies especially close in hysteria.

With the relief of symptoms it still remains necessary to treat the hysteria itself. With slight modifications all the methods described in the treatment of neuropathy and neurasthenia are applicable to this task

IV. PSYCHOSES

1. Congenital and Early Acquired Defect Psychoses

Feeble-Mindedness; Idiocy; Imbecility; Mental Debility

Congenital or early acquired feeble-mindedness is characterized by the absence of psychic faculties or by increasing difficulty in their attainment. The intellectual impairment is usually the dominant feature, but it is frequently associated with moral defects. In the milder forms of deficiency these may fill the more conspicuous place.

Etiology.—A great variety of organic brain diseases may cause feeble-mindedness. Among them, the more important are cerebral infantile palsy, meningitis, and hydrocephalus. Very often the sequellæ of these diseases are more or less distinctly discernible, if only in the form of exaggerated reflexes, hyper- or hypotonias, deformities of the cranium, etc. In another group of idiots, the mental defect is traceable to cretinism and myxedema. This type, together with mongoloid idiocy, is discussed in another division of this work. But while the etiology of these cases is well-defined, there are many others in which the feeble-mindedness does not seem to be related to any definite physical disease. Careful histologic examinations often show retardations or perversions of development of the brain. In pronounced cases the brain proves to be below normal in weight.

Among the causes of congenital feeble-mindedness, alcoholism in the parents, syphilis, and, injuries affecting the child during its fetal development or in parturition, stand foremost Mental deficiencies, in varying degree, often appear in children of psychopathic constitution and may become a feature of any of the symptom-complexes which the diathesis presents.

Symptoms.—The so-called stigmata of degeneration (see page 503), are frequently found. The head is often microcephalic, the forehead retreating (the Aztec type); or it may present hydrocephalic and other peculiarities of form. Speech is very often entirely wanting and the child makes known its wants by inarticulate sounds or by unusually loud screams. In other cases speech may be possible but indistinct, lisping, or otherwise abnormal. Epileptic convulsions are common.

Generally two types of feeble-mindedness can be distinguished clinically: 1. The torpid idiots who, undisturbed by any changes in the world about them, lie hour after hour upon the bed staring into vacancy, or often boring the entire fist into the mouth. 2. The agile or versatile idiots who, stirred by the constant desire to move, throw themselves about on the bed without ceasing, rock to and fro incessantly, run about the room without pause, and may not be held for more than a moment by any impression. The first group is distinguished by an absolute lack of attention and the second group by the ease with which the attention is distracted.

Fig. 146.—Five-year-old versatile idiot.

The pain sense is very often dulled and a pin-prick is hardly noticed. This fact, as Thiemich has shown, is a good objective sign for the recognition of idiocy in childhood. Frequently salivation is wanting. Even though there be no paralyses, the patient is late in acquiring his static functions, in learning to hold up his head, to sit, stand and walk. Many idiots have to be fed all their lives and the training in habits of cleanliness is a matter of great difficulty. Immoderate masturbation is a common practice with idiots.

Imbecility is the term applied to children who suffer with milder degrees of feeble-mindedness. They learn to feed themselves, to walk, and to speak, and are able to master a simple trade. Very mild grades of imbecility may go unrecognized for a long time. The parents often prefer to keep the sad truth hidden even from themselves as long as possible. The weak reasoning powers of the child are the less noticeable if the memory is good, permitting the child a memorized reproduction of his lessons. The memory, indeed, may be excessively developed in some one direction, say for numbers, music, etc., so that the child may be looked upon, for a time, as a prodigy. The essential criterion of mental ability, however, is the power to combine words with ideas, to correlate conceptions and to apply them. Thus it is often shown that in such children the simplest conceptions of space, time, relationship and cause are wanting; as are also, of course, the higher abstract conceptions of gratitude, truthfulness, envy, etc. Scientific tests of intelligence, chiefly by the methods of Ziehen and Binet take all these things into consideration. By the aid of colored skeins, picture

books, toys, short stories, etc., this is not a difficult matter, after the child has conquered his first timidity. With children who tire easily, the tests may not be completed at one sitting. Into their further detail we cannot enter here.

The term mental debility covers the mildest grades of feeble-mindedness. Children of this type are able to keep up with their school work in a measure and by one means or another they may even gain the higher classes in High School, beyond which they seldom go. Not infrequently their ethical defects are more pronounced than their intellectual weaknesses. Some of the cases, described as psychopathically weak, belong in this group. The primary lack of esthetic sense and moral principle, when it accompanies feeble-mindedness, is called moral insanity.

The moral defect, however, can be charged to intellectual weakness only when physical and mental impairment and diminished reasoning power can be determined; when evil associations, lack of proper home care, and other similar factors can be excluded; and when neither punishment nor rewards have any effect (Ziehen).

Diagnosis.—In the young infant the awakening mind is heralded by glances directed to bright objects, or to the mother, and later by the reaching out of the hand toward them. If these indications do not appear, there is good reason to suspect a low degree of intelligence, provided, of course, that the sense organs are normally developed. Later, the lowered reaction to pain is evident. The delayed development of speech, as late even as the third year, may by no means be identified with mental weakness, especially where the first child of the family is concerned and evidence obtains that he understands spoken words. Deaf-mutes are often mistaken for idiots and their mental development is neglected in a most irresponsible manner. Special caution must be given that myxedema be not overlooked, since good results may be achieved by its proper treatment.

Treatment.—Treatment directed against the cause of the mental condition is possible of success only in myxedema, cretinism and, possibly, congenital lues. Beyond this, thoroughly individualized pedagogic treatment which differs broadly, alike in its aims and its methods, from the ideals of the general school, is the only thing that will really help these children. The results which may be obtained by good institutional training are sometimes very satisfactory, although the high expectations of the parents are often grievously disappointed. The earlier the child is placed in such an institution, and this especially if the home environment is not all that it might be, the more beneficial is the treatment of the child. The expense of maintaining markedly deficient children and those who are especially difficult to train, may be very high. Special schools, rapidly becoming more numerous, accomplish very brilliant results. It is generally possible to train the majority of imbeciles to a trade.

2. Acquired Defect Psychoses

Under this heading may be classed the amaurotic familial idiocies (page 523), diffuse cerebral sclerosis (page 510), and some forms of brain

syphilis and of epilepsy. Two specific mental diseases of this group are important.

(a) **Progressive Paralysis.**—This disease has been described very often in childhood as the result of congenital lues. Its onset occurs during the second decade. The first physical disturbances are those of speech, syllabication, bradylalia, etc.; fixation of the pupils; loss of facial expression; tremor of the lips; and, later, inability to walk, intention tremor and cachexia. The patellar reflexes are present and often exaggerated. Tabetic symptoms, optic atrophy, lancinating pains, etc., are very rarely observed simultaneously. The psychic disturbances consist in a progressive loss of the mental faculties, which may lead to complete dementia. They often play a significant part in the disease-picture from the very beginning. The patient usually feels perfectly well. Hallucinations and delusions of grandeur are observed only occasionally; while the so-called paralytic attacks, with dizziness, unconsciousness, epileptoid convulsions, or headache, are common. The disease generally lasts for three or four years.

In its treatment nucleinic acid may be considered.

Hebephrenia; Catatonia or Dementia Præcox

The beginning of these psychoses appears, as a rule, during puberty. The disease may occasionally develop before the tenth year. At the onset, the condition is readily mistaken for neurasthenia or even hysteria. For detailed descriptions, the reader must be referred to text-books of psychiatry.

Psychoses Without Defects of Intelligence

Concerning these psychoses, it may be said, merely, that melancholia and mania occur in children; that acute hallucinatory insanity (amentia), has been observed; while chronic paranoia is extremely uncommon. The manifestations of each of these diseases resemble very closely the course they take in the adult.

VIII.

THE ACUTE INFECTIOUS DISEASES

BY

E. FEER,
Zurich.

REVISED AND EDITED BY

E. C. FLEISCHNER, M.D.,
Clinical Professor of Pediatrics, University of California

and

K. F. MEYER, M.D.,
Professor of Research Medicine, University of California.

GENERAL CONSIDERATION

Diseases caused by micro-organisms, frequently associated with constitutional symptoms and directly or indirectly transmissible from person to person, play a prominent part in general mortality and morbidity. The rôle which certain of the more important of these diseases take is shown in the following table:

Annual Mortality (after Rathman)

Diseases	1891-1900	1901-1903	1910
Diphtheria	30,400	12,700	9,700
Pertussis	14,000	11,900	9,300
Measles	8,400	8,400	7,300
Scarlet Fever	8,000	9,700	5,500

These comparative statistics, are, of course, not fixed. In the past decade, the deaths due to diphtheria have decreased materially, although during the last few years they have again increased in Germany.

The above named infectious diseases, with a number of others, show a special predilection for childhood, so that they have come to be known as *Diseases of Childhood.* Reliable morbidity statistics of entire countries are not obtainable, however, at the present time but mortality statistics which, taken as a whole, may be said to run parallel to them give valuable information. Thus, in Bavaria, from 1893 to 1902; out of 100,000 deaths among the male population, the following table (after Prinzing), shows those, that are attributable to each of the four principal infectious diseases at various ages of life.

Diseases	Ages					
	0–1	1–2	2–5	5–10	10–20	20–30
Scarlet Fever	27	52	45	19	5.9	0.6
Measles	319	435	73	13	1.8	0.2
Diphtheria and Croup	138	401	277	77	15.8	0.7
Pertussis	674	295	38	3.9	0.2	0.0

From this summary it will be seen, that the significance of pertussis and measles sinks to an unimportant minimum after the fifth year and that scarlet fever and diphtheria fail of significance after the tenth year.

Individually considered, these four important infectious diseases show peculiar differences as to the ages at which they prevail. This may be clearly seen in a graphic presentation of their relative occurrence during the early years of life, taken from the excellent statistics of Basel, Switzerland. A comparison may be made of the details in each individual disease.

Pathogenesis.—It must be assumed that the basic theory of immunity is thoroughly comprehended. The understanding of this theory is of great significance in the infectious diseases of childhood, and particularly with reference to diphtheria and its serum therapy. It does not present any differences in principle as applied to childhood from those which obtain in the adult. It may be briefly stated, as a matter of general acceptance, that during the period of incubation of many infectious diseases, antibodies, specific to the infective organism or its toxins, are formed. It is held, further, as von Pirquet and Schick have practically proven, that the disease begins when the formation of antibodies is complete, representing, indeed, the reaction between the antibodies and the specific toxins. Many acute exanthemata, such as variola, varicella and measles, closely resemble the serum disease, which follows a first injection or an initial vaccination. A hypersensitivity is observed only upon reinfection; as, for instance, in hemorrhagic small-pox, a virulent form which occurs almost invariably in those vaccinated persons who exceptionally succumb to the disease. Moro accounts for the rash, not as the result of the action of agglutinins, but as a manifestation of a specific hypersensitivity, which is analogous to the serum and tuberculin exanthems.

The transmission of the acute infectious diseases is generally a matter of contagion; that is, infectious material developed in the one patient passes directly to another, in whom the disease is thus produced. In several of these diseases transmission is chiefly through the medium of the air most frequently by so-called *droplet infection*, which means that small particles of mucus containing the specific organism are discharged by the act of sneezing or coughing and are then inspired or drawn into the mouth of neighboring persons. Infection by this droplet method occurs most readily and frequently in diseases of the respiratory passages, where the specific microorganisms abound. It is a common experience in measles, pertussis, and influenzal disorders and an occasional one in varicella. No doubt of this method of transmission existed even prior to our present advanced knowl-

edge of bacteriologic causes. These diseases have these facts in common—that infection by aspiration is almost without exception a matter of direct conveyance from one person to another; and that contagion carried by means of infected objects or by healthy persons in immediate contact with the patient is exceptionally rare, since the specific organisms soon perish, when they are removed from the human body. It is easy to see, therefore, that the origin of the infection in measles, whooping-cough and influenzal disorders is readily determined.

This droplet infection may be an active agency in many other infectious diseases, but it is evident that it will operate only when the infective organisms are in the mouth or the upper air passages. Attention has been called to this possible means of transmission in scarlet fever and diphtheria, which are among the most important of the diseases of childhood, but as an actual fact it is probably a quite uncommon thing. In these diseases *contact infection* plays a most active part. The specific organisms are contained in the secretions of the patient's mouth and nose and are transferred to others by direct contact. The germs, therefore, usually gain admission through the mouth and less often through the nose. In these two diseases, as in many others, indirect, as well as direct contact, is to be considered as a means of conveyance. Thus

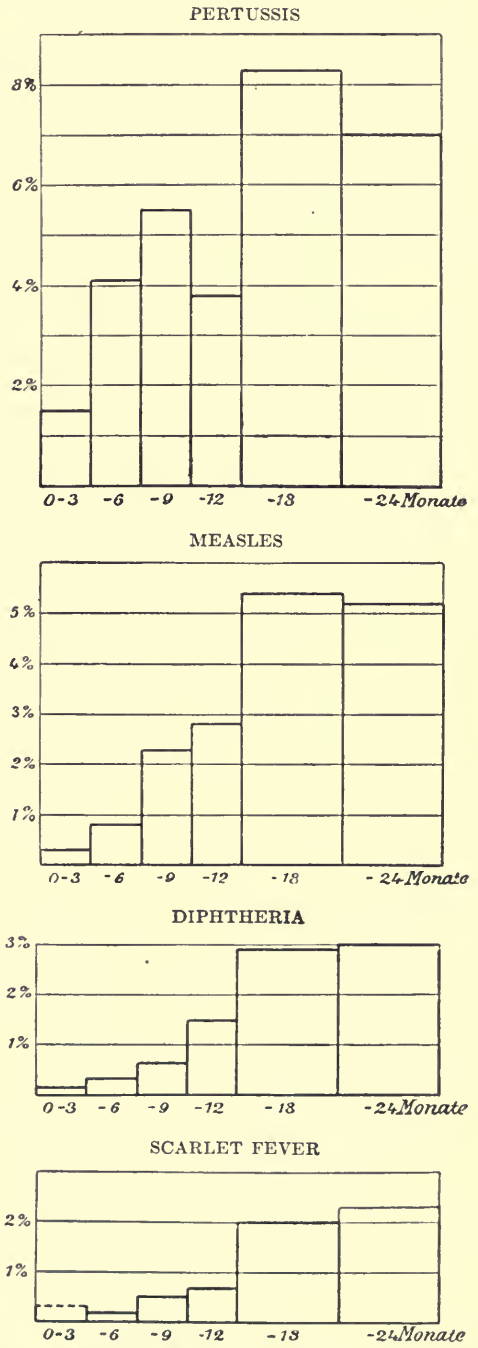


FIG. 147.—Predisposition of age to various infectious diseases during first twenty-four months.

the micro-organisms may be spread to healthy persons, by handkerchiefs, vomitus, food-stuffs, etc., and thence may be distributed to those, who have not been in direct touch with the patient. This indirect contact becomes significant only when the germs are able to survive for a variable period outside of the human body. This is actually true in scarlet fever, diphtheria, typhoid fever, small-pox, German measles, mumps, etc. It accounts for the fact, that in these diseases the method of transmission and the source of infection often remain wholly obscure. In scarlet fever, particularly, the specific virus lives for a very long while after removal from the infected body.

The question of *individual predisposition*, upon which the development of each and every case depends, is still in many respects obscure, on account of the fact, that the significance of many influencing elements, such as the virulence of the specific organism and the opportunities of infection, is still unknown.

The relative predisposition to disease at certain ages, already noted, depends mainly upon the fact that such disease is commonly incurred earlier in life. This fact comes out very clearly in the case of measles. One attack of the disease almost invariably immunizes the child for the remainder of his life. A second attack of measles is extremely rare. Since the susceptibility to measles is very general by the end of the first year and since almost all children of the present day are exposed to contagion at some time or other during their early childhood, measles has come to be regarded, under ordinary circumstances, as a disease of childhood, despite the fact, that adults are naturally as susceptible to it as are children.

Many other infectious diseases give a certain degree of immunity after one attack. We possess, however, very uncertain knowledge of such immunity. Not alone in measles, but also in small-pox, this acquired immunity is undoubtedly of great importance, but it is not so definitely demonstrable in the latter disease. The measure of immunity ordinarily accepted in such diseases as pertussis and scarlet fever is certainly misunderstood or over-estimated, a fact to which Gottstein very justly calls attention. In diphtheria acquired immunity evidently persists for but a very short time.

Granting that survival from single attacks of certain infectious diseases confers a lasting immunity, it still remains true, that childhood has a special predisposition to a number of these disorders. Many persons, either in later childhood or adult life, who have never had these diseases do not become ill even when exposed to infection, to which many younger individuals will succumb. Chicken-pox is an illustration in point.

Again, most people have measles, sooner or later, and many have pertussis, whereas a large number never have scarlet fever and diphtheria. Gottstein has attempted to express in numerical terms the disposition to particular diseases in various epidemics by means of the so-called contagion index. He has estimated the liability to measles at 95 per cent.; to scarlet fever at 40 per cent.; and to diphtheria at 10 per cent. to 15 per cent. These figures, of course, are of only relative value, but nevertheless they show an actual difference in disposition to the three diseases named. The

explain why measles epidemics spread rapidly, while scarlet fever epidemics are of slower development and diphtheria epidemics are very slow to spread.

The reason for the greater predisposition of childhood to certain diseases is still far from clear. As compared with that of adults it may be due in part to the lower resistance and in part to the greater permeability of the mucous membranes, which serve as ports of entry for micro-organisms. Both mechanical and morphological conditions may play some part in explanation of the fact, that the adult mucous membranes, with their denser epithelium, and particularly those of the nose, the tonsils and the pharynx, present a more effectual barrier to the invaders. Neither inborn nor acquired has the child any very extensive mechanism of defense by way of bactericidal or other protective bodies. In many cases the chances of infection are favored by lesions of the mucous membranes incident to catarrhal or other inflammations. This accounts for the prevalence of diphtheria and croup in those seasons in which diseases of the respiratory organs prevail, and it also suggests the reason for the predisposition of children sick with measles to secondary infection with croup. The tendency of patients with a lymphatic diathesis to diphtheria, scarlet fever, and other infectious diseases is the result of an evidently lowered resistance, due to the constitutional disturbance, to the organic changes, such as adenoid hypertrophy and the chronic pharyngitis it favors, which prepare soil for the germ invasion.

It must be said, however, that there are infectious diseases to which youthful predisposition is so strong, that the individual is attacked whether the local mucous membranes are normal or impaired. This is true of measles and influenza. On the other hand the lesions which the infectious disease produces in the mucous membranes very greatly favor the occurrence of secondary infections caused chiefly by pyogenic organisms, of the strepto- staphylo- or pneumococcic forms. These secondary infections determine the course of the disease in a very large number of infectious disorders and are the cause of the major number of resulting deaths.

While infancy, as compared with later childhood, shows a lesser predisposition to the true acute infectious diseases, at no age is the tendency to local and general infection with pyogenic organisms so great. This is especially true of the new-born and of infants during the first few months. It is to be explained by the tenderness of the skin and mucous membranes, by the lessening of immunity resulting from disturbances of nutrition in artificially-fed infants, and by the frequent existence of the exudative diathesis. All of these factors may be concurrent and may tend to increase the liability to infection and to decrease the resistance of the young infant. These facts are more fully discussed under the chapters upon Diseases of the New-born and upon General Sepsis.

The origin of epidemics is not very fully understood. It is clear in measles alone. In the case of this disease an epidemic is usually lighted up at regular intervals of a few years, with the accumulation, as it were, of a suf-

ficient number of children of susceptible age. In other diseases the causes of recrudescence are generally obscure. In many epidemics an exaggerated virulence of the provocative organism must be admitted. Illustration of this factor is found in the serious epidemic of scarlet fever, which occurred in England in the middle of the last century. This outbreak was so severe as to suggest the special predisposition of the Anglo-Saxon race, but it has been necessary to abandon this theory in view of the extraordinary recession of the disease in England during the last twenty years.

In diphtheria it is possible to test the question of virulence of the organisms on guinea pigs. It is not uncommon to find a very high degree of virulence in some severe epidemics or, instead, severe single cases without any demonstrable regularity in their occurrence. General experience goes to show, that in serious epidemics and in groups of especially severe cases a larger percentage of adults than usual will be found to be affected.

The opportunities of infection are very much greater in children than in adults. They are fostered by the intimate association of children in the home and, in general, by their gatherings in day nurseries, schools, etc. The transmission of contagion is greatly encouraged among children by their habitual carelessness in the disposal of their secretions and excretions. They are apt to soil face and hands and clothing with mucous discharges from the nose and mouth and thus the infected material is spread directly or indirectly. The communistic use of handkerchiefs and the close contact of children with the floors in creeping and playing add to the ample opportunities of infection. Such diseases as diphtheria, which are readily spread by direct contact, are encouraged by the uncleanliness of the sick or their surroundings. Dirt-conveyed diseases are more common among those of meagre circumstances than they are among the scrupulously clean. Young infants enjoy a comparative protection from these numerous opportunities of infection, since they move about less freely, are relatively isolated, and so come less directly in contact with other children.

The importance of isolation, as a means of prevention, is, of course, to be emphasized in those diseases, which spread directly from one person to another. General experience shows, that among those of inferior social condition, contagion will involve entire families in their earlier years. Children living in crowded quarters have measles during their early childhood, especially when they are grouped in day nurseries, kindergartens, etc. Among the middle classes the disease is apt to occur at school age, while the children of the wealthy, reared in careful seclusion, often escape attack until maturity.

Formerly, the occasional combinations of different infectious diseases, especially the acute exanthemata, aroused particular attention. Since, however, we have learned that each of these diseases is the product of a specific organism, the association of two or more types of infection is no longer deemed remarkable and is of interest only as it makes a diagnosis more difficult and prolongs the course of the illness. The combination of measles and scarlet fever, of measles or scarlet fever with diphtheria, or of

any of these diseases with chicken-pox, is not unusual and may even become common, if the possibilities of hospital isolation are not of the most effective order.

As a general rule the combination of any of these diseases does not effect their individual behavior to any great extent. When two severe infections are combined the prognosis is, of course, less favorable. At times, the incubation period of one disease, as in measles, is extended by the intercurrency of some other infectious disorder. The writer has seen the onset of chicken-pox interrupted by the development of pneumonia and its course continued after the pneumonia crisis has passed. The addition of a second infection to measles may readily aggravate the attack and lead to the most dangerous results. This is especially true of the secondary appearance of diphtheria, the spread of the latter into the larynx and the trachea in such an event being encouraged and often with fatal consequences.

The measles patient shows a distinct want of resistance to tuberculosis. Again, the appearance of measles during pertussis is a serious matter, since not infrequently, it leads to grave pulmonary complications. The same tendency is frequently noticed if measles is complicated with scarlet fever.

Convalescence from an infectious disease is variable in different individuals. Recovery is usually complete. In certain cases the attack may be followed by persistent and often obstinate sequelæ. Catarrh frequently remains after pertussis or measles, while deafness, heart lesions, and nephritis follow scarlet fever. Sometimes an exudative diathesis may develop, especially after measles or chicken-pox. Tuberculosis is frequently activated by measles.

On the other hand, it is not uncommon to find, that the general health of the child improves after an infectious disease. The author has observed such improvement very distinctly in cases of pertussis.

In the prognosis of these disorders a large number of factors must be taken into account, which have little or no significance in the adult. The better prognosis in the case of breast-fed children over the artificially-fed is always recognized. The influence of care and environment is enormous. The outcome more often depends upon these factors than upon the nature of the disease. Particularly do they govern the result in those disorders in which, as in measles and pertussis, secondary infections are of decisive influence. Rickets, the prevailing malady of early life among the poor, also has a marked effect upon the course of contagious disease. Special significance must be given to certain diatheses. Particularly unfavorable is the presence of a marked exudative or lymphatic tendency. It is most felt in diphtheria and scarlet fever. Fat, pasty children, with eczema, often succumb with unexpected rapidity by the second or third day even when the disease itself is not of severe grade. Neuropathic patients are usually affected with undue intensity and together with spasmophilics, suffer severely from pertussis.

Prophylaxis presents numerous problems. In diseases, such as rubella, which are always mild, it may be practically disregarded. In infections which are difficult of avoidance, as measles and pertussis, but which gener-

ally pursue a mild course in older children, prophylactic measures should safeguard the first three or four years of life. Since few permanently escape measles, pertussis or chicken-pox, and since they are apt to be more severe in adult life, it is hardly desirable to safeguard robust children in later childhood from these diseases too scrupulously.

Everything should be done, however, to avoid such serious infectious diseases as scarlet fever and diphtheria, the outcome of which we can never be assured. To this end, the most important measure is the *absolute isolation* of the patient and his nurse, a measure possible in but very few homes. In view of this limitation it is urgently necessary, that all cases of scarlet fever and diphtheria occurring in homes that do not permit such strict isolation, should be removed to a hospital. In order to make this rule broader of application, it is very desirable, that the private physician of the larger cities be permitted to treat his cases in the hospital.

To fully accomplish the ends of isolation the attendant should receive full instructions directly from the physician and rigorously observe them. As details worthy of mention, the provision of separate utensils, the disinfection of the hands, and the use, if possible, of a separate toilet for the sick room suggest themselves. The physician himself should wear a gown when entering the room of a patient with diphtheria or scarlet fever and should wash his hands and face upon leaving. The excreta must be disposed of with great care and especially in typhoid fever should be first disinfected. The same precautions should be observed in handling the secretions from the mouth and nose in diphtheria and scarlet fever. Effective isolation throughout the course of a case of infectious disease in the home, pains being taken to see that infected utensils are not removed from the room and that random intercourse between the nursing attendant and the family is not allowed, is of far more moment than later disinfection.

Nevertheless, after the patient has recovered, a thorough disinfection of the sick room and of all articles used in it is in order. The room should be fumigated with formaldehyde; the clothing and the bedding sterilized with steam; and books and other articles washed with a solution of bichloride of mercury or phenol. Too much must not be expected from such measures. Every now and then new cases will develop, in spite of the exercise of the greatest care; and, conversely, no spread of the disease may occur, though all the precepts of cleanliness have been neglected.

Again, of far more importance are the principles of prevention addressed to increasing the resistance of the child to infectious disease; especially in the way of rational living and a suitable dietary. Aside from the general promotion of physical vigor, care should be taken to avoid rickets and tuberculosis and to meet an exudative diathesis by proper treatment. While isolation is essential in all the more important infectious diseases, subsequent disinfection is especially important in scarlet fever and diphtheria, whereas after measles and pertussis it is entirely uncalled for.

The physician has certain duties to perform as the *guardian of the public health*, which for the protection of the schools are specifically prescribed by law in many states.

Children suffering from any contagious disease, though it be but German measles, must be kept out of school during the course of their illness. Unless superseded by state or local law, the following precautionary measures may be recommended. Children should not attend school until three weeks have elapsed from the onset of measles, reckoning the period from the appearance of the rash. Following pertussis, they may be readmitted when the cough has entirely disappeared. In the event of diphtheria they should be kept away from other children for at least fourteen days after the membrane has disappeared. Convalescents from diphtheria should not be allowed to come in contact with healthy individuals until two negative cultures have been obtained from the nose and throat at five day intervals. The Health Board regulations in different states are unfortunately extremely variable on this point. In scarlet fever they must not be allowed to reënter earlier than eight weeks after the outbreak of the disease; and certainly not until they have completely recovered from such complications as otitis media, etc. After this disease, the disinfection of the clothing and the repeated bathing of the body are very important.

If a case of contagious disease appears in a family, the well members must be excluded from school for two weeks following an outbreak of diphtheria and for three weeks after the onset of scarlet fever. This depends upon local health regulations, but in a general way it may be said, that contacts in diphtheria should be considered potential carriers until by culture they are proven to be otherwise. If the patient has been promptly removed from the home the other children may be readmitted to school eight days subsequent to the invasion of diphtheria and fourteen days following the removal of a case of scarlet fever. If measles invades a family, the children, who have not had the disease and are less than six years of age should be kept away from other small children for the space of three weeks. The unaffected members of a family in which pertussis has appeared should neither be permitted to attend kindergarten nor be placed in any day nurseries during the entire duration of the cough of the sick child. Similarly, the older children in such a family should be kept out of school.

The State which, by strenuous preventive measures has accomplished wonders against small-pox, cholera and plague, should make, in some localities, an equally strong effort to suppress other prevalent infectious diseases. This could be done by the free treatment in public hospitals of diphtheria and scarlet fever, and, perhaps, of measles and pertussis as well, by the free transportation of such patients in special railway cars and, in cities, in special ambulances, and by the creation of special playgrounds for those who have whooping-cough, etc.

SCARLET FEVER

Scarlet fever is a specific exanthematous infectious disease, characterized by angina and erythematous rash and a tendency to peculiar sequelæ. The disease was accurately described for the first time by Sydenham of London, at the end of the seventeenth century, who sharply differentiated it from other exanthemata.

Etiology.—Its causative organism is still unknown. By some observers a specific streptococcus, persistently found in necrotic and purulent tissues, often in the blood itself, and appearing even in mild cases, is considered the essential cause; but this relationship has not been accepted by the majority of clinicians. Definite etiologic proof has never been established and many arguments, among them the immunity acquired after a single attack has been urged against it.

The plea that the streptococcus is not found in very recent cases does not always hold. In a fulminant case, seen by the writer, which terminated fatally in sixteen hours, the apparently little changed tonsils were found to be internally necrotic and crowded with streptococci. Most authors unequivocally agree, that the course of the disease and its complications are largely governed by this streptococcic infection. True, a number of observers have found that cases both with and without complications have an equal number of streptococci in the blood.

The studies of Bliss and Tunnicliff have shown, that the streptococcus hemolyticus isolated from the throats of patients in the acute stage of scarlet fever forms a distinct biologic group apparently peculiar to this disease. An immune serum produced with such hemolytic streptococci, protected mice against cultures isolated from scarlet fever patients, but not against hemolytic streptococci from other sources such as erysipelas, otitis media and influenza. Similiar results have been obtained by means of agglutination tests.

Mode of Infection.—Scarlet fever very often spreads from the ill to the well. It is usually contagious at the onset and, in a measure, even a few days before the onset of the disease. It is quite certain, that the contagious principle exists from the first day of the manifestations of the disease and with gradually decreasing activity persists for some weeks. Transmission is especially frequent from light, ambulant, and often unrecognized cases, in which, sometimes, an angina without exanthem appears. It is usually a matter of contact infection, unless the patient coughs directly in the face of the exposed child. We do not know whether the contagium is to be traced to the particles of the skin or, as seems the more probable, is carried by the secretions of the mouth, by the pus of an otitis media, or by the desquamated skin itself infected from these sources. Children vaccinated by the subcutaneous injection of the mouth secretions of fresh cases have developed scarlet fever, which tends to show that these secretions, at least during the earlier stages of the disease, contain the infective organism. The long duration of the contagious period is clearly shown by the fact, that after full recovery, completed desquamation, and careful disinfection the discharged patient may, nevertheless, infect newly-exposed or previously absent members of the family. Not infrequently, transmission takes place through the medium of healthy people, utensils, articles of clothing, etc., upon which the virus may remain active for several months or even for a year or two. In sporadic cases the source of the infection may hardly ever be traced, but during epidemics a direct and apparent contagion is more common.

The port of entry of the virus cannot be definitely established as long as the organism itself is unknown. Numerous observations, however, appear to indicate, that entry is usually effected in the pharynx. This view is apparently sustained by cases in which scarlet fever has developed through wounds, when angina, in other instances always present, is absent. In this event a dirty discharge issues from the wound and the exanthem begins on the neighboring surface; suggesting that in such exceptional cases the infective agent penetrates the skin at the point of injury—a result observed in a tracheotomy wound, a scratched chicken-pox pustule, etc. Cases of alleged scarlet fever after burns must be considered in the author's judgment, as in part at least, toxic erythemata.

The Incubation Period.—This period usually covers from three to five days. In some instances, and particularly in scarlet fever originating in wounds, the incubation may not exceed twenty-four hours.

Children between the ages of three and six years are most frequently affected. A few cases are seen in the second half of infancy. It is very rare from the third to the sixth month. It is very exceptional in the new-born and probably occurs only when the mother has the disease. Cases reported, sometimes with comparative frequency in the new-born, (see Tables, page 573), are probably confused with a severe grade of erythema neonatorum. The disease is not uncommon up to the twentieth, or even the thirtieth year, after which its tendency is to entire disappearance. One attack confers very lasting immunity, although second attacks are by no means very rare and are certainly of more common occurrence than in measles.

Predisposition is generally less marked than in measles or pertussis, so that only a moderate percentage of exposed and non-immunized persons take the disease. This percentage, in fact, does not exceed twenty even in serious epidemics. In large families, even without a resort to prophylactic measures, only a single case may occur; while in measles, and usually in pertussis, all those who have not had the disease commonly contract it.

One peculiarity of scarlet fever lies in the fact, that the disease may occur sporadically for many years, with occasional increase in the number of cases, but without its entire disappearance at any time. Furthermore, it occurs in large epidemics at very irregular intervals of from five to ten years. These epidemics grow slowly and disappear as gradually, without at any time reaching such great proportions as do the epidemics of measles. (See curve on page 600.) Another peculiar characteristic of scarlet fever is, that the disease may be extraordinarily benign for years or even decades and then suddenly and inexplicably assume a frightful and fatal virulence.

Scarlet fever is found throughout Europe and North America, but seems to be uncommon in the Far East.

Pathologic Anatomy.—Even though the angina of scarlet fever often superficially resembles diphtheria, the disease process is of a much more phlegmonous type, is more penetrative, and shows a greater tendency to purulent degeneration than does the diphtheritic form. With this more severe angina an inflammatory necrosis of the affected tissue develops, with a diffuse superficial and deep exudate of the mucous membrane, which soon

becomes filled with streptococci. This so-called coagulation necrosis very frequently penetrates deeply and not only destroys the visible pharyngeal structures but also infects the regional nodes, which suffer in part a purulent degeneration. The streptococci may also cause a purulent phlegmon in the mediastinum, in the middle ear, or in certain joints, due in part to the spread of the virus of the scarlet fever and in part to an independent production of sepsis. At autopsy, in persons dying of scarlet fever, an hyperplasia of the entire lymphoid tissue is frequently found.

Scarlet fever presents so extremely invariable a picture of symptoms that it is quite impossible to formulate a description, that will apply to all cases. To make the matter clearer it seems best to describe, first, some average forms of moderate intensity and then to detail the numerous variations and complications, which present themselves.

The Usual Disease-picture.—After an incubation period of from three to five days, during which there are no symptoms, the onset occurs suddenly with vomiting, high fever, and so severe a disturbance of the child's general health, that he goes to bed voluntarily. Older children complain of headache and sore throat and younger ones appear apathetic, restless, and may be delirious. Occasionally convulsions occur.

The thermometer registers a temperature of 39°-41° C. (102°-104° F.). No organic lesion is demonstrable. The throat shows a marked injection, often sharply circumscribed at the hard palate. The submaxillary lymph nodes are slightly enlarged and painful and the inguinal nodes also may be affected. Even though the intense reddening of the throat may arouse suspicion of scarlet fever in the mind of the experienced observer, the diagnosis cannot be definitely made until the rash appears.

The eruption usually develops in from twelve to twenty-four hours after the onset of symptoms. It usually appears first on the neck, the breast or the back, but in exceptional cases it may be first seen on the extremities. It spreads rapidly to the entire body, commonly extending to the thighs and the arms and then to the forearm and hands and to the legs and feet. In the course of about two days the rash is fully developed and covers the entire body with the exception of the face, which usually shows but a congestive reddening of the cheeks. The nose, upper lip and chin remain entirely clear. This pale triangle of which the chin forms the base, stands in vivid contrast to the bright red skin of the rest of the body and forms a characteristic feature of scarlet fever.

The rash consists, at first, of very small, discrete bright red spots, between which normal skin is still discernible. Successive crops of spots become distributed between those of first appearance, so that in one or two days a confluent exanthem is produced, which takes on a brighter and brighter hue. The initial form of the small spots can be made out in but a few places, as the inner surface of the thigh and the back of the hands. The rash disappears completely upon pressure, and upon its removal the individual red spots return first but are immediately followed by the reddening of the entire skin.

After the eruption has existed for a short time the skin from which the redness is removed by pressure appears yellowish. This is especially observed in the skin of the abdomen. As a whole, the skin is somewhat

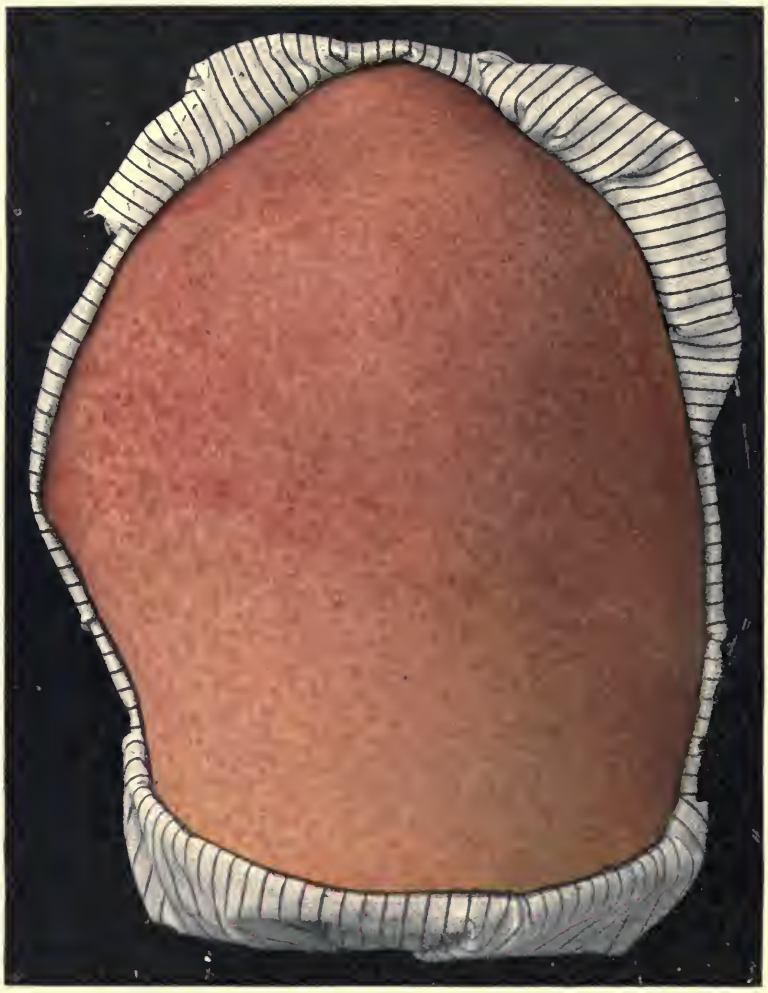


Fig. 148.—Exanthem of scarlet fever on the shoulder, in part as scarlatina miliaris. From wax model (Dr. Henning) in the Vienna Children's Hospital, Prof. von Pirquet.

swollen. With the continued spread of the exanthem and with the constant appearance of new spots the bright red eruption becomes darker and darker, so that the patient's skin finally appears almost purple.

Each individual spot is initially a bright red, smooth, rounded area hardly a millimeter in diameter. As it develops, this spot becomes slightly raised, so that in a cross-light the skin has the appearance of fine-grained

leather and feels rough to the touch. This follicular swelling is especially prominent over the back of the hands and feet, the forearm and legs, since the eruption is more elevated here than in other parts. At times the spots become slightly vesicular, with a cloudy whitish content, the vesicles desquamating early at their apices. This form is known as scarlatina miliaris, but is an eruptive variation without any serious significance. The formation, however, of unusually large elevated spots is rather indicative of a severe type of the disease. Occasionally very small hemorrhages appear over the flexor surfaces of the elbows, in the axillæ, or in other parts where the skin is rubbed or scratched by the clothing. By applying a tourniquet above the elbow for a few minutes petechial hemorrhages will invariably occur below the joint within a short time (Rumpel–Leede). If the skin is scratched with the finger-nails a white line appears as the result of a

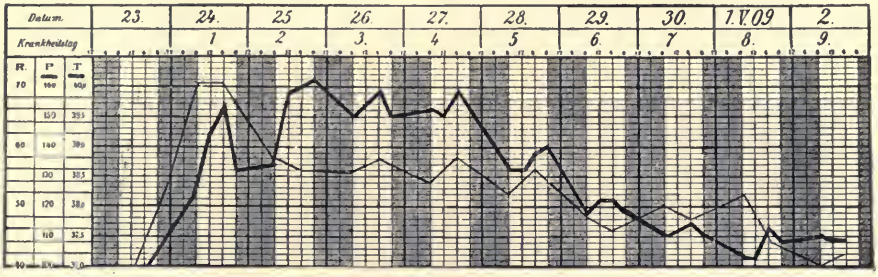


FIG. 149.—Typical curve of scarlet fever. Six-year-old girl.

vasomotor spasm (*raie blanche*). The eruption often causes annoying itching and children will scratch.

The exanthem usually reaches its height in from three to five days. It gradually fades in the same order in which it appears. By the fourth to the seventh day, or by the beginning of the second week, the rash has usually disappeared entirely, often leaving a little roughness and slightly increased pigmentation At this stage *desquamation* of the skin commences. It may begin, and often does, before the rash has entirely disappeared but, as a rule the scaling begins during the second week or even later. It is first seen over those parts of the body, as the neck and axilla, where the skin is the thinnest. Upon the face the detritus consists of extremely fine scales. In other parts small circular areas of epidermis are sloughed and the entire skin is then shed in small pieces (see Fig. 151). The more severe the eruption, the earlier does desquamation occur. The skin sloughs are largest where the skin is thickest. Thus the largest pieces separate from the palms of the hands and the soles of the feet, where desquamation is long postponed, often until the sixth to the eighth week from the onset of the disease. Usually the growth of the finger-nails is impaired, particularly on the thumb. A ridge or groove is formed at the root of the nail. This occurs at the outbreak of the disease, but the deformity appears from beneath the skin-fold after some six weeks and grows outward to the end

of the sixth month. Similar changes of the nails are seen in many other acute diseases, but never so distinctly as they are in scarlet fever.

The *mouth* and the *throat* also present characteristic symptoms. A redness and swelling of the tonsils, the soft palate, and the pharynx, which appear on the first day, become more marked during succeeding days and cause more or less difficulty in swallowing, and the consequent refusal of food. Children of sufficiently advanced years complain of sore throat. The regional mucous membranes show a bright flaming red, of a degree of color hardly ever seen in simple angina. The swollen tonsils often meet in the median line and yellow masses protrude from the lacunæ. The submaxillary nodes are enlarged and painful. The angina reaches its height

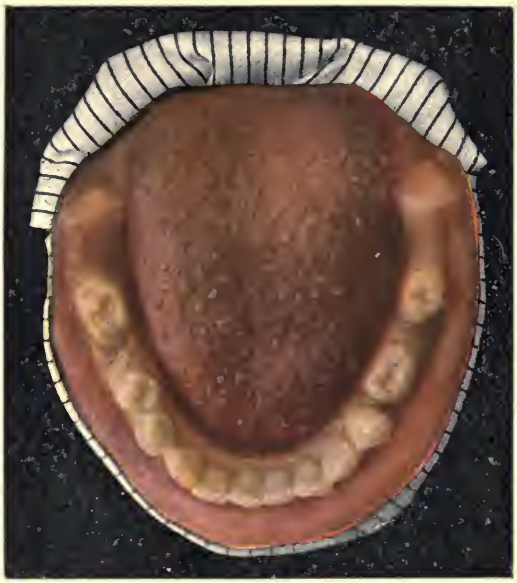


Fig. 150.—Scarlet fever tongue (strawberry), marked swelling of the papillæ.

in three to five days, after which it gradually subsides. Just at this point a dreaded necrotic angina is apt to develop (see below).

During the first few days the tongue is heavily coated, but soon it becomes clean and on the third or fourth day shows a clean, bright red surface upon which its markedly enlarged papillæ stand out distinctly, giving the so-called strawberry tongue. This characteristic feature, however, may be absent.

The *fever* during the first three to five days runs persistently between 39°-40° C. (102°-104° F.), or even higher. It does not show any marked remissions and with the full development of the exanthem may go even higher. The intensity of the fever is dependent more upon the severity of the angina than it is upon the severity of the skin eruption. A lytic, step-by-step fall of the temperature occurs, the normal point being reached by

the end of the first or the beginning of the second week, as a rule, in uncomplicated cases (see Figure 149). Not infrequently, however, an unexplained fever may continue during the entire second week and after the rash has disappeared, without any evident cause or complication (Fig. 152).

The *pulse* is markedly increased from the onset of the disease and, in children, to a rate that is hyper-proportional to the rise of temperature. In young patients with a temperature of 40° C. (104° F.), the pulse is often 160 to 180 a minute without giving any cause for anxiety. An increased frequency is sometimes noted even in cases without fever.

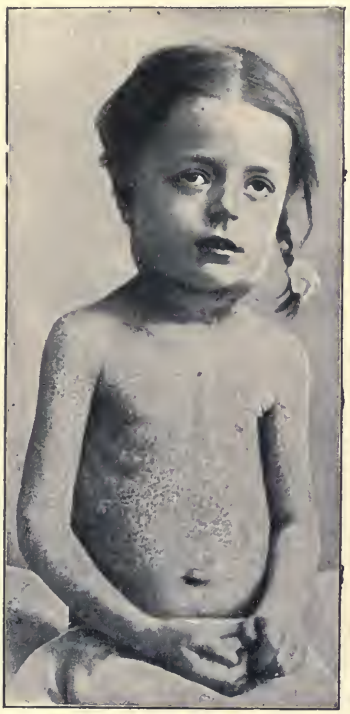


FIG. 151.—Five-year-old girl on the fourteenth day of scarlet fever. Bilateral phlegmon of the cervical lymph nodes and marked desquamation on the trunk and arms.

The patient's *general well-being* is often profoundly affected. Apathy or restlessness, insomnia, anorexia, and weakness are usual manifestations. Vomiting often occurs even after the first day or two.

The disease involves the respiratory tract slightly, if at all, and does not affect the ears or the conjunctiva. A moderate degree of congestion of the nasal mucosa may cause some difficulty in breathing, but is accompanied by very little secretion.

Aside from the increased frequency of the pulse-rate and an occasional slight systolic murmur, the heart gives no special indication of disturbance during the fever.

The *blood* frequently shows a neutrophilic leucocytosis which does not disappear until the second or third week. At times there is an increase also of the eosinophiles, discoverable at the end of the first week, but, according to our observations, often lacking. The leucocytes of recent cases attended with high fever often contain certain inclusions (Döhle). These are frequent, however, in other infectious diseases, such as lobar pneumonia.

Enlargement of the lymph nodes of the submaxillary region may be quite marked and may make movements of the head painful. Other superficial nodes, among them the cervical, inguinal and axillary groups, are also distinctly involved. The *liver* and the *spleen* are occasionally distinctly enlarged. The *urine*, during the course of the fever, is cloudy, scanty, and concentrated. Often it contains some albumen, casts, and red blood-cells, but it clears up with the fall of the fever if a secondary nephritis, which is so common a complication, does not ensue. Its reddish color is due to the presence of a quantity of urobilin, which is a characteristic of scarlet fever. Acetonuria is often present.

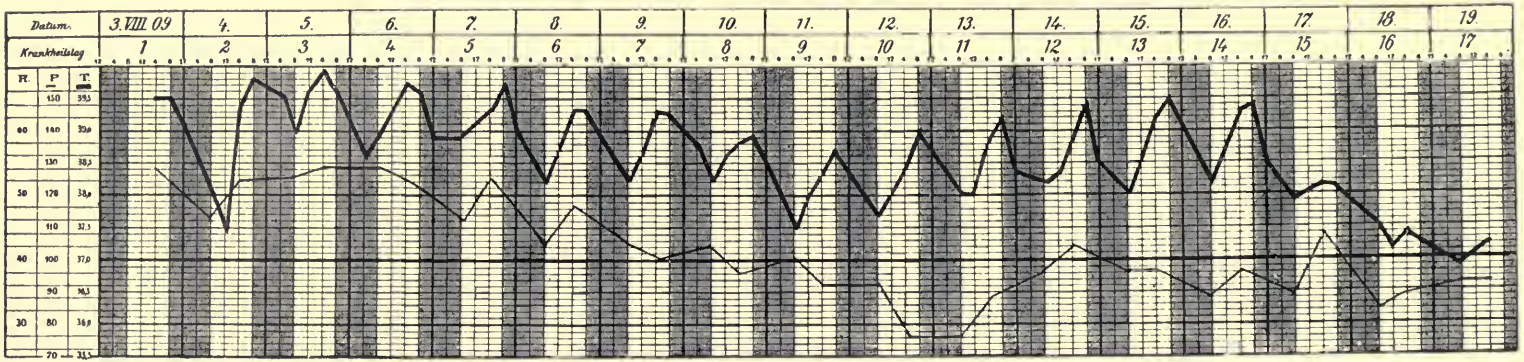


FIG. 152.—Scarlet fever. Persistent fever without complications. Angina without exudate. Seven-year-old boy.

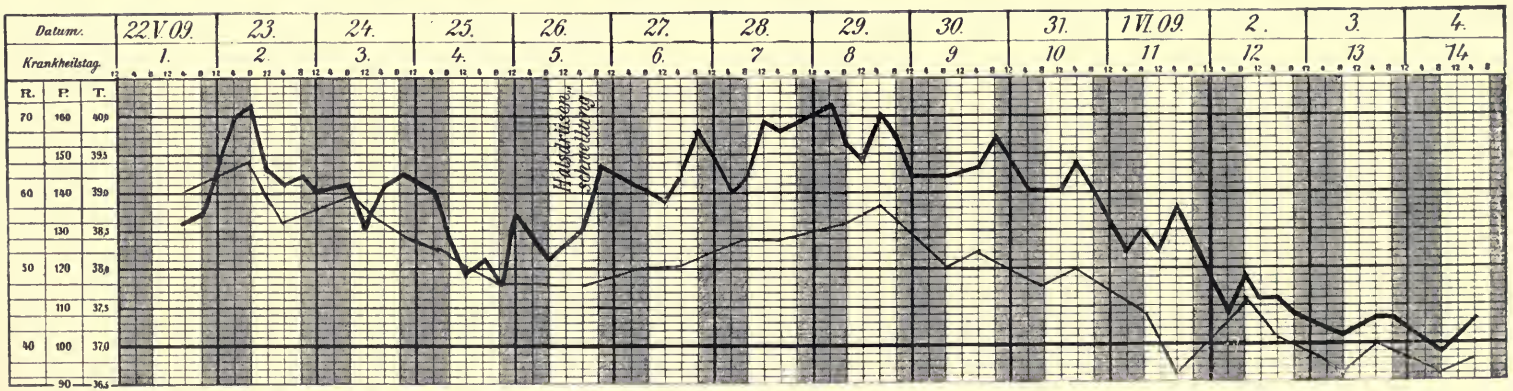


FIG. 153.—Scarlet fever. Swelling of the cervical lymph nodes (fifth day) causes continued fever Two and one-half-year-old girl.

With the decline of the fever these various symptoms disappear. By the middle or end of the second week the patient is fully convalescent. In fact, it is often difficult to keep him in bed and away from other children until desquamation is complete.

Peculiarities, Sequelæ and Complications of Scarlet Fever

The description given serves for a mild typical form of scarlet fever as it may be seen in a majority of cases. Variations in form of even mild attacks, but particularly in those inclining to a greater degree of severity, are so extremely common among sporadic cases as well as in epidemic outbreaks, that it hardly seems proper to attempt a classical description. Nor is it correct to call the numerous disturbances, which so continually appear at the close of the fever period, such as phlegmon or scarlatinal nephritis, etc., complications of the disease, since they are as apt to follow the mildest, as they are the most fulminant types. It is better, indeed, to term them sequelæ, since they make their appearance within a definite period of three to six weeks and are produced by the virus of the scarlet fever itself.

The mildest expressions of the malady are so common and so prevalent, that Sydenham and Bretonneau felt, at one time, that they could scarcely call scarlet fever a disease; while later they were convinced by hard experience, that scarlatina may be one of the severest afflictions of mankind. Frequently the angina, the fever, and the malaise are so insignificant, that the sick child's parents become aware of his definite illness only through the appearance of the skin eruption. Indeed, the whole course of the disease may be so mild, that it is entirely overlooked until the ensuing desquamation or the development of nephritis calls attention to the fact, that the child has had scarlet fever. The angina may be so very slight, the fever never exceeding 38° C. (100° F.), and the eruption so trivial, that a diagnosis in sporadic cases is often impossible. The author is quite positive, that he has seen such a case in a family the other members of which had more serious types of the disease. On the other hand, *rudimentary forms* are seen in which, though the eruption is extremely transient or wholly lacking, other symptoms are very distinct. When but slightly developed the rash is usually most clearly visible upon the back. In rare instances the appearance of the eruption is delayed until the third to the fifth day after the onset of the disease. Cases occur without eruption (*scarlatina sine eruptione*), chiefly among older children or adults; and naturally these are readily mistaken for angina or diphtheria if the development of unequivocal forms of the disease in other members of the family does not assist the diagnosis.

In sporadic cases of the fulminant type the erupted spots are usually large. In rare instances, during convalescence, the skin tends to a peculiar local reddening and to a necrosis, resulting from traumatic irritation (*erythema post-scarlatinosum, Schick*).

The *severe toxic type* of the disease stands in sharp contrast to the mild and easily overlooked forms. The child suddenly becomes ill, with high

fever, vomiting, convulsions and delirium, which may pass into complete unconsciousness. The respiration is deep and toxic, the pulse extremely rapid and hardly palpable. The lips and skin are cyanotic, the hands and feet cold, despite the high fever. The throat is swollen and red and the skin is mottled or shows large blue spots, but no scarlatinal eruption. Death may result, sometimes in one or two days, more frequently in from three to five days.

In cases which are not so rapidly fatal, a more severe angina and a more marked inflammation of the lymph nodes of the neck develop. A distinct eruption, sometimes visible in only limited areas, may appear a day or two before death. Excepting in an epidemic, such fulminant cases are very rare, but they may be met with now and then in families in which other children are suffering from milder forms. According to Czerny, this fulminant form occurs chiefly in children of exudative diathesis.

Again, we see cases of violent onset with high fever, severe malaise, marked angina, in which, nevertheless, all symptoms disappear after five or six days, when the disease goes on to a favorable termination. Sometimes, in such cases the picture of a simple or lacunar angina is replaced by that of necrotic angina, or diphtheroid scarlet fever. The surface of the tonsil is partially or entirely covered by a whitish exudate, which may seem, at first, very thick and may have a membranous character closely resembling true diphtheria. Soon, and sometimes from the onset of the disease, this exudate becomes more deeply imbedded in the mucous membrane; it has a pasty appearance and can be removed by scraping in large pieces, as in a case of diphtheria. It is less fibrinous, however. Frequently the infiltration spreads to the anterior palatine arch. Seldom it spreads to the posterior wall. Coincidently with this more or less diffuse necrosis of the mucous membrane, appears a swelling of the lymph nodes of the neck. The inflammation often spreads to the peri-nodal tissues. Movement of the head becomes extremely painful and is avoided by the patient. When the phlegmon in the region of the submaxillary gland is hard and tense and spreads to such an extent that the bilateral swelling meets beneath the chin, the condition is serious.

Necrotic angina frequently spreads to the nasopharynx or may originate there. The median raphe of the soft palate is then seen, from behind, to be white and infiltrated; nasal breathing becomes difficult and as a result of the invasion of the nares an irritating seropurulent excretion flows from the nose and produces small sores upon the upper lip. The lips are often so badly fissured that opening of the mouth causes severe pain. The buccal mucosa is markedly reddened and a dirty whitish deposit, easily removed, is seen along the edges of the gums. A case of this kind may be considered relatively favorable if the necrosis does not exceed the limits we have described. Within five to eight days the necrosed tissues may slough without any more serious injury. But in severe cases the necrosis goes deeper and in a few days the tissue of the tonsils becomes a discolored brownish mass. The destruction may extend, also, to the pharyngeal arches, the uvulas and the pharynx. An excessive muco-sanguino-purulent secretion cover,

the invaded area and prevents closer inspection. The necrosis may even spread to the epiglottis and to the false and true vocal chords, causing hoarseness and stenosis of the larynx.

This deep necrosis is certainly due to a streptococcic invasion. The streptococci may be found massed in the pseudomembranous exudate of the condition. The same infection may cause a dry necrosis of the lymph nodes of the neck, especially in the sublingual and cervical groups. From the neck, the infection may spread to the mediastinum causing a purulent mediastinitis, which may involve the trachea and may even produce general sepsis and metastatic abscesses in the various larger joints, in the peritoneum, or in the pleura etc.

If the necrotic angina involves a large area, it may be a direct cause of death, fatal termination ensuing from general exhaustion, in from a few days to three weeks time, as a result of sepsis. It may be fairly said, that the presence or absence of angina determines the outcome of the disease and indicates the severity of the epidemic during which the case occurs.

The ear is very frequently affected in the course of the disease, even with a simple angina, but an otitis media is especially associated with diphtheroid infection of the nasopharynx. Purulent otitis media usually makes its appearance by the middle or end of the first week, but occasionally develops much later. It usually destroys the entire tympanic membrane after but a very short period of pain, followed by a purulent discharge. The otitis of scarlet fever is peculiar in that it readily causes necrosis of the auditory ossicles and infection of the mastoid. It may result in permanent disturbances of hearing or in complete deafness. Mastoiditis and septic sinus thrombosis are not uncommon sequelæ. The great majority of cases, however, terminates quite favorably.

Necrotic angina occurs not only in cases that are initially severe, but also in those that during the first four or five days are apparently mild. The temperature even begins to fall, but the lysis stops at the end of the first or the beginning of the second week and the temperature rises again without, for the time being, any apparent reason. At this period it may be understood that any new rise of temperature indicates a new localization of infection or a new complication. Frequently it is the evidence of necrotic angina, of otitis, or severe lymphadenitis (Fig. 151). On this account a careful notation of the temperature curve is important. There are cases in which a diphtheroid angina precedes the eruption. These are easily mistaken for true diphtheria and are erroneously isolated in the diphtheria ward. Older and stronger children recover from the severe forms of necrotic angina and its sequelæ and even deep ulcers and extensive loss of substance may heal, but the general health of the patient is always profoundly affected. Very frequently however, consequent septic processes will cause death weeks later, despite the fact that the angina may have healed.

The *respiratory organs* as a general thing, are but slightly affected. In severe cases, respiration is often embarrassed on account of the closure of the nares or the stenosis of the larynx, incident either to the severe inflam-

ation of the mucosa or to the pressure arising from involvement of the mediastinum. In some instances the hoarseness and stenosis are so aggravated as to suggest interference. Rarely, however, is the stenosis sufficient to justify this and hardly ever does the procedure prolong life.

Purulent bronchitis and broncho-pneumonia are not uncommon complications, nor is an exudative pleuritus which always becomes purulent and sometimes is associated with a purulent pericarditis.

Occasionally the conjunctivæ are as markedly affected as in measles.

Scarlatinal rheumatism is the term applied to the painful swelling of individual joints, which may appear at the end of the first or during the second week of the disease. It occurs particularly in the hands, fingers, knees and feet and its course is attended by an increase of temperature. The swelling is often slight and without any redness. This affection should not be confused with pyemic disease of the joints. It always disappears within a short time.

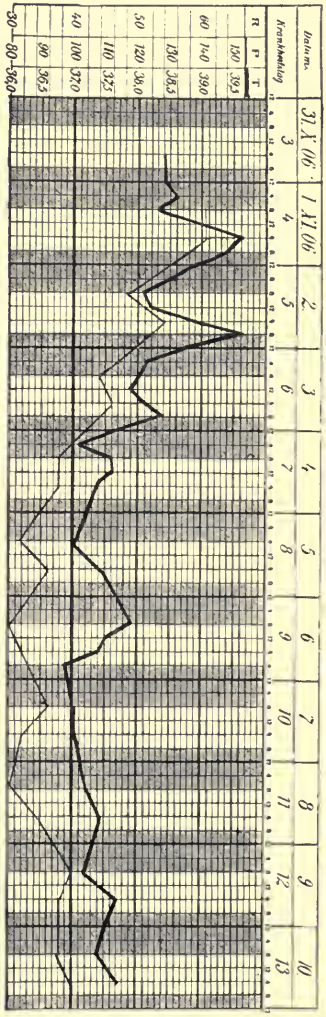


Fig. 154.—Milder form of scarlet fever. Bradycardia from the eighth to the eleventh day. Twelve-year-old boy.

The heart is frequently involved. Even the unusually rapid heart-beat common in scarlet fever shows that the toxins of the disease have a special affinity for the heart. In severe cases the increased heart action may persist for weeks after the fever has disappeared. It is associated at times, with signs of cardiac weakness. Actual disease of the heart is quite common even at the beginning of the attack. It has been especially studied by Schick. Even in mild, benign, cases it is often possible to demonstrate, at the end of the first or in the course of the second week, impure heart sounds at the apex with bradycardia and enlargement of the heart to the left (Fig. 154). Added to these findings, a systolic murmur is frequently heard in the pulmonic and apical areas. All these phenomena, lasting for several weeks may eventually disappear. It is probable that no myocardial changes are present in these mild forms, but that merely an atony of the heart exists, often associated, according to Sederer and Stolte, with loss of body-weight It is not to be forgotten, however, that in the course of scarlet fever endocarditis may develop

very insidiously during the fever period at the onset of the disease and that it may lead to permanent valvular lesions and particularly to mitral insufficiency. Pericarditis, as a complication of scarlet fever, is less common.

The Gastro-intestinal Tract.—In severe cases appetite entirely fails even when there is no difficulty in swallowing. Excessive diarrhœa is often a sign of sepsis.

Commonly the nervous system is but slightly affected. Somnolence and delirium may appear in case of severe infection with uremia. Maniacal and depressive conditions may appear temporarily during convalescence. True meningitis is rare and is usually a manifestation of general sepsis, sinus thrombosis, etc.

Fever falls by lysis only in uncomplicated cases. The fall of temperature usually sets in at the beginning of the second week, but even in cases without particular localization, it may not be completed before the end of the third week. Even though the temperature has reached the normal level and no severe throat affection or other complication exists, the disease may not be ended. *Sequelæ* may still threaten.

Irregular rises of temperature, which are always a subject for careful study, may appear after the end of the second week and even as late as the sixth week. Very often no reason for such a rise can be found, even after most careful observation, and it is termed a relapsing or typhoidal scarlatina. Frequently, however, a sequel may presently appear in the form of a renewed swelling of the cervical lymph nodes, an otitis media, an endocarditis or occasionally even an angina, or a recrudescence of the rash. Now and then, one has to deal with a pseudo-relapse, when a case mistaken for scarlet fever has been placed in an isolation ward for this disease and then actually acquires it.

Nephritis is the most important and the most common sequel of scarlet fever. Usually it appears in the third week of illness or at the earliest by the twelfth day but it may develop as late as the fourth or even the sixth week.

Nephritis is often heralded by fever, vomiting and headache, while again attention may be called to it by the development of edema, or by the discovery of blood or albumen in the urine. The disease in itself, is always essentially an injury to the vascular structure of the kidney, a glomerular nephritis, by which the excretion of water is especially impaired. The urine is almost always bloody; at times it contains much blood and is scanty. The greater the diminution in the quantity of the urine, the more serious is the case. The amount of albumen present, up to one per cent., and the appearance of red blood-cells and of a variety of casts, are of less prognostic importance. The course of this complication is often marked by an irregular fever (Fig. 155). Frequently a marked dropsy, which may involve the peritoneal, pleural, or pericardial cavities, develops. Cardiac dilatation, accompanied at times, by bradycardia, is of early occurrence. Blood-pressure is increased. Headache and vomiting are frequently symptoms and often occur with the onset of uremia, which is again associated with bradycardia, and may lead to convulsions and amaurosis. Death

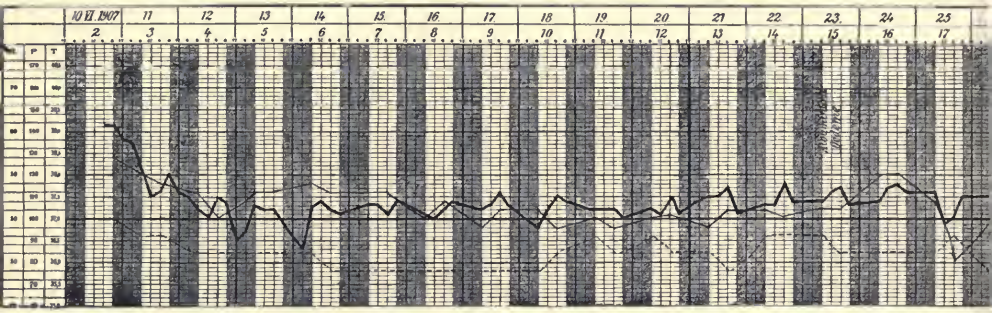
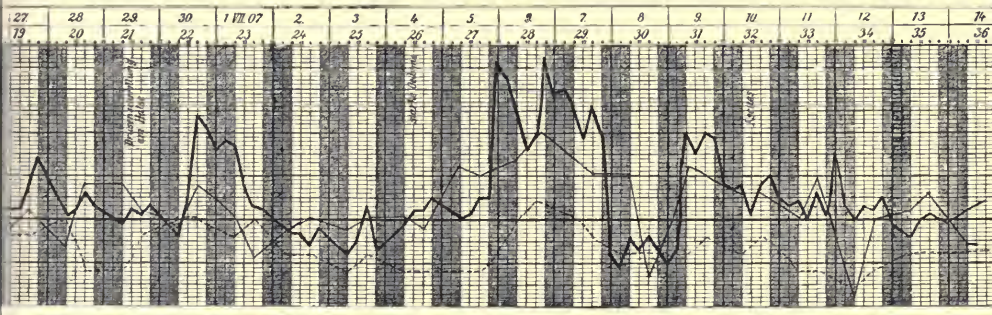


Fig. 155.—Scarlet fever with nephritis (15th day



mphadenitis colli (21st day). Eight-year-old girl.

may result from uremia and dropsy or dilatation of the heart. In most cases recovery from nephritis follows in from four to six weeks; more rarely it may be a matter of months. Not infrequently it runs into a chronic form, which may later develop into a contracted kidney.

The frequency of nephritis varies greatly. It ranges from two to thirty per cent. of all cases, depending upon the conditions of the individual attack and the general character of the epidemic in which it occurs. It is not always the severe types of the disease, that seem to show a peculiar tendency to it. Nephritis, indeed, may develop after the very mildest forms and even in cases, which have escaped observation. For a more detailed description of the nephritis of scarlet fever the reader is referred to page 431.

The complication of scarlet fever with diphtheria deserves special consideration. Even before the science of bacteriology had evolved, careful observers had differentiated a diphtheroid form of scarlatina, without involvement of the trachea and not followed by paralysis, from true diphtheria. The membrane in the former instance, despite its frequent and very close resemblance to that of diphtheria, does not show the Löffler bacillus. In the smear and in the culture chiefly streptococci are found. Mixed infections, however, are not uncommon. When the two diseases are coincidently epidemic and when cases are not properly isolated, perhaps, in contagious hospitals. Clinical differentiation by reliance upon appearances in the throat, is often initially impossible. In doubtful and severe cases, and in the absence of the exanthem, it is well to give an injection of antitoxin without waiting for a bacterial diagnosis. When diphtheria develops late in the course of the scarlatinal infection, after the initial throat symptoms have disappeared, it is more easily recognized.

As a matter of fact the course of scarlet fever is subject to numberless variations and recovery is prejudiced by unforeseen focal conditions, sequelæ and complications. Even in serious cases the organism often conquers after weeks of fever and relapse, but unfortunately not always without permanent injury. The most frequent irreparable damage is that to the auditory apparatus, which may result in deafness. Valvular heart lesions are less common and chronic nephritis is most rare.

The diagnosis of scarlet fever, in advanced cases, is readily made from the skin eruption, the fever, the characteristic angina and the strawberry tongue. The possibility of a scarlatinal origin is to be considered in even the mildest forms of angina and even in the absence of the exanthem. Much greater problems present themselves to the physician than appear in measles. The eruption by itself may never be considered conclusive and mistakes are especially possible in the absence of angina. The rash may be so light and so transient and so atypical as to serve only to create doubt. Lasting for only a few hours or at most, a day it is commonly not due to scarlet fever. "Scarlatina sine eruptione" may be recognized with certainty only in the presence of coincident and unmistakable cases or when a diphtheritic exudate appears without diphtheria bacilli.

The greatest and often quite insurmountable difficulties are presented

by those infectious and toxic erythemata, common among children, which resemble scarlatina. Particularly the fourth or Duke's disease produces an eruption very similar to that of scarlet fever, without exhibiting any of its remaining symptoms which in scarlatina, also, of mild degree, may be absent.

Ordinarily, scarlet fever is readily distinguishable from measles since it lacks the prodromal coryza and the Koplik's spots. It is to be noted, however, that in severe cases of scarlatina a marked conjunctivitis may develop. The large macular eruption characteristic of measles is never general in scarlet fever, although it is found in small areas and notably on the extremities. Even in the confluent form of measles some areas will always be found in which the eruption is characteristic. In rubella the spots are always discrete; they are generally larger and with more free spaces between them than in scarlet fever. A scarlatinoid serum exanthem may present considerable difficulty of differentiation but it usually spreads from the point of injection; it is not associated with angina; it is transient and rapidly changing so that rubeolar and urticarial eruptions may be coincidently or alternately present. In recrudescent desquamative scarlatinal erythema, angina also is lacking. Moreover, the condition is characterized by early and unusually severe desquamation and repeated attacks. It is an extremely uncommon disease.

In addition, there are a number of infectious diseases in which a scarlatinoid erythema may appear. In lobar pneumonia, typhoid fever, la grippe or influenza, and in the acute disturbances of nutrition of infancy it may be present. A similar rash results sometimes from the use of such medicinal agents as quinine, iodoform, mercury, atropin, etc. The early eruption of chicken-pox on the thighs and shoulders may resemble that of scarlet fever. The redness of the skin in crying or blushing, now and then reminds one of the rash of scarlet fever. Occasionally in young children, an exanthem of miliaria rubra, caused by perspiration, spreads over the entire body. It may be distinguished from the scarlatinal rash only by the fact, that the eruption is raised above the level of the skin. The color of the skin in scarlet fever, as seen when pressure is made upon the eruptive surface is an important feature.

As may be inferred, it is often impossible to pass upon a case of scarlet fever from the eruption alone. A smooth diffuse erythema of transient appearance confined to small areas, pressure upon which does not enable one to recognize the individual maculæ, and surrounded by a coarse urticarial eruption is, generally speaking, not of scarlatinal origin. Associated symptoms and especially those of the throat and tongue must always be taken into consideration. Even the blood findings may aid in making a differentiation. Polynuclear leucocytosis, with eosinophilia is indicative of scarlet fever. Leucopenia, with a reduction of the polynuclear cells suggests a serum exanthem. Quite frequently, however, the question remains undecided unless other neighborhood cases occur, or the unrecognized attack leads to desquamation or is followed by nephritis. The development of nephritis, with recurring enlargement of the cervical nodes, but

without accompanying angina suggests the scarlatinal cause of the disease and may account for a recent unexplained sore throat.

There are many non-scarlatinal erythemata in which desquamation does not occur or is very slight. If it appears, it is always earlier and more rapidly concluded, while it does not so markedly involve the hands as in scarlet fever.

The prognosis must be made always with extreme care, since it is impossible to anticipate surprises even in mild cases, as the above exposition has made sufficiently clear. Every remission of fever may usher in an unpleasant and dangerous sequel and complication. There is no disease so treacherous and uncertain as is scarlet fever. The course of the disease in any individual case is very greatly influenced by the general type of the local epidemic. The fatality varies with the characteristics of the prevalent complaint from one to fifty per cent. The disease is most fatal in children of from two to four years of age. Mild forms will recover without treatment while severe cases sometimes terminate fatally despite it. The most dangerous complication is the necrotic type of angina. In its absence a favorable course may be expected. The prognosis is unfortunately influenced where the lymphatic diathesis exists. Hardly a disease is met, which gives the physician so great a sense of helplessness as do the toxic or necrotic forms of scarlet fever.

Prophylaxis depends upon the strict isolation of the patient until desquamation is completed or until middle ear or lymph nodes complications are entirely cured. Usually this is only attainable in the hospital.

Thorough disinfection of both room and contents after the patient's recovery and the lifting of quarantine is essential. (See Page 579.)

Treatment.—In mild cases expectant treatment is indicated. While it is customary even in mild cases, which may be convalescent within a few days to keep patients in bed for three or four weeks, this is really a diplomatic measure to relieve the physician of blame should nephritis develop within this period. In hospitals two weeks is considered a sufficiently long time. The writer is fully assured that this prolonged rest in bed is not in any degree preventive of nephritis. Neither to any demonstrable extent is the strict milk diet recommended for the first three or four weeks (Pospischill). Severe nephritis occurs in cases in which both of these measures have been stringently carried out. On the other hand, children who are permitted to run out of doors after the third or fourth day very often escape nephritis altogether.

The diet should consist at first of milk gruels and flour puddings with water and fruit juices. In the second week, toast, barley soup, apple sauce and mashed vegetables may be added; in the third week further additions of eggs and bread may be made while meat may be allowed in the fourth week. For older children or if there is marked difficulty in swallowing, the milk may be flavored with a little cocoa with which milk toast, rice, stewed apples, oranges etc., may be given.

In severe cases anorexia is often marked and so difficult is it to tempt the appetite that a little meat may be permitted. To very sick children,

especially with high fever, liquids should be given generously and if necessary, by enteroclysis. In young children water serves to cleanse the mouth after eating.

The fever hardly ever requires special attention. Antipyretics must be avoided, since they are apt to weaken the heart. If the temperature runs higher than 39°-40° C. (102°-104° F.) and is accompanied by headaches and a sensation of heat, etc., one or two tepid baths, 32°-33° C. (90°-92° F.) will give good results. If there is undue somnolence the tepid bath may be followed by a cold douche. Very high temperatures may be reduced by wet packs, with water at room temperature, continued for ten or fifteen minutes. These packs may be repeated two or three times. Cold applications to the chest or abdomen, similarly applied and repeated once or twice a day, are distinctly beneficial. If the fever remains persistently above 40° C. (104° F.) an ice-cap placed alternately upon the head and over the region of the heart is to be recommended.

Cold packs are not always borne well and therefore, must be carefully controlled.

When desquamation begins, a daily tepid bath is to be recommended. This aids the rapid exfoliation of the skin and prevents the scattering of its detritus. For the latter purpose, the inunction of the body surface following the bath with a bland ointment, as ordinary cold cream, is also useful.

The care of the mouth and nose demands strict attention. Children who are old enough should be required to gargle regularly with warm water, a very dilute solution of hydrogen peroxide or of boric acid. Younger children should be given water immediately after eating.

In young children, suffering with severe angina, who cannot gargle, the mouth may be frequently irrigated with one of the above solutions, the head being held forward, provided the measure can be accomplished without too much resistance. Sometimes the patient comes to like these irrigations.

If the inflammation of the tonsils and the pharynx becomes very severe, cold applications to the neck renewed every half-hour or oftener prove very acceptable. If the fever is high an ice-bag even may be used. Upon the subsidence of the fever, ordinary cold applications may be again substituted and later changed to fomentations. If an exudate appears, gargling with older children will suffice; but with younger patients the throat should be sprayed several times a day with a solution of hydrogen peroxide. This is particularly efficient when irrigation cannot be employed. Insufflation with equal parts of sodium sozoiodolate and precipitated sulphur has an apparently favorable effect. At the same time if these measures cannot be carried out without the use of extreme force, they should be avoided. Inflammation of the lips resulting in rhagades and ulceration, will often make the opening of the mouth even for the purpose of inspection, painful. The accumulated secretions should be removed from the lips frequently and lanolin should be freely applied.

In diphtheroid angina, ice-bags or ice packs may be recommended during the first day of the attack. A little later, however, tepid or hot packs are to be preferred. If the lymph nodes are swollen, ice may be applied dur-

ing the continuance of high fever, but it should be replaced very soon by cold applications renewed hourly and later by cataplasms. The latter should be employed systematically with nodal swellings of long duration. They favor resorption when this remains possible and if not, they tend to promote softening. An abscess should be incised only when the accumulation of pus is large; otherwise only dry necrotic tissue may be encountered.

The nose should be cleansed regularly. If a copious discharge appears, it should be removed with pledgets of cotton, and the nostrils and upper lip should be protected from erosion by applications of lanolin. Frequently the insufflation of sodium sozoiodolate as already recommended or of bolus alba (Trumpp) gives relief.

In otitis media, with reddening and bulging of the drum membrane the instillation of a ten per cent. solution of phenol in glycerin may relieve pain. Necrosis of the membrane indicated by its white color usually occurs rapidly and paracentesis need not be considered. The treatment of scarlatinal otitis and of the infections of the mastoids which frequently ensue, is of the usual order. Mastoiditis may occur even as late as from three to six weeks. Brain abscess and sinus thrombosis are rare. Deafness does not often result.

The rheumatism of scarlet fever disappears without any treatment. Rest and the use of the salicylates however, give much relief from pain and probably hasten recovery.

Daily examination of the urine for ten to forty days following the onset of the disease may be readily done by the mother at home. The boiling of the urine, with the addition of nitric acid, suffices. It should be insisted upon in every case. Upon the appearance of nephritis it is well to measure the twenty-four hours' output of urine. Marked diminution, below 400 to 500 c.c. always suggests the danger of uremia.

If uremia develops, blood letting to the extent of 100 to 200 c.c. often gives excellent relief. In the event of uremic convulsions lumbar puncture may be tried. If the patient in coma or convulsions cannot take water by the mouth, large quantities should be given by enteroclysis. For further details in the treatment of nephritis, the reader is referred to page 433.

Stimulants are very often necessary in the course of scarlet fever, since its toxins are especially destructive to the heart and the vasomotor system. In mild cases sufficient stimulation may be secured by adding a fairly large allowance of strong coffee to the milk. With the weakening of the heart, indicated by a small and frequent pulse, camphor and caffein must be employed (see p. 415). In serious cases even these remedies fail.

Recently the serum therapy of scarlet fever has been attempted in a number of instances. Antisera from the horse, infected with streptococci are generally used. Good results have been reported in a certain percentage of these cases, but final judgment upon the merits of the method must be withheld. Moser's serum seems to have earned the fullest confidence, but even with its use results have been attained only during the first few days of the disease and in purely toxic cases. Doses of 150 to 200 c.c. of the serum are required and must be given in a single injection. Since with such a dose, the development of a severe grade of serum disease is more probable

than a favorable effect upon the scarlet fever, the remedy can hardly be said to be indicated in general practice.

Convalescent serum, plasma or whole convalescent blood has been used in the treatment of early toxic cases of scarlet fever both in America and abroad, and has given encouraging results in the limited number of cases observed thus far. Reiss and Jungman recommended the intravenous injection of 50-100 c. c. of pooled convalescent serum, while Zingher made use of the intramuscular injection of whole convalescent blood citrated or uncitrated, which he injected in quantities of 120-240 c. c. The convalescent serum of fresh whole blood is obtained from patients, who are two or three weeks convalescent from scarlet fever. These donors should be free from syphilis and tuberculosis. Fresh normal blood has no specific action in septic cases of scarlet fever, but it supplies definite nutritive, stimulating and normal bacteriocidal substances and can be used to advantage in toxic cases where convalescent serum or blood is not available.

MEASLES (Morbilli)

Measles is a febrile infectious disease, characterized by a macular eruption of the skin, preceded by an acute affection of the mucous membranes of the mouth, the conjunctiva and the upper respiratory tract.

While measles is supposed to be a very ancient disease, it was not clearly differentiated from scarlet fever and small-pox before the eighteenth century. It is the most common of the infectious diseases and is distributed over the entire earth.

Its *causative organism* is still unknown, in spite of numerous researches. The virus is extremely volatile, since the disease is very readily conveyed from one person to another and even without direct contact. It is moreover, very short-lived; and, consequently, the disease is almost always carried directly from the sick to the well. The infective principle dies in a very short time outside the human body. Cases of indirect transmission by infected utensils or by healthy individuals as carriers have been observed but they are so extremely uncommon that practically they may be disregarded.

In the Faroe Islands, where opportunities of observation were excellent, transmission by healthy persons was never proved. Indirect distribution of the disease has occurred, in all probability, only when the uninfected carry with them from the sick bed some infective material, which they convey to some other person before time has permitted it to fall below the temperature of the body. Physicians assuredly go directly from the house of the measles patient to other homes without the least danger of carrying contagion. Everything points to the conclusion, that the virus loosened from the diseased mucous membranes is sprayed into the surrounding atmosphere by sneezing and coughing. In former years in fact, successful inoculation was practiced by the use of the secretion from the nose, eyes, or mouth of the patient with measles.

The infectiousness begins in the prodromal stage, the stage of the disease covering a period of three or four days before the appearance of the

rash and it persists throughout the exanthem, disappearing within eight or ten days, or even less, of the appearance of the rash. Infection is most apt to occur two or three days before the outbreak of the exanthem, so that secondary cases within a family develop in eleven or twelve days, some two weeks intervening between the entry of the infection and the eruption of the rash.

Predisposition to measles is extraordinarily great among all peoples and continues throughout life even to old age. Infancy alone, presents an exception. The disease is at least extremely uncommon during the first four months of life. Older infants have it in very mild forms. That measles occur chiefly as a disease of childhood is simply due to the fact, that most persons are infected early and that one attack confers an almost absolute and life-long immunity. Children whose mothers suffer from the disease while carrying them are without exception unimmunized thereby. A second attack of measles is a great rarity. The author has had the opportunity of observing but one case in which recurrence could be proved without question. According to reports current among mothers two or three attacks might be considered usual; reports that are doubtless due to the confusion of measles with all manner of infectious diseases and toxic erythemata. The prevalent appearance of measles in early life is evidence of the general predisposition and the almost invariable immunity conferred by the one attack.

Measles usually appears epidemically and spreads rapidly, reaching its maximal prevalence in a short time. The disease disappears as rapidly when the non-immunized human material is exhausted. Small isolated villages may escape measles for ten or twenty years, until some case is imported. In larger towns it will occur in distinct epidemics every two to four years (Fig. 156), with intervals comparatively free of cases. In the great cities single cases, which carry on the contagion from one period to another are always to be found. These isolated cases may cause local outbreaks, without the occurrence of large epidemics. If the disease is carried to an unfrequented island, where it has never been or in which there has been no epidemic for decades, the entire population may be affected with the exception of its young infants. Carefully gathered statistics of Faroe Islands have shown that 99 per cent. of its people have been susceptible to the disease. Its first invasion of Samoa, in 1893 caused the death of 4000 persons, half of whom were adults. Nearly all the inhabitants were affected.

Children in the great cities are, as a rule, infected between the ages of two and six years; nevertheless this cannot be said to be the period of greatest predisposition. For unknown reasons a person's predisposition is temporarily varied, so that a child may escape the first opportunity of infection only to be infected later. The seasons exert no essential influence upon the development of epidemics. Nevertheless, a certain tendency to this prevalence of the disease in the cold months and especially in the spring, may be recognized. This is apparently explained by the greater susceptibility of the respiratory tract at these seasons, supported by the well justified assumption, that the contagion of measles enters through the air passages.

The mortality varies widely from year to year and from one epidemic to another. In large cities it often runs as high as three to five per cent. at large and up to thirty per cent. in hospital practice. In the first two or three years of life the fatality is greatest. In ordinary epidemics deaths of children of over five years are uncommon.

The pathologic findings give us no insight into the nature of the disease, but usually emphasize its fatal complications. If a case comes early to autopsy the skin lesions are still prominent. A marked hyperemia is found. About the efflorescence, markedly paled in death, the small vessels of the papillæ are greatly distended and are surrounded by a round cell infiltration. These phenomena are especially marked about the sebaceous glands and the hair follicles. They probably explain the papillary eminences in the centre of the erupted spots (Heubner).

Symptoms.—Commonly it is possible to recognize four stages in measles; first, the incubation period; second, the prodromal or initial period,

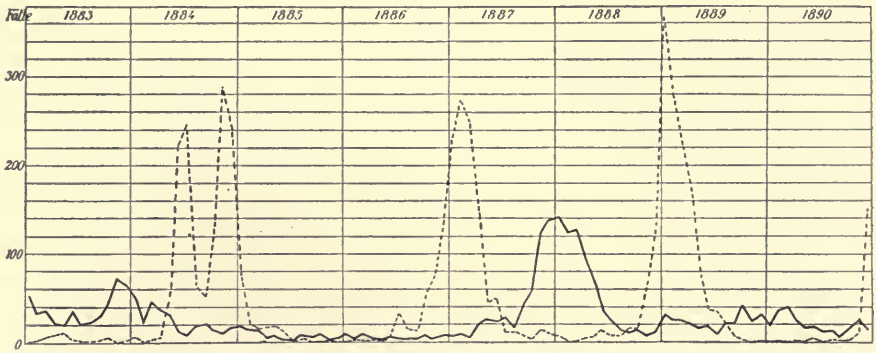


Fig. 156.—Morbidity in the city of Basel, Switzerland, of measles and scarlet fever from 1883–1890 graphically represented according to the frequency during the various months. Measles..... Scarlet fever———

the stage of the enanthem; third, the exanthem; and fourth, the stage of convalescence.

The time elapsing between the infection and the appearance of the rash, that is, the incubation plus the prodromal period, covers with marked regularity from thirteen to fifteen days. This circumstance often affords a clue to the source of the infection. This period is very exceptionally extended to sixteen or even twenty days, the delay being occasioned frequently by the presence of intercurrent infectious diseases.

The stage of incubation alone, usually covers ten, or eleven days, and the prodromes occupy three or four days. Cases, however, are often seen in which the actual incubation takes only from seven to nine days and the prodromes a matter of five to seven days, the rash still appearing within fourteen days.

The disease-picture in general shows fairly uniform features. The incubation period is hardly ever marked by any noticeable disturbances. Now and then there is slight disorder of digestion, catarrh, and malaise. The

temperature varies. At times it is febrile and very definitely and significantly so for a few days before the onset of prodromes.

The *prodromal stage or enanthem* is characterized by an affection of the mucous membranes of the eyes, mouth and respiratory passages. This stage sometimes has an insidious onset, but commonly is accompanied by distinct signs of fever and catarrh. The child previously happy and well suddenly becomes tired, the conjunctiva grows red, a nasal discharge and an annoying cough begins. The reddening and swelling of the conjunctiva may be quite severe and may be accompanied by a free flow of tears, by profuse watery or purulent secretion, increasing from day to day and by marked photophobia.

The nasal mucosa swells, frequent sneezing occurs and a thin and sometimes purulent discharge appears.

Simultaneously, or within a few days a dry hard cough sets in, which is unaccompanied by any auscultatory signs. The laryngeal quality of the cough and the hoarseness, which develops upon crying show that the vocal chords are affected. In patients especially predisposed severe attacks of pseudocroup often occur. The oral mucous membranes, the tonsils and the pharynx are injected and show a hypersecretion of mucus.

The temperature during the early prodromata rises to 38.5°-39.5° C. (101°-103° F.). With marked remissions it falls even to the normal point on the second or the third day, the drop giving way to a sharp rise upon the appearance of the eruption. At the onset the general health is frequently but slightly affected, although severe disturbance may follow. Headache, loss of appetite, occasional vomiting, at times diarrhœic stools, and broken sleep incidental to the severe coughing may ensue. Within two or three days these symptoms subside with the fall of temperature, to recur more severely with the development of the skin eruption.

The entire picture resembles at first that of a severe coryza, of influenza or of la grippe. Not infrequently the inexperienced or superficial observer makes the correct diagnosis only when the eruption appears. During this initial period, however, a careful examination of the oral cavity often makes it possible to establish the diagnosis of measles several days before the appearance of the rash.

The reddening of the tonsils, the throat and the oral mucosa does not differ at first from the injection observed in any other severe catarrhal affection. But soon, a distinctive sign by way of spots characteristic of measles appear upon the buccal mucous membrane. Their appearance antedates by two or three days the skin eruption. Their significance was first clearly recognized by Koplik and they have become known as Koplik's spots. Upon the dull red surface of the mucosa, usually opposite the lower molars, a number of these bright red spots the size of a pinhead are to be seen. At the centre of each is a smaller white point, which looks like a fine granule of salt (Fig. 157). This white point feels as if raised; it consists of fatty epithelial detritus, and may be rubbed off if a little force is used.

The number of the Koplik's spots varies greatly. At times only two or

three are present and are so small as to be very difficult to find; especially so when the red base is lacking, which is not at all uncommon, particularly in anemic patients.

The spots are best seen by holding the cheek away from the teeth with a wide looped tongue-blade. In other cases they are very distinct and numerous, spreading over the entire mucous membrane and resembling thrush. They are often found upon the inner surface of the lower lip. The spots increase in size and number until the skin rash erupts and then, within a day or two, entirely disappear. The little white heads first fall away, sometimes leaving hemorrhagic spots, and, later the red base gradually fades.

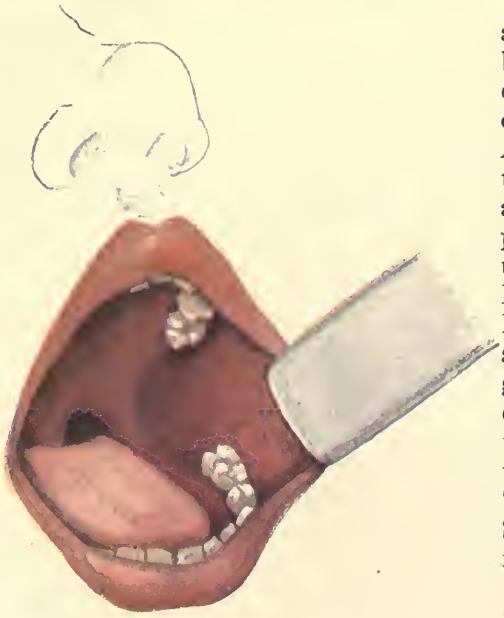


FIG. 157.—Koplik's spots. Above the lower molars, under the tongue blade, the five whitish spots with red areola (actually smaller).

Occasionally Koplik's sign appears as early as four days before the exanthem; and in one occasion it has been seen on the fifth preceding day. As evidence they are extremely important, since they attend no other disease and permit a positive diagnosis upon their presence alone. However, the recognition of Koplik's sign is not, in itself, alway easy. Bright daylight, good eyesight and careful observation are requisite for the discovery of the spots, which are few and small. Thrush is easily excluded. Small crumbs of toast or minute particles of curd may cause some confusion, but these are easily wiped away. If the patient is seen one or two days before the appearance of the exanthem the sign is seen in nearly all cases. If the physician is not called until the rash has appeared these spots may have passed away. They are most likely to be absent in infants, in cachectic patients and in those persons in whom the affection of the mucous membranes is slight.

The true enanthem in the mouth appears a day or two before the skin eruption and usually later than the Koplik's spots. It is a macular eruption on the mucous membranes analogous to the later skin eruption. In small star-shaped, red spots, which may be as large as a lentil, it appears sprinkled over the mucosa of the soft and hard palate and the epiglottis. This enanthem, however, is not so distinct nor so characteristic as Koplik's sign; and in comparison with it, has lost value as a means of determining an early diagnosis.

The Exanthematous or Florid Stage.—All the initial symptoms usually grow more severe on the day preceding the appearance of the exanthem. The fever which prior to this time may have shown a characteristic fall, rises high and the reddening and swelling of the conjunctiva, the photophobia and the coryza become more intense. The cough is hoarser and more annoying. The general well-being is markedly disturbed. The physician called in this stage catching the suspicion of measles from the attendant circumstances, usually finds on inspection the first indications of a rash in the form of small red spots, commonly located about the ears, on the face (Fig. 158) the neck or the scalp. From these points the rash spreads rapidly to the back, the trunk and later to the upper arms and finally to the legs. By about the second day it covers the entire body and in still another day is fully developed. The sight of the entire body covered with these bright red spots is an impressive picture.

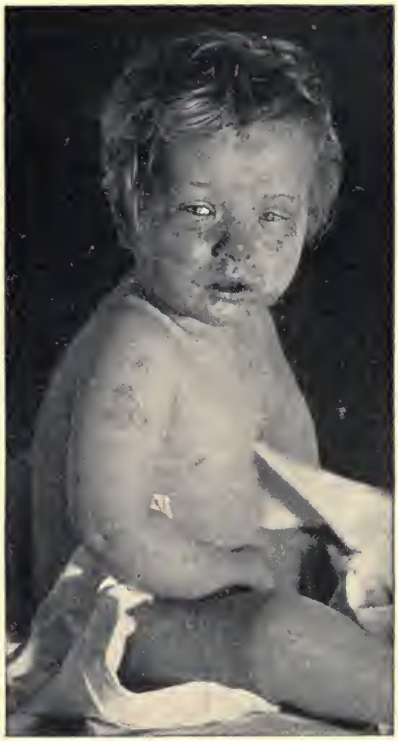


Fig. 158.—Beginning measles eruption in the face, which with the conjunctivæ and photophobia gives a typical physiognomy.

Each individual spot begins as a small follicular elevation rapidly enlarging to the size of a pinhead or a pea. It may be flattened at first, but commonly it is raised from the very beginning and is very soon marked by a small but prominent papilla at its centre. This central papilla corresponds to a sebaceous gland or a hair follicle. The color is at first a bright red, gradually deepening to a flaming red. As the spots enlarge they assume an irregular star-shaped outline and are uniformly raised above the level of the skin. Sometimes a small vesicle appears at the papular centre of the spot. The prominence is distinctly palpable and may be seen if the light falls across it at a tangent. These minute papular elevations at the centre are characteristic and two or three of them may be found on the larger spots.

The early eruption is scant and the individual spots are small but they increase rapidly in size while new ones continually crop out between them. In many parts of the body they become confluent and cover large areas of surface so that, at times the clear skin only forms a few small intervening islands (Fig. 159). The face, the trunk and the back are most frequently the seat of such a diffuse eruption. In nervous patients the rash may cause severe irritation. When it is fully developed and has reached its greatest

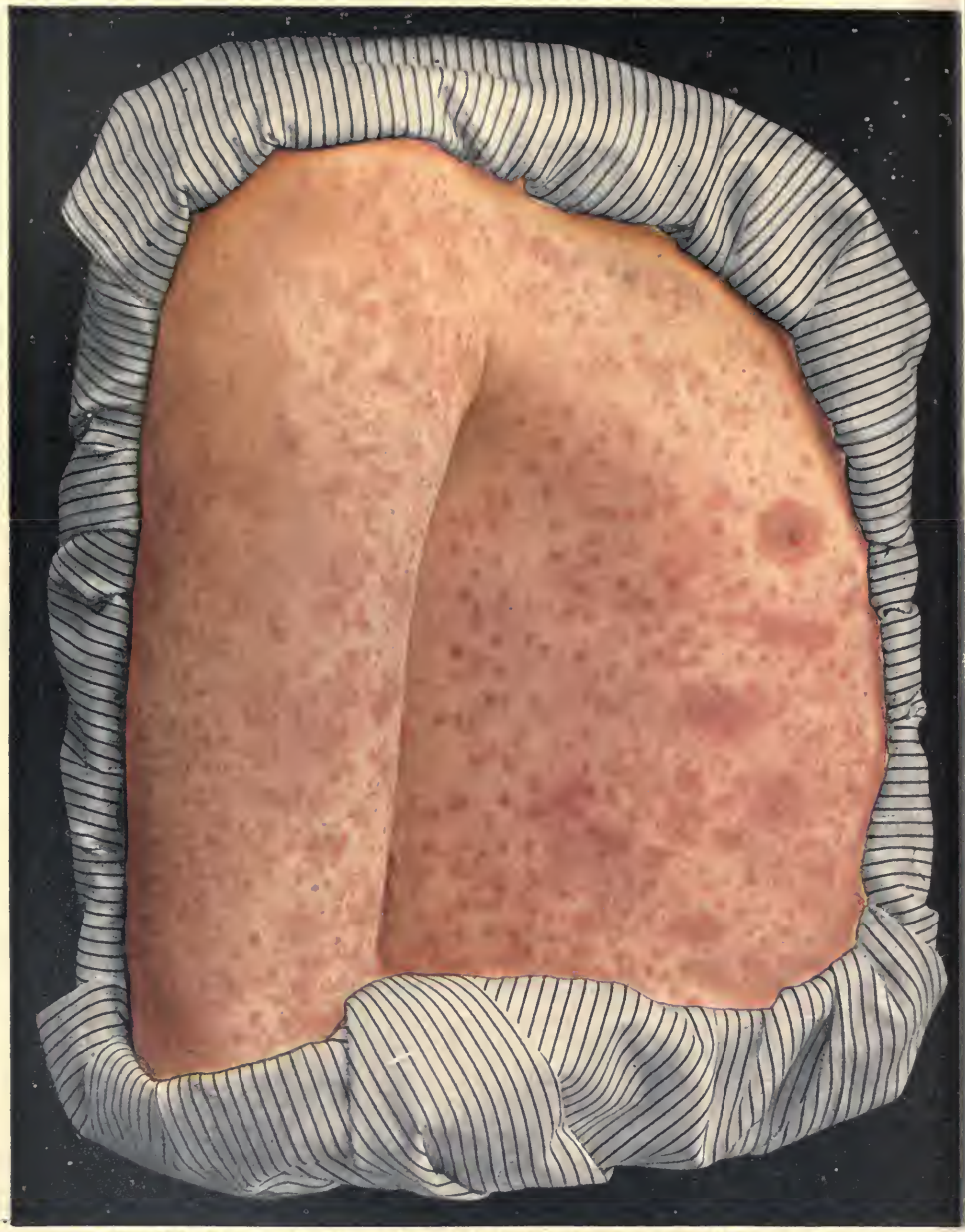


FIG. 159.—The exanthem of measles at the beginning, the separate eruptions are still small.

intensity, it does not continue for long in full bloom. After a very few days it begins to abate over the upper parts of the body and recedes rapidly in the order of its appearance. The recession usually takes about two days, so

that it disappears within four or five days of its outbreak. The early eruption usually disappears completely upon pressure, but in that of later date a slight discoloration remains in consequence of the fact, that some coloring matter has escaped from the blood. This pigmentation becomes more and more distinct especially in robust, highly vascular individuals. It remains for ten or even twenty days after the rash has disappeared. A delayed diagnosis of measles may often be made from this peculiarity alone.

A fine desquamation of the skin is observed as soon as the rash begins to pale. Over the trunk and the extremities this desquamation is so slight and so minute that it is often overlooked, so that one is hardly justified in terming it a fourth stage of the disease. On the face and occasionally on other parts of the body it is often more apparent and bran-like in appearance. The coarse lamellated desquamation of scarlet fever, particularly on the hands and feet, is never found in measles.

The *temperature curve* in uncomplicated measles is often characteristic. The peculiar drop occurring one or two days before the beginning of the exanthem has already been mentioned. As the rash appears the temperature rises quite high and reaches its maximum within twenty-four hours. It usually remains at this height for another day until the rash is fully developed and has reached its most active stage, falling by crisis to the normal level in a day or two (Fig. 160). Even in mild cases the temperature rises comparatively high; 39°-40° C. (102°-104° F.) is common and an increase to 40° or 41° C. (104°-105° F.) is not uncommon. If during the stage of the exanthem the fever remains high for more than four days complications should always be suspected. The temperature curve described is the rule, but variations frequently occur without indicating any disturbance in the normal course of the disease. Thus the temperature may fall by lysis during the height of the exanthem; but two distinct rises may almost always be recognized, the one in the prodromal period and the second during the first or second day of the exanthem.

During the florid stage, the general health of the patient is usually markedly disturbed and he is seriously troubled by cough, photophobia and headache. Delirium may appear with the development of high fever. The appetite is completely lost and even fluids are obstinately refused.

The conjunctivitis increases during the florid exanthematous stage. The lids are swollen and the free secretion becomes purulent and causes agglutination of the lids in the morning. Inspection of the bright red conjunctiva inflamed to the edge of the cornea is more difficult because of the severe photophobia.

The rhinitis also increases. Severe swelling of the nasal mucosa makes breathing difficult. The purulent secretion from the nose erodes the upper lip. Epistaxis occurs from time to time.

Otitis media frequently accompanies these conditions. It arises from the passage of the inflammation through the Eustachian tube. Catarrhal otitis media is indeed a frequent finding and seldom gives symptoms, but the purulent form is also common in younger children and particularly in those with adenoids.

The tongue is dry and heavily coated. The pharynx, tonsils and palate are of a bright red hue and over the latter the enanthem may still be recognized. The buccal mucous membrane is dull, no longer glistening and often still shows the Koplik's spots on the first and even the second day. A white pasty deposit is often found on the gums and is easily removed. This is found also in other severe infectious diseases but is never so marked as it is in measles. The lips become dry, chapped, slightly fissured and painful, a condition which may embarrass the opening of the mouth and interfere with feeding.

The early and annoying dry cough often ceases suddenly upon the appearance of the rash, giving the impression, that the hyperemia of the deeper mucous membranes has been relieved by the development of the skin eruption. Symptoms of laryngeal stenosis may abate in a similar fashion.

The lungs usually seem normal on percussion and auscultation, but

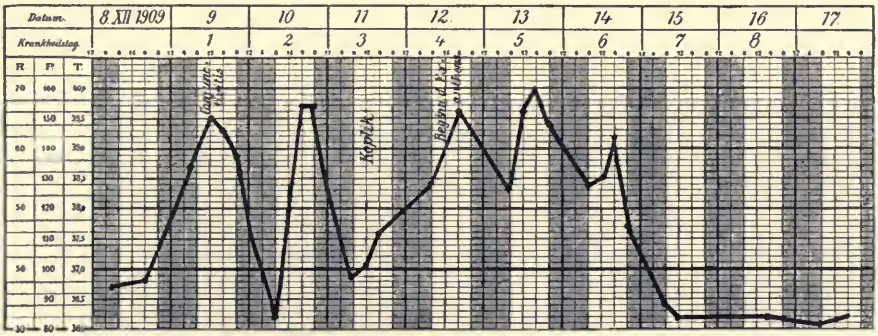


FIG. 160.—Typical temperature curve in a mild case of measles, four-year-old girl.

quite frequently medium and fine moist râles are heard. If there is a marked degree of bronchitis the respiration is visibly difficult and labored.

The heart and the blood-vessels commonly show nothing abnormal. The pulse is rapid in correspondence with the fever. In uncomplicated cases in young children its frequency often runs to 160 or 180.

The blood in the early part of the incubation period shows an increase of leucocytes. During its later days there is some leucopenia (Hecker) resulting chiefly from a decrease of lymphocytes.

This leucopenia is most distinct during the exanthem. At this time the eosinophiles disappear.

The kidneys are not affected in ordinarily mild cases. During the high fever a transient albuminuria is often found. Severe cases occasionally develop acute nephritis at the height of the disease. This quickly heals. If nephritis appears during convalescence, in the third or fourth week it probably always means that scarlet fever has been mistaken for measles. A distinct diazo-reaction is invariably present in the urine during the florid stage. An intravenous injection of this urine into guinea pigs shows that it is toxic.

The stools, especially in the young are often diarrhœic at the beginning

of the attack and this weakens the patient. During hot seasons and in certain epidemics the intestinal tract is sometimes seriously affected. In young children colitic symptoms not infrequently develop, and are extremely obstinate, causing frequent slimy stools, which may induce exhaustion and death. In these instances the intestinal mucosa shows marked follicular swelling and even extensive ulceration.

The palpable lymph nodes are always slightly enlarged. In severe attacks occurring in exudative or tuberculous patients the swelling of the cervical nodes is often very great. The spleen commonly shows no demonstrable enlargement.

The Stage of Convalescence.—With the fall of fever all the general symptoms subside rapidly. The clinical condition changes completely within a day or two. The appetite returns and sleep becomes normal. The cough becomes less frequent and looser. The inflammatory changes of the eyes, nose, mouth, lips and bronchial tubes improve more gradually, but nevertheless, so rapidly that ordinary cases are fully convalescent within a week after the appearance of the eruption and are entirely normal eight or ten days later. The pigmentation of the skin over the eruptive areas, a slight pallor, a lessening of the usual turgor and at times, a slight catarrh of the affected mucous membranes may remain for a short time longer.

Unusual Course and Complications.—A relatively large number of cases of measles conform to the above outline and follow such a definite course. Anomalies, of course, are much less frequent than is the occurrence of complications, chiefly affecting the respiratory tract.

Cases are seen so light that the prodromes are hardly noticeable and the disease is observed by the parents only when the rash appears. Again the exanthematous period may often pass in two or three days without marked fever and without affecting materially the general condition of the child.

In rare instances a severe toxic form of measles is seen. At the very onset of the exanthematous stage, the patient becomes extremely ill, with high fever, somnolence, small and very rapid pulse, and unusually severe catarrhal symptoms which are followed by an intense exanthem the temperature going to 40°-42° C. (104°-106° F.) and death, preceded by the signs of asthenia, ensuing in a few days. This fatal course is most frequently seen among children with a status lymphaticus. Occasionally the discovery of streptococci in the blood shows, that one has had to deal with a septic measles. The mildness or severity of individual cases is frequently determined by the nature of a given epidemic.

In infancy, measles is often extraordinarily mild. The fever and the catarrh are insignificant; the exanthem remains light and pale and the recognition of the disease may be very difficult and the more so since Koplik's sign is often very slight or entirely absent at this age.

Adults are usually more severely affected by measles than are children although death is rare under ordinary circumstances.

Among sick and tuberculous children the course of measles is often very

severe. The skin eruption may remain slight, but pulmonary complications develop very readily or a new spread of the tuberculous infection results in death.

Certain deviations with respect to individual symptoms may be noted. Fever may remain very low and the characteristic curve may be entirely lacking. Fall of temperature by lysis in the final stages is common even in the usual course of the disease. One should always be prepared for complications when the temperature does not fall with the disappearance of the eruption, or in the event that it rises again after a few days.

Afebrile measles is a great rarity, but cases with very slight and transient fever are common. They are often seen among cachectic infants. The exanthem and the catarrhal symptoms may be so slight as to be entirely overlooked.

The exanthem shows numerous variations, even though it be so typical and so well developed in the great majority of cases that the laity can make a correct diagnosis. In extremely rare instances the eruption of measles is preceded by a transient scarlet rash.

There is much difference of opinion as to whether a form of measles occurs without exanthem in which only the fever and the catarrhal symptoms appear. Since the incubation period is a very constant one (fourteen days) it would seem that this question should be easily settled in private practice. Most authors admit that they have never seen a case of measles without exanthem. Through many years of private practice and in spite of special pains in the observation of hundreds of cases, the writer has never noted such a case, the diagnosis of which could be clearly made by the aid of Koplik's spots. Recently, however, he has seen one instance occurring in the midst of a household epidemic. It occurred in a feeble infant of four and a half months of age. The patient became ill after the usual incubative interval with fever, catarrh and distinct Koplik's spots, but it never showed even a trace of an exanthem.

Cases in which the exanthem is slight, appearing in but a few small areas, and disappearing completely within a day or two are frequently encountered. Such cases are less common in the very mild forms of the disease than among cachectic children or those who are ill with other diseases. An incomplete and weak exanthem is seen also in cases, which develop severe complications such as bronchiolitis or pneumonia in the prodromal stage. If pneumonia appears at the outbreak of the skin eruption, the development of the exanthem may be arrested and the child becomes cyanotic and pale, so that the rash can hardly be seen. This is a bad indication. It has given the laity its general fear of the "going in" of the measles. Similarly it appears, that the development of the rash in other than its regular order of appearance, or its incompletion is an unfavorable sign.

Even cases of measles which from the first pursue an entirely favorable course, may either show a localized or a general eruption of a hemorrhagic quality. This has no serious significance. The writer has seen a severe hemorrhagic exanthem in three children of one family, all having but a

mild form of the disease. When the eruption is distinctly cyanotic the condition is more grave, since it indicates an impaired circulation. The rare cases in which hemorrhages into the skin occur as a result of sepsis and in which hemorrhages from the mucous membranes of the nose, intestinal tract, etc., also appear must be differentiated carefully from the ordinary hemorrhagic exanthem. Occasionally one may have to deal with a general sepsis in which, from the very first, an appearance of a macular exanthem simulates measles.

A preëxisting eczema of the face will often become a brighter red and more macular two or three days before the appearance of the rash. Very frequently one finds eczema aggravated or redeveloped by measles, or again, suppressed during the passing eruption.

The character of the exanthem of measles often presents variations that are important from a diagnostic standpoint. The efflorescence may from the first be so distinctly papular as to make one think of chicken-pox. At other times they do not rise above the level of the skin. This is especially true in the anemic. Occasionally they are vesicular or urticarial. Gangrene of the skin has been known to follow the exanthem. The writer has seen one instance in which the entire musculature of the upper arm was exposed in consequence.

A recrudescence of measles must be mentioned as of very rare occurrence appearing after an interval varying from two to eight weeks.

The involvement of the mucous membranes varies greatly in intensity. In the eye all degrees of inflammation are seen from a slight conjunctivitis to a severe blennorrhea, which may lead to a clouding of the cornea and destruction of the eye. Frequently a severe blepharitis occurs which with the conjunctivitis may require weeks for recovery. With a marked rhinitis an erosion of the nostrils and diphtheroid ulcerations of the upper lip are not uncommon. At the beginning of the prodromal stage a catarrhal or lacunar angina sometimes develops and if the usual symptoms in the mucous membranes are not apparent, it may cause some confusion until the rash appears.

The inflammation of the mucosa of the mouth may lead to the formation of deep and obstinate aphthous sores or ulcerations. These may occur also on the lips giving great difficulty in opening the mouth. A comparatively large percentage of cases of noma, a rare condition, appears in measles patients especially if these are of an asthenic type. An unusual localization of noma is on the vulva which, in ordinary cases shows only a moderate inflammation with a thin purulent secretion.

Laryngitis may, at times, become very severe, even in the enanthematous stage, causing intense hoarseness, attacks of pseudocroup and persistent stenosis. It may so closely simulate true croup, that the patient may be placed in an isolation ward for diphtheria. The laryngeal symptoms generally disappear, however, so soon as the rash develops. The more severe grades of inflammation occasionally cause ulcerations of the vocal chords and adjacent surfaces. This may result in hoarseness, and even

aphonia, with a moderate degree of stenosis, persisting for many months and yielding only to energetic treatment.

Bronchitis may become very diffuse even at an early stage and may give rise to many medium and fine râles and to dyspnœa. More commonly however, a widespread bronchitis is observed in the exanthematous stage. Very often it is of a capillary type and affects the posterior lung areas. At times it involves the entire lung and frequently causes death.

Grave forms of bronchitis and pneumonia are the more usual complications of measles. The younger the patient the more liable are they to occur. After the third or fourth year they become much less common. Weak and rickitic children are the easiest victims.

Pneumonia, the most frequent and the most fatal complication of measles may arise from an initial bronchitis. In rare instances it develops even in the prodromal stage; more frequently it coincides with the onset of the rash; and still oftener it follows a few days later. The earlier its development the more severe it is. The more incomplete the eruption or the more rapid the disappearance of the rash before it reaches the florid stage, the greater the likelihood of this complication. Usually it is of the broncho-pneumonia type. Often it develops in this form so rapidly and so massively, that if an entire lobe is involved the clinical picture resembles very closely that of lobar pneumonia, excepting that its course is more protracted and that the critical fall of temperature is wanting. In other cases it develops insidiously and presents no definite findings for several days. The reappearance of fever or an arrest of the fall of temperature with the occurrence of dyspnœa alone points suspiciously in this direction (Fig. 161).

Pneumonia in measles frequently terminates fatally. Although recovery sometimes is seen even after the disease has dragged along for weeks. A peculiar form, happily uncommon, causes a necrotic destruction of the affected lung tissue (Heubner).

The frequency of this complication varies within wide limits in different epidemics and with some relation to the season of the year. The fact, especially emphasized by French authors, that the pneumonia of measles is often contagious, is remarkable. One patient in a measles ward contracting pneumonia may convey the latter disease to other patients in the same ward. This, in itself, shows how very liable cases of measles are to secondary infections. The contagious element, however, in these cases, is not the pneumonia itself, but rather the causative secondary bronchitis, although the infective organism is either the pneumococcus or the streptococcus. The author has observed, that with the coincidence of epidemics of both measles and la grippe, the complicating pneumonia of measles is unusually common. The development of measles with pertussis is especially unfavorable since it increases the liability to pulmonary complications.

Pleurisy is frequently associated with broncho-pneumonia. It is usually fibrinous or seropurulent and readily escapes observation often being discovered only at autopsy. Large exudates are unusual and when they occur are almost always purulent and of streptococcic type, and must be drained by rib resection.

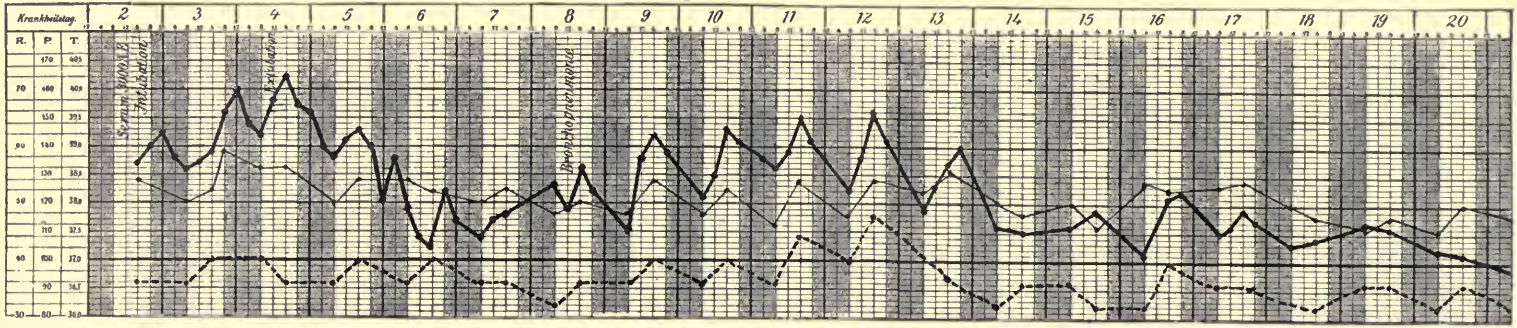


FIG. 161—Broncho-pneumonia following measles, two and one-half-year-old boy.

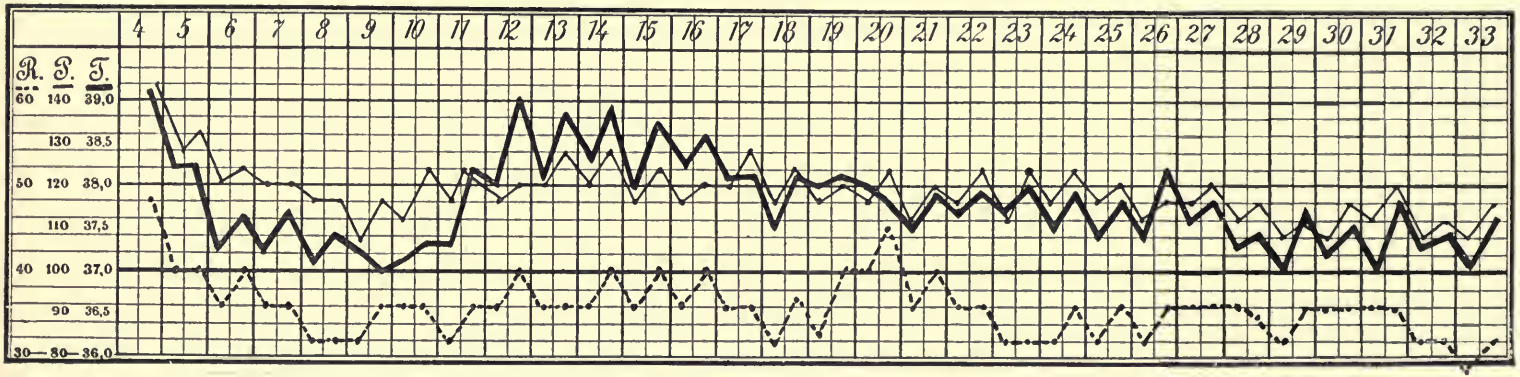


FIG. 162—Measles followed by tuberculosis of the bronchial lymph nodes which causes fever, gradually falling after the twelfth day. Four-year-old girl. This case showed a typical shadow in the Roentgenogram.

Otitis media, the frequency of which has already been noted, often causes rupture of the tympanic membrane, with purulent discharge. It is much more benign than that of scarlet fever and usually heals without causing permanent injury. Inflammation of the mastoid cells and fatal sinus thrombosis are uncommon. Paracentesis cannot prevent these complications and it is of merely palliative value in the event of severe pain. Proliferation of adenoid tissue as a result of measles is frequent. The circulatory system is much less seriously affected than in scarlet fever. The development of a valvular lesion as the result of endocarditis or pericarditis is of rare occurrence.

The nervous system is markedly affected in severe cases of measles. Somnolence and delirium are not uncommon during the period of high fever. General convulsions unassociated with spasmophilia, are less frequent. Occurring during the height of the eruption they tend to an unfavorable prognosis. Very occasionally a purulent meningitis ensues, but more frequently a tuberculous meningitis is seen during convalescence or even develops months later. A few cases of neuritic paralysis have been reported.

Aside from the pulmonary complications of measles the coincident appearance of diphtheria gives the greatest concern to the physician. The mucous membranes of the respiratory tract in measles are as susceptible to the diphtheria bacillus as they are to every other infecting organism. During diphtheria epidemics this complication is especially to be dreaded but it is quite common even in ordinary times. It is the more important that the physician should recognize this, because the diphtheritic invasion often occurs not in the pharynx, but in the larynx, and the bronchi, causing "measles croup." The occurrence, on the other hand, of hoarseness, and laryngeal stenosis in uncomplicated measles may readily obscure the diagnosis. The differentiation of these symptoms as mere manifestations of measles, from the similar consequences of a superimposed diphtheria offers great difficulty, when there is no membrane in the throat. To await the report of bacteriologic findings consumes time valuable for treatment.

Complicating measles diphtheria is peculiar in the rapidity of its spread from the larynx to the smaller bronchi causing death in a short time. If intense hoarseness alone is present and if this is relieved, when the rash appears there is no great cause for anxiety since this symptom is very common in early measles, while complicating diphtheria usually appears at a later period. If, however, there is marked aphonia and laryngeal stenosis growing more serious during the florid stage, it is safe to conclude that diphtheria is present even though the pharynx is entirely clear. Indeed, laryngeal diphtheria should always be suspected when increasing hoarseness and stenosis appear after the eruption of the exanthem. In such cases a large dose (20,000 I.U.) of diphtheria antitoxin should be given immediately and repeated in twenty-four hours. In fact, the dose of the antitoxin should be larger than would be given in primary diphtheria because experience shows that patients with measles have a remarkably low resistance to diphtheria. Even in later childhood, death may result in two or three days from the development of the stenosis. To wait until the stenosis is

grave and until all diagnostic doubts are removed is to be too late. Intubation and tracheotomy are useless then because membrane formation has occurred in the bronchioles. In fact intubation and tracheotomy should be delayed as long as possible, since the mucous membranes are extremely vulnerable during measles and tend to necrosis: Moreover with a severe grade of inflammation of the larynx and trachea in patients with measles a fibrinous exudate may develop, which is not diphtheritic, but due to streptococcic or diplococcic infection. To be safe, however, these cases may wisely be given antitoxin.

The lowered resistance of the organism during measles also becomes evident in the frequent liability to tuberculosis as a sequel. The fact, established by Preisich and von Pirquet, that the cutaneous tuberculin reaction is temporarily lacking during the florid stage of the eruption very probably means, that the organism is without protection against tubercle bacilli and their toxins at this time. So, also, it satisfactorily explains the fact that an inactive tuberculous lesion often becomes active during measles. As a result, a demonstrable tuberculosis of the bronchial nodes, or at the hilus (Fig. 162) or even a miliary dissemination may occur. A pre-existing active tuberculosis is often aggravated. It may also be understood how it is that with a lymphatic diathesis after measles, signs of scrofula, phlyctenule, enlargement of the lymph nodes and cutaneous tuberculides appear since scrofula represents the reaction of a lymphatic diathesis to tuberculosis. An unexplained continuous fever following measles must arouse suspicion at once, that a quiescent tuberculous process has been stirred to activity. The frequently protracted broncho-pneumonias of measles are, as a rule wrongfully suspected of being of a tuberculous character.

The diagnosis of measles is readily made in the great majority of cases and offers fewer difficulties than in scarlet fever. The febrile prodromal stage, the catarrh of the upper air passages and conjunctiva, the Koplik's spots and the typical exanthem hardly permit an error in the great majority of cases.

The eruption alone cannot be considered conclusive evidence since many other diseases cause similar skin conditions which must be differentially excluded.

A severe papular eruption may resemble variola for a day or two, but after that these two exanthemata develop very differently. In small-pox, moreover, the temperature falls when the eruption appears; in measles, on the contrary it rises. Scarlet fever hardly ever causes any confusion, since the eruption is much finer. Confluent measles may seem to resemble scarlet fever upon superficial examination but areas can always be found especially on the arms and legs, where the large spotted measles rash persists. Furthermore, scarlet fever has no catarrhal prodromata but instead exhibits a severe angina, a strawberry tongue, etc.

Rubella is very similar to rubeola. The eruption, however, is paler and more minute, the Koplik's spots are absent and the catarrh and fever are slight. Infectious erythema is less likely to cause confusion. In this disorder, also the catarrhal stage is lacking and the exanthem is character-

istically confluent on the flexor surfaces of the arms. In sepsis multiform eruptions, among them sometimes a rash resembling measles, may appear, but associated symptoms make the differentiation easy. Roseola syphilitica, if the eruption is sudden and severe may temporarily resemble measles. In diseases of the la grippe order, rubeoloid rashes occasionally occur and may lead to error on account of the coincident presence of catarrhal conditions. The exanthem, however, is more transient and irregular than in measles and Koplik's sign is never present.

Following vaccination and in the disturbances of nutrition of infancy erythemata resembling measles are not infrequently seen, but these do not affect the mucous membranes. Toxic erythemata also occurring after serum injection or following the use of certain drugs are very likely to lead to temporary error. The question they raise however, is soon determined by the variability and atypical forms of these eruptions, by the fact, that urticarial and scarlatinoid types will develop side by side, and by the rarity of their spread over the entire body. In doubtful cases the presence or absence of Koplik's sign characteristic only of measles, the recognition of the early febrile stage, the occurrence of the catarrhal prodromata and conjunctivitis, the regular spread of the rash, the diazo-reaction, and the blood findings (leucopenia suggesting measles or the serum exanthemata, a neutrophilic leucocytosis and eosinophilia favoring scarlet fever) will have due weight. The diagnosis may be assisted by the definite incubation period and by the knowledge of other neighborhood cases. The absence of any possible source of infection is also to be considered.

The prognosis may be made with some degree of assurance at the beginning of the attack since the course of measles is almost always the classical one. The insidious, the unsuspected element so usual in scarlet fever is lacking in measles. In strong healthy children of over three years of age a favorable prognosis may be almost invariably made. The disease holds more danger it is true, for younger children and especially for the feeble and rickitic, who so often die of broncho-pneumonia. A coincident infection with diphtheria is dangerous at any age.

During the convalescence from measles of children who have suffered with tuberculosis, in whom nothing more than a positive tuberculin reaction gives evidence of infection, the recrudescence of tuberculous disease or the possible development of miliary tuberculosis is to be reckoned with.

An abortive or cyanosed eruption suggests an unfavorable prognosis and so does the coincidence of severe bronchitis. A favorable outcome is prejudiced by cold weather, by bad environment, or by want of care.

Prophylaxis should attempt to guard children under three or four years of age from contagion. If the patient is isolated at the beginning of the prodromal stage, it is often possible to prevent the spread of the contagion to other children in the family. Isolation after the eruption has appeared is always too late. Since every person must have measles at one time or another, it seems proper not to protect strong healthy children of four years or more, from the possibility of contagion. The weak, the sickly and especially the tuberculous should always be shielded. Day nurseries and kin-

dergartens often form dangerous nests of infection during an epidemic and should be avoided and closed at such a time. The closing of the public schools is not justified. Only in exceptional instances does the summer vacation interrupt the course of an epidemic. This is shown by the curve of Basel for the year 1884. (See Fig. 156.) During the prevalence of the disease very young children should be kept away from public places.

The disinfection of living rooms and their contents after measles is superfluous and useful only for the prevention of secondary infections, as pneumonia, etc. Even a room occupied but a day before by a measles patient, may be turned over to a person who has not had the disease without any danger.

Treatment of mild cases should be expectant. Upon the first suspicion of the disease, the patient should be put to bed in an airy, warm room which proper provision for atmospheric moisture is made. Strong light should be avoided, but semi-darkness is necessary only in event of severe photophobia. The complete darkness, formerly much in vogue, has no advantages and interferes with sleep at night. During the febrile period the diet should be in fluid form and readily digestible. In infancy, it should consist of milk and gruels. If there is any tendency to diarrhœa, the milk should be reduced and the calories supplied by the addition of dextri-maltose. Older children may be allowed milk, milk toast, barley soup, gruels, fruit-juices, etc. In obstinate anorexia, which is so frequently observed, it will suffice to give large quantities of water. Before the appearance of the eruption large draughts of hot, dilute, sweetened tea may be given, while the patient is kept well covered in bed, with the intent of favoring the full eruption of the exanthem. If the rash is delayed a hot bath is to be recommended. Ordinarily no active measures are required so far as the fever is concerned. In extreme pyrexia, cold compresses or an ice-cap to the head and light bed covering will suffice. The laity is quite justified in its fear of cooling measures, which if not properly controlled may certainly do harm. Considering the short duration of the fever they are not indispensible.

The mucous membranes should be the chief concern. The exercise of extreme cleanliness, the avoidance of unnecessary attendants and especially of contact of those who suffer with catarrhal affections, are matters of prime importance. Patients with measles who develop grave bronchitis or broncho-pneumonia, should not be kept in the same room with other patients who are free from these complications.

A dilute solution of acetate of lead is often useful in severe conjunctivitis. (A teaspoonful of lead acetate to one litre of cold water.) Applications of it should be changed every few minutes for a period of one-half hour, several times a day. The purulent secretion should be removed from the lids by a cotton probang moistened in a tepid boric acid solution. The nares should be frequently and thoroughly cleansed of all secretion. Lanolin or a one per cent. ammoniated mercury ointment may be used in the nostrils, when the inflammation is marked. Children who are old enough should rinse the mouth frequently with warm water in which a little borax has been dissolved. With younger children it must suffice to give them

water to drink after each feeding. In severe stomatitis a spray of hydrogen peroxide (2 per cent.) is useful. Aphthæ and ulcers may be painted with a 1 per cent. solution of potassium permanganate and later with a 2 per cent. solution of silver nitrate or with a dusting powder of iodoform.

If the cough is very annoying but the bronchi are free from excessive mucus a desirable quieting effect may be obtained with codein. For children of one year codein sulphate in the proportion of gram 0.03 to 100 c. c. of water and for children of five years in the proportion of gm. 0.1 (grs. ii) to a similar quantity of water, may be given in doses of 5-10 c. c. The croup kettle gives great relief in cases of hoarseness, pseudocroup, and stenosis. By this or other means a thorough moistening of the air of the apartment should be secured. Children of sufficient age may be allowed to inhale salt vapor. In laryngitis cold applications about the neck may prove useful and sometimes stenosis may be relieved by leeches applied over the sternal notch. If laryngeal diphtheria is even suspected an injection of 4000 units of serum should be given at once.

During the stage of the exanthem mild bronchitis requires no treatment. The more severe grades of bronchitis with fine râles or a broncho-pneumonia should be treated in the usual way.

In measles the application of cold packs or cold baths requires careful consideration. The bad effects of this practice are seen, especially in younger children whenever the skin fails of brisk reaction both as to warmth and color. Cold applications in measles are sufficiently unpopular among the laity, due to the idea that the rash may "strike inward" a supposition not, without a measure of truth. Warm baths 34°-32° C. (95°-90° F.) on the other hand, are without danger. If the skin is cyanotic, the extremities cold in spite of the high fever, and the rash but poorly developed a short hot bath at 37° C. (98.6° F.), rapidly raised to 40°-41° C. (104°-105° F.), often brings the rash out and improves the circulation. A similar bath followed by a cold douche, or Heubner's mustard pack (see page 372) are indicated in bronchiolitis or broncho-pneumonia. Under these conditions such stimulants as caffein and camphor may be necessary to combat cardiac weakness and vasomotor paralysis.

It is best to keep every child who has even an ordinary attack of measles in bed for eight days after the fall of the fever. After this interval the child may be permitted to sit up and very soon or within a week or so, according to circumstances, season, and the age of the child to go out of doors. Should convalescence be delayed, or the catarrhal conditions prove obstinate a visit to the country may be indicated. Such an expedient is particularly desirable when variations of temperature, with or without a positive tuberculin reaction, lead one to suspect the reactivation of an old tuberculous focus.

RUBELLA

(German Measles, Röteln)

Although the distinctive character of rubella has been discussed for several centuries, it has been generally recognized as a specific infection for

some thirty years and its identity is now denied only by those who have never seen an epidemic.

Rubella may be defined as an extremely benign contagious disease characterized by a mild measles-like eruption upon the appearance of which the initial catarrhal symptoms, fever and other general symptoms disappear.

The causative organism and its port of entry are still unknown. The transmission of the disease is usually direct from one person to another and only upon close contact. It is possible that in exceptional cases, the disease may be carried by a third person or by objects handled. Sporadic cases are not common. The disease usually appears as a local epidemic and most commonly in the spring. Such an epidemic frequently continues for several months without attaining any great severity. It may put in an appearance at irregular intervals after an absence of many years. The writer has had the opportunity to observe two such epidemics.

Individual predisposition is much less marked than in measles. If however, the disease develops in an institution, school or asylum, as it often does, one-half or more of the inmates are commonly attacked.

Children are especially predisposed between the ages of three and twelve and most markedly within school age. The disease is often to be seen however, in late infancy. It is even said to occur congenitally by transmission from the affected mother.

It is contagious even at the close of the incubation period, the contagion reaching its height at the onset of the eruption and disappearing as the eruption fades. The virus is of brief viability.

The incubation period covers usually from seventeen to twenty-one days and rarely falls within two weeks. The author has usually seen secondary cases develop within infected families in from eighteen days to three weeks.

Prodromal symptoms are usually lacking. Occasionally the appearance of the rash may be preceded by general malaise, sore throat, slightly reddened conjunctivæ, coryza, and even slight rises of temperature. These symptoms are so slight, however, that the physician only hears about them when he is called in on account of the rash. Most cases are so mild that they are seen only in family practice and so prominent a clinician as Henoch had never met definite epidemic cases.

Symptoms.—The exanthem is usually the first noticeable sign of the disease. It first appears on the bridge of the nose, around the ears, and over the forehead and cheeks and on the hairy scalp. It spreads rapidly and may cover the entire body in half a day. Small flat or slightly raised light red spots appear, the size of a pinhead and rapidly increase to the size of a lentil. They are clearly circumscribed and are usually round or oval in form. Their outline is not so jagged and irregular, nor are they so large or of so dark a red nor so prominently raised as the papules of measles. By way of further comparison the individual spots are all of the same size; they are equally distributed and are hardly ever confluent being separated by much clear space. In the latter respect they have a similarity to scarlet fever, although the spots are larger than in that disease. Upon

the cheek only the eruption sometimes appears in a fine network. At the beginning the face looks congested and remarkably red, so that during an epidemic the teacher is often able to make the diagnosis and send the child home.

The eruption is often so pale that it is overlooked. The individual spots will at first disappear upon pressure, but later they will occasionally leave slight pigmentation, which is never so marked as it is in measles. Sometimes a fine dust-like desquamation of the epidermis is seen.

On the body, the eruption is often most distinct at any point on which the clothing rubs. Generally speaking the face, back and exterior surfaces of the limbs are most markedly affected. The eruption is rarely distributed uniformly over all parts of the body at one time. It appears usually in crops, the eruption upon the head having begun to fade by the time it develops upon the trunk and this, in turn, fading as the extremities become involved. Frequently large areas of the body are spared. The rash, in any one spot, only continues in full bloom for a day or two. It disappears rapidly, entirely passing away in from two to four days. The author's observations suggest that successive relapses occasionally develop, even into the second week. Thus it is seen that rubella most closely resembles the eruption of measles excepting that it is paler and less dense. Sometimes the rash is very indistinct, the margins of the spots leaving a washed-out appearance and being connected with each other by small bridges give the skin a mottled appearance. Very rarely the rash is so fine as to resemble scarlet fever, showing this quality in certain areas only, as over the chest or upon the thighs. In the course of epidemics the author has observed that no cases have occurred in which the entire exanthem was scarlatinal in type. Such cases would appear to be properly classed with the so-called fourth disease. Sometimes the eruption takes on an urticarial form.

The affection of the mucous membranes is unimportant. A slight reddening of the conjunctiva, an insignificant coryza, some sneezing, a mild hyperemia of the pharynx and tonsils, a swelling of the lymph follicles of the soft palate and occasionally fine scattered hemorrhages in the mucous membrane over these areas are observed upon the appearance of the rash, or even a few days before the exanthem develops. These symptoms are so uncertain and so frequently occur *ab initio*, that they have no diagnostic value. It is an important point, however, that Koplik's spots are always absent. In some cases there is slight hoarseness and cough, but bronchial râles are almost always lacking.

The *swelling of the superficial lymph nodes* is an important and a constant symptom. The nodes over the mastoid process and in the occipital and cervical regions, particularly, will usually swell before the exanthem appears, so that adults and children of observing years will complain of their painful and visible swelling some two to four days before the development of the rash and will come to the physician in consequence. These nodes may reach from the size of a bean to that of a hazel-nut. They are at times tender upon pressure and they disappear in a week or two. Frequently the axillary and inguinal nodes are also enlarged.

Recently rubella sine eruptione has been described (Koplik). It has been recognized during epidemics of the disease by the occurrence of swelling in these groups of lymph nodes without other symptoms.

The temperature may show only occasional minor rises during the entire course of the disease. In the prodromal stage it may reach 38°-38.5° C. (100°-101° F.) and during the first day of the eruption even 39° C. (102° F.) but it rarely goes higher. Then it falls rapidly, even though the eruption persists. Many cases run their entire course without fever.

The blood shows no essential changes in the number of leucocytes at the onset of the eruption.

The general health is very slightly affected and in a major number of cases is not disturbed. It may almost be said that the only actual symptom of the disease is the eruption. In fact, the physician sees very few of these cases and these few only because of the fear that they may be measles or scarlet fever. When cases follow one another in the same family the parents consider it unnecessary to call a physician. Very rarely high fever, a marked angina or bronchitis make a more serious clinical picture, a development more frequent among adults than in children.

The lungs, heart, circulation, brain or meninges are not involved in this disease. In exceptional cases a transient nephritis has been observed.

Serious symptoms or complications such as necrotic angina, severe nephritis, marked desquamation of the skin, inflammation of the joints broncho-pneumonia, etc., frequently ascribed to this disease, certainly indicate its confusion with scarlet fever, measles or some other infectious disease.

It follows that the course of rubella is almost without exception mild and of brief duration. It may be considered the most benign of all known infectious diseases.

Diagnosis.—Isolated cases may be very difficult or even impossible of diagnosis. During an epidemic the diagnosis is an easy matter. Added to the peculiar exanthem, the typical swelling of the occipital lymph nodes, the absence of any marked affection of the upper air passages and the negligible disturbance of the general health, assist in the recognition of the disease. As in all the exanthemata, a diagnosis cannot be made from the eruption alone, but all other factors and symptoms must be taken into consideration.

Rubella resembles mild forms of measles very closely and is quite often confused with it. Many physicians formerly regarded the disease as merely an attenuated form of measles. However, the exanthem of measles is usually more distinct, of deeper red, more elevated above the surface of the skin, more pronounced and diffuse, while the attending fever is higher and the involvement of the conjunctivæ and the upper air passages is more marked. The exanthem of measles in feeble and anemic children is often indistinct and undeveloped and therein resembles rubella more nearly than any other condition. The Koplik's spots always definitely suggest measles and contraindicate rubella. Nevertheless doubtful cases always occur, which are differentiated only by the nature of the prevailing epidemic. Strangely enough epidemics of rubella and of measles often follow each other.

If the length of the incubation period can be determined it assists in the differentiation of the two diseases, since that of rubella is at least fourteen days while that of measles is but ten or eleven days. Further, the absence of prodromes suggests rubella. So also, does the fact that the patient has already had measles, or contracts this disease at a later date.

In rubella, the diazo-reaction in the urine is said to be lacking. The von Pirquet cutaneous tuberculin reaction does not fail, as it does in measles.

The characteristic description of the disease should not permit of its very frequent confusion with scarlet fever.

An exanthem resembling rubella occasionally follows an injection of serum or the use of certain drugs or the practice of vaccination. Similar eruptions may occur in the new-born or in infants suffering with disturbances of nutrition, in cases of la grippe and in various other infectious diseases. At times these imitative conditions may demand that all the circumstances surrounding them be taken into consideration (*vide* measles p. 613).

In so benign a disease special prophylaxis is unnecessary. Isolation should be enforced only in cases in which the diagnosis is not clear. Feeble and rickitic infants must be guarded against infection if possible.

Treatment.—No special treatment is required. If there is fever, rest in bed and a liquid diet should be ordered. Children of school age should be kept at home, for the protection of other pupils for eight or ten days; but feeling well and in good weather, they may be allowed to go out of doors.

INFECTIOUS ERYTHEMA

Infectious erythema is a well defined infectious disease, which runs its course without any serious disturbance of the general health. Its most important symptom is a large macular and frequently confluent exanthem covering the face and the extensor surfaces of the limbs. It resembles measles or a multiforme exudative erythema. Many other names, as local rubella, megalerythema epidemicum, etc., have been given to it.

The disease has been described only in recent years. It was first observed, on various occasions, in Gratz and later, in other parts of Germany. An extensive epidemic of it was seen by the author in Basel in the year 1903. Almost all writers now agree that it is disease entity.

Sporadic cases occasionally appear and frequently fail of diagnosis. Commonly, however, the disease occurs in small epidemics confined within an institution or a single school. Spring is the season in which such outbreaks are usually seen. They are said to occur coincidently with measles and rubella.

Most cases develop at an early age. The youngest patient observed was at one year and the oldest at twenty years of age. The method of contagion is not definitely known. Direct transmission seems infrequent although it has certainly been noted (Pfaundler). According to most reports the period of incubation varies from seven to fourteen days. It seems fairly constant, since the writer has seen two children in each of two families and

three children in a third household attacked at the same time without being able in any one instance to trace the source of infection.

Restlessness, general malaise, and slight sore throat occasionally mark the prodromal stage, which, however, is usually absent.

The exanthem is generally the first sign of illness. It appears first and most distinctly on the face and the extremities. On the cheeks large bright red and markedly raised spots which often resemble variola are seen. These enlarge rapidly becoming confluent within a short time. During this development the central portion of these plaques is often flattened and becomes paler. The cheeks show a marked degree of congestion, being very red, hot and infiltrated. A sharp demarcation from the normal skin at the irregular, raised edges of the exanthem, especially upon the chin and about the ears is quite characteristic. The region of the nose and mouth frequently remains clear; the forehead is usually affected but there the rash is lighter in color. Besides the face, the extensor surfaces of the arms from the shoulders to the fingers are most frequently and markedly involved. The rash is also definite over the buttocks and upon the legs, where the selection of the extensor over the flexor surfaces is not so great. The eruption usually spreads symmetrically over both sides of the body. Occasionally, it begins on the limbs, on the shoulders, or the buttocks in the form of raised red spots, which feel hot and spreading become confluent in geographic, crescentic or garland-like figures. The exanthem is exceptionally most marked on the flexor surfaces of the arms, becoming confluent and covering large areas, gradually diminishing toward the extensor surfaces to small rubeolar or urticarial spots. The trunk usually remains free from the rash or shows a slighter, paler and more mottled eruption within the first two or three days.

The rash often takes on a cyanotic or brownish tint. It usually disappears rapidly and occasionally leaves a slight pigmentation. It is followed by no distinct desquamation.

The duration of the eruption is usually about a week. Not infrequently a brief recrudescence is seen in some areas after the initial rash has begun to disappear, which is due to such external causes as perspiration, irritation of the clothing, etc.

During the period of eruption other manifestations always subside and entirely disappear. Frequently there is a complete absence of fever during the entire course of the disease. At times, and especially at the onset of the attack, a subfebrile temperature of 38° to 39° C. (100°-102° F.) is recorded, but even this minor rise is uncommon. General disturbances of any degree of severity hardly ever occur. There may be a measure of restlessness, disturbed sleep, some itching, a painful tension of the skin over the face and occasionally a sore throat. A reddening of the conjunctiva and of the pharyngeal mucosa may be present, the latter resulting in cough. In some instances at the onset a lacunar angina is observed.

A minor enlargement of the lymph nodes of the neck is occasionally seen. There is no record of any definite complications. The disease always

goes on to recovery and leaves no after consequences in its train. That the few fatal cases reported are true examples of this disease is an open question.

A diagnosis is readily made during an epidemic from the peculiar type of the eruption and the areas it characteristically affects. It is very readily confused with measles, but the catarrhal prodromes and the generally diffuse exanthem of the latter should differentiate it. Koplik's spots are never seen in infectious erythema. It has a resemblance to rubella only when the rash upon the face in the latter infection is very definite and confluent. The eruption in rubella is never so pronounced or confluent over the rest of the body nor is it localized so peculiarly. Multiforme exudative erythema is of longer duration and its rash is more variable, exhibiting the vesicular, the bullous, or the urticarial forms. It usually affects chiefly the dorsal surfaces of the hands and feet. It resembles the epidemic disease in so many other respects, however, that Escherich is inclined to regard the latter as an abortive form of muliple erythema. The author cannot support this view.

The disease requires no treatment.

DUKES' "FOURTH" DISEASE

In 1900, Dukes described, a new form of contagious exanthematous disease resembling, in all its symptoms, a mild scarlet fever, but with which it is not supposed to be identical. Since Dukes found it also non-identical with either measles or rubella he called it "The Fourth Disease."

After observing several epidemics of this malady, Dukes describes it in the following detail:

Usually it has no other prodromes than a slight sore throat and nausea. Within a few hours the body is covered with a dense eruption consisting of very small punctiform and slightly raised spots of a pale red color. The lips and the nose are not usually involved. According to Weaver on the contrary they may be also affected. The conjunctivæ and the pharynx are reddened and the cervical lymph nodes are enlarged, but less markedly so than in rubella. No strawberry tongue is observed. The rash disappears rapidly and is followed by a slight coryza, which persists for a week or two.

At times a slight albuminuria lasting but a short period is noted as a sequel. The general health is but slightly or not noticeably disturbed; the fever is absent or slight; convalescence is short and without complications. The disease is contagious for two or three weeks.

Every physician frequently meets such cases, either of sporadic appearance or in the course of mild epidemics of scarlet fever and their occurrence with other scarlet fever cases alone permits of their recognition. Dukes however, distinguishes this disease from scarlet fever and for very good reasons. First, and notably because it often attacks children, who have already had scarlet fever or it affects those who subsequently develop scarlet fever. Secondly, because it is always mild and without complications; and, third, because its incubation period of from nine to twenty-one days is accounted longer than that of scarlet fever.

Filatow probably describes the same disease in 1886 under the name of

rubeola scarlatinosa, which he intended to designate as a separate disease, but not as a variety of rubeola.

The longer incubation period might suggest rubella but Dukes distinguishes it from this disease by virtue of the fact that it frequently affects those in later childhood, who have already had rubella. Other authors and among them Heubner deny the specificity of the disease, considering it an abortive form of rubella or scarlet fever. Since the exanthemata due to sera or to certain drugs show, that one and the same substance may produce now a scarlatinoid eruption, and again a rubeolar, rubellar or urticarial rash, it is certainly not permissible to lay too much stress upon the morphologic form of an eruption in drawing distinctions between these several diseases.

The writer would call attention, however, to the fact that in epidemics of true rubella, cases with a purely scarlatinoid exanthem have hardly ever been described. In the course of two large epidemics of rubella, he has seen cases which presented a rash of scarlatinal character over small areas, but never one in which the entire eruption was of that type.

With many other physicians he has often seen cases which ran a course similar to that of very mild scarlet fever, in which a diagnosis of scarlet fever was nevertheless excluded on account of existing epidemiologic conditions, an epidemic of this disease having preceded the present prevailing malady. These cases may have been identical with "The Fourth Disease" but the author cannot establish positive proof of the fact.

The discussion concerning the existence of "The Fourth Disease" is not yet closed. The determination of its entity can come alone from the discovery of a distinct causative organism, or from more exact studies of its hematology and serology. Very probably we shall be justified in recognizing with Dukes and Filatow the differentiation of the Fourth Disease from scarlet fever. Indeed it is not impossible that there are several scarlatinal diseases.

VARICELLA

(Chicken-pox)

Varicella is a contagious exanthematous disease, usually causing only slight general manifestations and characterized by an eruption of roseolar spots, some of which develop into vesicles, which commonly dry up without pustulation and healing, usually leave no scar.

The causative organism is unknown; apparently it is not present in the vesicles, since, in contrast to small-pox, vaccination of other persons with fluid from the vesicles has been unsuccessful.

Children up to the age of ten years are most frequently affected. During the first three months of life cases are uncommon, but even the new-born are occasionally affected. After the tenth year the number of cases rapidly decreases. The disease is very uncommon in adults. The author has seen it, however, in a negress of thirty and in a man of sixty. In both instances infection from children could be proven.

The predisposition among children is very great. Frequently all the children in a family will be attacked upon exposure. One attack almost

always confers a permanent immunity. Second attacks are exceedingly rare. The fact that adults are so rarely affected is due rather to acquired immunity than to decreased predisposition. The disease is generally more common in the cold season because indoor life favors transmission.

The *contagious quality* of varicella is very great. Usually conveyance is direct from person to person. Some authors doubt that indirect transmission by the healthy person or through the medium of utensils can occur. The writer is quite certain, however, that he has seen such cases, howsoever infrequent they may be.

The virus of the disease is very light, so that in hospital wards transmission seems to occur along the lines of air currents, whence has been begotten the German designation of "Windpocken" and the French term "la petite verole volante." The mode of transmission and the port of entry are still unknown. The contagion is most active at the beginning of the exanthem. By the time the vesicles appear the contagion has usually taken place; possibly it even precedes the eruption. How long the period of contagion lasts is problematical, but doubtless it passes by the time the vesicles have dried up. The viability of the virus outside of the human body seems to be very limited.

In Europe the disease is endemic. There are small or large epidemics, frequently of regional distribution, originating in playgrounds or schools.

The histologic study of varicella usually shows a fan-like structure, the vesicle being situated, as in small-pox, between the epidermis and the corium. The differences between the vesicles of varicella and those of variola are merely those of degree. Morphologically they often resemble each other in every particular.

In the majority of cases the incubation period up to the first appearance of the rash, covers about fourteen days and is probably never less than thirteen. Frequently it extends to seventeen or nineteen days. Such an extension of the incubation period is occasionally due to the incidence of intercurrent infectious disease.

In contrast to variola, prodromal symptoms, are entirely lacking in the majority of cases, or are so slight, that they are apt to be overlooked in small children. Rarely, a slight fever, restlessness and disturbed sleep are noted for a day or two before the appearance of the eruption. Only in very exceptional cases does the temperature rise to 39°-40° C. (102°-104° F.), with the accompaniments of vomiting, intense headache and backache. In one instance, that of a boy of four years, the writer has witnessed severe convulsions. Sometimes a very transient rash, very similar in form to that of a slight scarlatina may be observed for twelve to twenty-four hours prior to the appearance of the typical eruption, or coincidently with it, or immediately following it.

Clinical Picture.—Generally the disease is first noticed by the child's parents when the exanthem occurs. The eruption usually begins on the face and scalp, speedily developing on the body and the extremities. At first discrete roseolar spots of the size of a pinhead appear. Some of them enlarge rapidly to the size of a lentil and a part of them become slightly

raised and papular. A number of the papules show a tiny vesicle rising from the centre within a few hours. This vesicle may occupy the entire surface of the papule in a short time. The edges of the varcella vesicle are often continuous with the plane of the normal skin, but again a large inflamed base appears with the vesicle standing in the centre of the raised red papule and surrounded by a red ridge. The vesicles are of the average size of a lentil and are often of oval or hemispheric form. Sometimes their content is clear and at other times slightly cloudy from the beginning. A shallow depression is rarely seen at the apex of the vesicle; the cloudiness increases rapidly and the contained fluid becomes more or less purulent. In a day or two it begins to dry up and then shows a distinct depression at the apex. Quite frequently the vesicles burst. In from three to six days from their first appearance they have dried to a brown crust. With the disappearance of the red inflammatory areola, which is often entirely lacking, a characteristic hard brown scab is formed by the dried pustule. This scab usually falls off at the end of the first week, but may remain adherent through the second or third week. Commonly it leaves no scar.

It is characteristic of the disease that only a part of the rash becomes vesicular or pustulated, the remainder of the spots disappearing after they have reached the roseolar or papular stage. In the majority of cases new spots, some of them vesicular appear between the old ones for a period of several days or even a week, so that all stages of the eruption may be seen within a small area.

The extent of the eruption is extremely variable. Sometimes only four or five spots can be found over the entire body, again in other cases there may be many hundreds. On the face they may be as dense and as massed as in small-pox. If the exanthem becomes markedly purulent, the patient sometimes gives forth a peculiar and indescribable odor. As it dries the eruption of varicella often causes severe itching, especially over the scalp. This is most noticeable in neuropathic patients and induces scratching and consequently severe infection of the vesicles.

The *mucous membranes* and particularly the mucosa of the mouth are frequently affected. The eruption is most commonly noted on the soft palate but it also appears on the tongue, pharynx and cheeks. Large vesicles form beneath the epithelium, which speedily break down and leave shallow aphthous sores, which heal quickly. The suspicion of diphtheria, which may be raised at first sight of these patches is quickly allayed upon careful examination even in cases in which the appearance of the skin eruption is postponed—a rare occurrence. In some instances vesicles form in the nose and are accompanied by a sanguino-purulent secretion. They are also found occasionally in the external auditory canal. They occur much less frequently on the conjunctiva than in the mouth. They are sometimes very annoying in the latter location, but they usually heal well. It is fortunate, that on the cornea they are extremely uncommon, because there they may cause intense inflammation with clouding and even destruction.

The vulva is often affected, single or multiple vesicles appearing which soon become macerated. The intense itching that occurs in this region

makes the child scratch the parts and as a result a secondary purulent infection, phlegmon, ulceration and in severe cases, even necrosis develop. The resulting painful urination is also annoying. This affection of the vulva is especially frequent and is apt to be intense in cases in which a vulvitis has preëxisted. Eruptions on the glans penis and on the inner surface of the prepuce are more rare. An extremely troublesome but fortunately rare localization of the vesicles is upon the vocal cords. So situated, they are apt to cause hoarseness, croupy cough, and even stenosis, with attacks of asphyxia which may require intubation. Deaths resulting from this complication have been reported. Occasionally the vesicles on the vocal cords appear before the skin eruption and the symptoms may then be mistaken for those of true croup.

The temperature, in contrast to that of variola, generally rises at the onset of the eruption. If fever has attended the prodromal stage the temperature may rise to 39° C. (102° F.) or more. In other cases the temperature is merely subfebrile and very transient. If fever is present, it usually continues as long as new crops of vesicles appear and therefore may last for a week or two. The appearance of a new and extensive crop often causes renewed accessions of temperature. The fever may remain at 39°-40° C. thus (102°-104° F.) for days, while the disease itself is not necessarily severe. Cases are also reported and by no means infrequently, which are wholly free from fever. This conclusion is, however, based on temperature records taken only twice a day. Other cases are mildly febrile during the first day of the eruption

Peculiarities of the Exanthem

In many cases the effloresence is very scanty. Five to ten vesicles or but a single one, may be discoverable over the entire body. In such cases the diagnosis is of course, possible only in the light of concurrent cases. The difficulty is all the greater, because the general symptoms are usually slight in proportion to the poverty of the eruption. In other instances the crop of vesicles may be extraordinarily dense and in some areas particularly over the face or forehead, may even become confluent. In severe cases, the skin between the vesicles is reddened and swollen. In these aggravated forms of eruption the individual spots are often noticeably raised and even distinctly papular as in variola. The vesicles become markedly purulent, the inflammatory areolæ broad and conspicuous. The entire picture may present a great similarity to that of true small-pox, a likeness, which is emphasized when high fever and marked disturbance of the general health ensue, events which are not at all uncommon with an intense exanthem. Definitely purulent vesicles leave scars which may be permanent following varicella. Indeed often very mild cases leave a few scars, especially on the surface of the abdomen. The experienced observer is often able years later to make a diagnosis of a past attack of chicken-pox from the circular form, the pigmented margin and the localization, preferably on the trunk, of the numerous remaining scars.

Too warm clothing and resulting perspiration often aggravate the erup-

tion. The formation of vesicles is favored by the use of heavy bandages, moist applications, etc., or by the employment of such counterirritants as the mustard plaster. It is important, therefore, that such applications be avoided, since an increase of the eruption tends to make the disease itself more severe.

The vesicles do not as a rule exceed the size of a lentil. Occasionally, however, a very large vesicle measuring as much as one centimeter in diameter and resembling pemphigus may be found among the rest. The content of the vesicles rarely becomes hemorrhagic and this probably occurs only in children who have a hemorrhagic diathesis or are in an extremely cachectic state. The exanthem is especially prone to pus formation in weak, tuberculous infants or in those who have eczema. Pus formation, the pustules appearing upon a markedly raised inflammatory base and enlarging for several days, is favored by a lack of cleanliness, by scratching with dirty finger-nails or by maceration of the skin with urine or feces. In such cases, one has to deal with a secondary infection of the staphylococcus or streptococcus. The pus formation doubtless depends also upon unknown factors, since even perfectly healthy and clean children may show a peculiar tendency to severe inflammatory changes around the vesicles and to the formation of scars, while in others the transition from the pustules to normal skin is direct.

In cachectic individuals the pustules sometimes become very large and lead to a gangrene of the skin. Large, punctiform ulcers, extending into the fascia and the muscle tissues reaching, at times, a half-inch in diameter, may be formed. It is not surprising that these should lead to metastasis, general sepsis, and death. In no other infectious disease is gangrene of the skin so common as in varicella. This termination is especially to be dreaded in exhausted and atrophic children in hospitals, to which varicella may be introduced.

In many cases of the disease the general health is not disturbed, and in most of them in fact, is not essentially affected. Restlessness, disturbed sleep, diminished appetite and itching are common symptoms. Headache, vomiting, and jactitation appear in but few cases. If the eruption is severe, high fever may accompany it. In adults the disease usually presents more serious symptoms than in children. With them the exanthem often takes on a variola-like character, so that the rare cases of varicella, that appear among adults may cause the physician much hesitancy and great diagnostic difficulty. This is especially true if no connection with other cases of undoubted varicella can be traced.

Complications are very rare. Besides those already mentioned, nephritis may occasionally appear as late as the second week and even of a hemorrhagic type. Usually it disappears in a short time.

The course of the disease is very light in the majority of cases. The eruption commonly lasts but three to seven days and children may be counted well by that time. The writer has seen one remarkable case in which a fresh crop of the exanthem was arrested after the first day by the

development of a lobar pneumonia. The crisis appeared on the eighth day and was succeeded by an intense eruption.

The diagnosis is usually easily made. The discrete exanthem with typical vesicles, later indented at the apex and subsequently drying up is to be immediately recognized. Seen even later the dry brown scabs on the clear skin permit a definite diagnosis in retrospect. If the eruption is scanty, however, the diagnosis may be very difficult and may be definitely made only in the light of epidemiologic conditions, or with the coincident appearance, or the development within a period of fourteen days of other cases in the family. Frequently distinct vesicles do not appear or are so scarce that they may be overlooked. The eruption may not develop beyond the appearance of a faint roseola or of flat papules. One or two small vesicles perhaps may be found only by careful search. The experienced physician will hardly find serious difficulty even in such instances, if the skin was normal prior to the eruption. The diagnosis, however, becomes very difficult if the eruption occurs on a skin, which had been previously affected with a purulent or papular eczema, an impetigo, scabies, etc., the individual elements of which resemble varicella. In such cases a few varicella vesicles scattered among the preëxisting lesions may be readily overlooked.

The differentiation of this disease from variola[1] is of great importance. Usually the two are easily distinguished. In small-pox there are severe prodromes and the temperature falls at the beginning of the exanthem. In varicella distinct prodromes are usually wanting. The fever, if it is present at all usually rises at the beginning of the eruption. In variola the eruption is most severe on the face and palms of the hands. It begins on the face and rapidly spreads to the neck, body and extremities. Distinct papules with marked depressions at the apex and with extensive pus formation appear in the latter disease. The exanthem of variola is complete within three days, so that it is in the same stage of development in all parts of the body at once. On the other hand new crops of vesicles appearing in from three to seven days are so characteristic of varicella, that all the several stages from the fresh roseola to the dried pustule may be seen side by side. In variola, the formation of the vesicles requires several days, but in varicella, they may be formed within a few hours. In this latter, secondary fever due to pus formation is generally lacking. Nevertheless, all these differences are very slight and are purely qualitative. This is especially true in those mild forms of small-pox or varioloid seen in vaccinated persons, which may be very similar to varicella. Moreover, it is to be constantly remembered that there are severe cases of varicella, which cannot be distinguished from variola morphologically and in which there may be severe prodromes and distinct papules and pustular formation. On the other hand, there are cases of variola, which run a mild course with an eruption scantier than in varicella. It follows that the most experienced physician may be unable to make a decision for several days, unless aided by the concurrence of undoubted cases of variola on the one hand, or by a

[1] On account of limited space a detailed description of small-pox has been omitted.

definite history of contagion from varicella on the other. It is well to consider all doubtful cases as variola and to maintain strict isolation until the diagnosis is established. Such cases should not be isolated with small-pox, however, without previous vaccination. At present there is no way in which the efflorescence of varicella and variola can be distinguished, since the pathologic anatomy of the two conditions is identical. From the variola pustule the disease may be transmitted by vaccination to a healthy person, while this is impossible from the varicella vesicle. This test, however, can hardly be applied. In a general way a suspicious exanthem in an unvaccinated person or in one who has not been vaccinated for over seven years, especially if it be in the adult is rather indicative of variola. This is particularly true if the eruption is scanty and becomes distinctly papular and pustular. Animal experiments may be used as an aid to differential diagnosis. If the cornea of a rabbit be vaccinated with the contents of a small-pox vesicle, certain inclusions, known as Guarnier's bodies, are found in the cells of the cornea on the second day. These bodies may also be obtained by the use of vaccine pustules but not by the contents of the varicella vesicles.

While we must appreciate the weight of the argument of those, who believe that variola and varicella are one and the same disease and that the clinical picture and the morphology of the exanthem show no differences in individual cases, there can be no doubt to-day that the two are separate and distinct diseases. The single fact, that varicella is just as common in countries where vaccination is compulsory as in those where vaccination is not enforced, is sufficient proof. Either vaccination or one attack of variola doubtless protects against this disease but not against varicella. Unvaccinated persons are affected by variola, in spite of the fact that they may have had varicella. In unvaccinated persons, who have had chicken-pox, vaccination takes, but this is exceptional in those who have had small-pox. During epidemics it has often happened that a child placed in the small-pox hospital on a mistaken diagnosis has often caused an outbreak of varicella there and *vice versa* a case supposed to be chicken-pox may have created a small-pox epidemic in the varicella ward. The difficulty of differentiation has often occasioned the conscientious physician great anxiety and he does well to share the responsibility with an epidemiologist. The danger of confusion is especially great in Germany where small-pox is so uncommon, so that a case may not be recognized when it does appear. It may be that the hyper-lymphocytosis which often occurs in variola will have a diagnostic value.

The difficulties of the differentiation of varicella from other diseases are not so great. Occasionally a large pemphigus-like character of the vesicles result from their continued enlargement, but typical varicella vesicles may usually be found near by or repeated, new crops, appearing for weeks may show that the disease is really pemphigus. Secondary lues in exceptional cases may show an exanthem similar to varicella. The writer has seen a case in an adult in whom the distinction could not be made from the morphologic appearance during the first few days. Not infrequently forms of strophulus are seen in lymphatic children in which the usual papule becomes

vesicular with or without a basal urticaria, a prurigo varicelliformis. The firmness and often the glassy hardness of the vesicle is a definite criterion of distinction.

The prognosis is good in the large majority of cases. The disease is severe, or even fatal in very exceptional cases only and this severity is usually shown in feeble, cachectic patients. In healthy children varicella is very rarely fatal. Nevertheless, the author has seen a strong infant of three months with a hardly noticeable rash die of sepsis at the end of the first week. In children suffering with purulent eczema the course of the disease is generally less favorable, since the exanthem usually becomes markedly infected. Frequently it has been observed that a latent or an insignificant exudative diathesis or an incipient tuberculosis becomes active after recovery from varicella.

Prophylaxis must not be neglected in view of the possible danger of a serious course or a fatal termination. Infants and feeble children should be isolated whenever it is possible as soon as a case appears in the family. In children's hospitals the danger of a case being brought in is especially to be dreaded. If, however, this has occurred and it cannot always be avoided on account of the long incubation period, the continuance of the disease in the ward can be limited only by forbidding new admissions for three weeks and then disinfecting the room. If there is a suspicion that the case may be one of small-pox, it must be carefully isolated and all the children in the house vaccinated, since vaccination protects against small-pox even a day or two after exposure.

The treatment of mild cases is expectant. So long as there is fever and as fresh crops of the eruption appear, the patients should be kept in bed and on a light liquid diet. The severe itching may be relieved by a 1 per cent. mixture of salicylic acid with talcum powder, by painting with mentholated alcohol (½ per cent.), or with a 1 per cent. thymol ointment. The finger-nails should be cut short. Baths, packs and other procedures tending to increase the eruption must be avoided. If the mouth is involved, it should be rinsed and the throat gargled with some bland solution such as boric acid or 2 per cent. hydrogen peroxide. A painful ulcer in the mouth may be touched with a 2 per cent. solution of silver nitrate. The eruption affecting the vulva, must be kept scrupulously clean and should be protected from maceration by the use of powder or ointment and strips of gauze. Large pustules containing much pus may be covered with a drying paste and ulcerous or gangrenous areas must be bandaged.

VACCINATION (cow-pox)

In India, it was observed in ancient times that one attack of small-pox conferred very definite protection against a second attack and various methods of vaccination with old small-pox virus were practiced. In the eighteenth century many persons were inoculated in Europe, after Lady Montague had introduced the practice to the western world from Constantinople. But inoculation was later discontinued and even prohibited in many countries since people not infrequently died from an induced small-

pox. Another unfortunate circumstance was that real epidemics repeatedly arose from this practice.

The present method of vaccination dates from the famous English physician, Jenner, who first practiced it in 1796 and who received for his discovery many rewards from the English Parliament. Observing, that a certain udder disease of cows popularly termed cow-pox, occasionally infected the hands of milkers and that those who were so affected later escaped small-pox, he conceived the idea of inoculating persons with the content of the vesicles developed in the disease in the cow in order to protect them from future attacks of small-pox. The results justified his surmise. Persons so vaccinated proved to be insusceptible to intentional inoculation with small-pox virus. Jenner's theory, that this vaccine is nothing more than an attenuated variola on the body of the cow has, however, been proved to be correct but recently by Voigt and others. With the passage of the disease through the animal it loses its power to spread through the air and to cause a general skin eruption.

At the beginning of the last century Jenner's method of protective vaccination was introduced into many European states with remarkable results. It soon appeared, however, that the protection which it conferred did not persist as long as that resulting from the disease itself and that vaccinated persons again become increasingly susceptible to small-pox with the lapse of years. During epidemics it has been found, that children vaccinated, early again become more and more liable to the disease after the seventh year and are affected, in fact, in increasing numbers. In such cases, however, the malady usually appears in an attenuated form known as varioloid. In older people the protection secured by vaccination commonly persists for a longer time.

The vaccination law in Germany in effect since 1874, requires that every healthy child shall be vaccinated by the end of the calendar year following its birth and that the vaccination must be repeated in the twelfth year. This law fulfills a practical requirement and as a result but very few cases of small-pox have been seen in that country since that time. These few are usually brought in from some foreign country and can never cause a widespread epidemic.

In countries in which vaccination is not compulsory or in which vaccination laws are indifferently enforced, we still see large epidemics of variola, which can be stopped only under good hygienic conditions and strict isolation of the sick. Nevertheless since the days of Jenner, the antivaccinationists have never ceased their agitation and renew their protests and petitions against compulsory vaccination from year to year. They raise the plea that vaccination does not protect against small-pox. How preposterous is the claim is shown by the comparison of the experience of modern times in those countries where there is compulsory vaccination with the history of these countries before Jenner's discovery, when small-pox was the most serious disease known. It is further proved by the epidemics which have frequently occurred in countries after compulsory vaccination had been abandoned as in England and in several of the Swiss cantons.

The objection has been brought forward, that syphilis and tuberculosis are transmissible by vaccination. Formerly, when it was the custom to vaccinate from persons to person with humanized lymph, lues was actually transmitted quite frequently as a result of the careless selection of persons giving the vaccine. No case in which tuberculosis was so transmitted has ever been proved. Since calves' lymph has been solely employed, the animals being slaughtered and examined before the vaccine is used, the transmission of syphilis has become impossible. The statement of the anti-vaccinationists, that the health of children may be injured for life is to be discussed.

The best time to vaccinate children is between the fifth and the twelfth months. Infants fed at the breast may be vaccinated during the first week. Artificially-fed children should not be vaccinated during the hot months of the year unless there is immediate danger of small-pox. If a case of small-pox occurs in a family, a member of which has not been vaccinated, immediate vaccination may still be protective against the disease, since immunity is fully established in eight days. The lymph to be used must be obtained from a licensed institution and should not be older than three months. The *technic* is extremely simple, but it requires the same asepsis as any other surgical procedure. The child is bathed on the preceding day, and prior to vaccination the selected area of operation is to be cleansed with ether. The outer side of the right arm is usually chosen or, in event of revaccination the left arm. Four shallow scratches about one centimeter long and crossing each other two by two are made. The entire area covered should be about three square centimeters. A vaccination lance or a large surgical needle thoroughly sterilized is used. A drop of lymph is previously placed upon the instrument from the capillary tube, so that it will run into the scratches. These scratches should be so slight, that they produce only a faint red line. There must be no free blood. The vaccine virus does not need to be rubbed in. The wound is allowed to dry for ten minutes. During this time the child must be so held, so that it cannot touch the vaccinated area and that the clothing does not rub over it. After the drying is completed it is well to cover the area thoroughly with collodion. This procedure the writer has found extremely useful and when employing it, he has never had a vaccine infection in other parts of the body or in other members of the family. Usually, a light cotton bandage is placed over the area, which may remain in place for several days and serves to protect the vaccination from scratching. If a little of the cotton adheres to the wound, this is not to be disturbed. If it is necessary to change the bandage it may be trimmed away from the area with the scissors. Vaccination shields are unnecessary and may even prove harmful, if they prevent the drying of the vaccinated spot. In girls the vaccination may be done upon the outer surface of the thigh, if the avoidance of a scar upon the arm is desired. The chest immediately below the breast may be preferred.

The child may be bathed the day after the vaccination, but after that tub baths should be discontinued until the scab has dried up, which will be about the fourteenth day.

After the first vaccination the inoculated area immediately shows a slight reddening as the direct result of the trauma. This disappears in a day or so. Then a thin brownish line marks the scarified spot. Within two or three days the brown line is edged with a red border which soon changes to a reddish ridge or vaccination papule. This papule becomes more and more prominent and wider from day to day. After the fifth or sixth day it grows pale at the centre and a flat areola rising prominently from the surrounding red skin, is formed. The whitish papule enlarges continually until the ninth day, but after the seventh day it becomes yellowish. Its centre becomes depressed over the point of the original scratch. The papule itself is now vesicular and when squeezed discharges a clear lymph, the humanized lymph, which was formerly used for further vaccinations. The red areola now becomes still wider and rises as a plateau above the surrounding unchanged skin. Its area varies in size. Frequently the sites of the four separate scratches become confluent and form an erysipeloid area. This may attain to a diameter of three or four inches. The reaction reaches its height in nine or ten days, after which the papule begins to dry up rapidly from the centre outward. Meanwhile the papule has been growing more and more yellowish and when it begins to dry up it changes into a hard brownish scab. This falls off in from two and a half to four weeks and leaves a red scar, which pales very slowly. With the further drying of the papule the red wall surrounding it fades rapidly, but becomes pigmented at the edge. It will usually have disappeared completely some twelve or fourteen days after the vaccination. According to von Pirquet the organism forms two distinct antibodies against the vaccine virus; the first, a lysine, which liberates a toxin from the virus around the point of inoculation, causing the formation of the areola, the vaccination process being terminated by the formation of a toxic antibody.

Intercurrent acute diseases, such as scarlet fever, measles, etc., may delay the development of the vaccine reaction for a short time.

The general health usually shows no disturbance during the first few days after vaccination. After the fifth or sixth day, a slight febrile rise may set in, which even under ordinary conditions may go to 39° C. (102° F.). Accordingly the vaccinated child is often less active than usual between the seventh and the tenth or eleventh days. Sleep may be disturbed and the appetite diminished while the vaccination remains painful. The regional lymph nodes are slightly and occasionally markedly swollen so that the child may complain of pain if lifted beneath the arm.

A subsequent examination, which should ordinarily be made on the seventh or eighth day, shows a large white papule with a slightly developed inflammatory areola around it. The reaction does not reach its maximum until the ninth or tenth day. After the seventh day a little of the vesicular contents may occasionally escape. The parents should be warned against the customary procedure of applying some favorite salve, since this will delay the normal retrograde changes and prevent the vesicle from drying up and may invite pus formation.

Revaccination is essentially different in its course. It has been exhaust-

ively studied by von Pirquet who derived the conception of his well-known cutaneous tuberculin reaction from these studies. One vaccination permanently changes the power of the organism to react to future vaccinations or to infection with variola.

If the second vaccination takes place a few months after a positive reaction has occurred, a very small papule appears in a very few days and disappears as rapidly. In this event the disease has been nipped in the bud. A few years after the first vaccination the susceptibility to vaccinia is usually found to have increased again. Hence the formation of a papule and areola will occur as in the first vaccination but the reaction is less intense. The papule remains small and the entire process reaches its maximum height by the seventh day. As a result the scar formation is also slight. In the adult the reddening is at times very widespread and may even extend to the forearm. The swelling of the lymph nodes is severe and painful.

Peculiarities of Course and Complications

In general it may be said that the younger the child—when vaccinated, the milder is the reaction. Children who are vaccinated for the first time after some years and those who are unvaccinated until adult life usually react intensely with the formation of a large pustule, a widespread erysipelatous areola, a marked fever and with serious disturbance to the general health. Edema may appear on the arm.

In anemic and cachectic children the reaction is usually peculiar in that the areola is slow of formation and poorly developed, giving the papule the opportunity to spread. The severity of the reaction is also determined by the virulence of the vaccine and by the quantity inoculated. With a virulent vaccine small eruptions of the size of a pinhead, often develop in the areola from the fifth to the seventh day, which go through all the same retrograde changes as do the eruptions of small-pox on the skin at large. These accessory or secondary pox were a very common manifestation, when vaccination was formerly done with true small-pox virus. With weak or diluted vaccine the reaction may be retarded and may not reach its maximum intensity for eleven or twelve days. The reaction occurs more rapidly in summer than in winter. High fever is common with strong children and is no cause for anxiety, if it disappears within the second week. If this subsidence is not noted, some complication is to be suspected.

A vaccine exanthem occasionally appears during the decline of the reaction. It usually develops between the eighth and twelfth days, but it may be of either earlier or later occurrence. It is generally similar in form to that of measles and is seen on the face, the trunk and the extensor surfaces of the extremities. At times it resembles scarlatina or a miliary pemphigus.

This exanthem may be understood when it is remembered that a rash always appeared at about the same time in persons inoculated with small-pox. This rash was usually varioloid in form. Since the virus of variola is attenuated by repeated passage through animals, the exanthemata which may develop, when vaccine produced in the usual manner is used, are also slight and are ordinarily absent.

Albuminuria may appear for a short time but has no particular significance.

The result of vaccination may be considered positive when at least one focus of inoculation develops a normal pustule. This usually satisfies the requirements of the vaccination laws. Its acceptance is justified by the fact that a second vaccination produces no further reaction and the failure of a non-vaccinated child to react to vaccine is an extremely rare occurrence. If the first vaccination causes no reaction at all, it should be repeated within eight days. Then it frequently happens that the still latent virus of the first vaccination increases the reaction. Such latency accounts for the fact that a reaction may at times be delayed for weeks. This peculiar circumstance is probably due to the fact, that the virus does not properly penetrate the skin and is finally brought in touch with an efficient soil through the friction of the clothing, etc.

Injuries, actually attributable to the vaccination may be avoided by a careful technic and by due selection of the individuals to be vaccinated. Secondary infection of the pustule with pyogenic organisms hardly ever occurs if the wound is properly treated and more particularly if the patient is not allowed to scratch the pustule. If such an infection does develop, it heals rapidly under suitable treatment. A late erysipelas may find an opportunity of development in the infected pustule. The condition generally regarded as a vaccine erysipelas is really a part of the vaccination process and means merely an unusually large areola, which carries with it no unfortunate results. A diagnosis of true erysipelas is only to be made, if the disease is ushered in by fever arising after the disappearance of the areola. Extensive pus formation in the pustule and attending suppuration of the regional lymph nodes are not to be expected in healthy children, whose vaccination wound has been kept dry. Such conditions will heal rapidly if they are treated with applications of an aluminium acetate solution or with an iodoform dressing. While general sepsis might arise from a vaccination as from any other wound, yet scrupulous asepsis both during the operation and during the reaction is an absolute protection.

Considering the enormous number of infants vaccinated in countries, in which vaccination is compulsory it is not surprising, that disease is often concurrent, a purely accidental coincidence and without any causal relation. Such cases are of course, immediately seized upon by the antivaccinationists and even by parents and are counted due to the inoculation. For this very reason physicians should be all the more scrupulous in seeing to it that no injury is done that can be charged either to him or to the event. In very anemic or cachectic children or in those who are sick, vaccination should be postponed. In children of exudative diathesis, an eczematous eruption following vaccination is frequently observed (Czerny) and in the tuberculous the appearance of scrofulous manifestations may be excited. Luetic, atrophic or seriously rickitic children should not be vaccinated, since the vesicles tend to ulcerate in such patients (Klotz).

A great danger attending vaccination lies in the danger of spreading from the inoculation site, resulting in the formation of pustules in other parts of the body. Such an extension is always possible, but it is a very rare occurrence,

if the vaccination point itself receives proper attention. The most unpleasant consequences almost always follow in infants, who are suffering with eczema. If but a trace of fresh vaccine is carried to the eczematous surface by the infant itself or by the nurse a severe and widespread eruption of vaccinia may result. The face is most frequently involved. The condition may be accompanied by serious illness and permanent and disfiguring scars may remain. Governed by the period of transmission umbilicated pustules, vesicles, or mere papules appear. It follows that children with eczema or

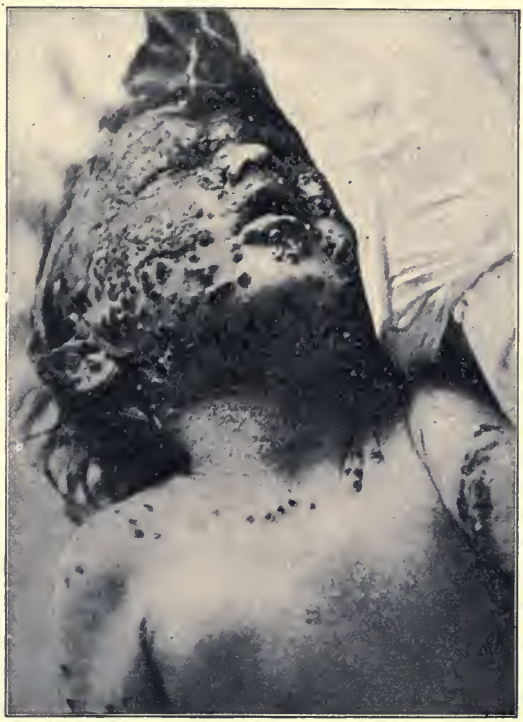


FIG. 163.—Eczema vaccinatum, eighteen-month-old child. Infected from the vaccination pustule of a vaccinated sister. Sixth day of the disease.

with any other irritative skin lesion should be vaccinated only under very exceptional circumstances and then the area of vaccination should be covered with collodion and bandaged, while the eczema, also, should be carefully protected.

A spontaneous general vaccinia probably spreads by way of the blood. In such a case the entire efflorescence will show a uniform stage of development.

Another and even greater danger ensuing upon vaccination is often overlooked and this lies in the possibility of the accidental inoculation of unvaccinated persons, who come in contact with the vaccinated child. Such an inoculation may be due to the introduction either of the fresh vac-

cine or of the content of the pustules. As a result of such contact with the vaccination of the child, a pustule may form in some such critical spot as the eye of the mother or the nurse who may be unvaccinated or in whom vaccination may be of remote date. Unvaccinated children living in close contact with the recently vaccinated child, and especially those who have such skin affections as eczema stand in special danger. A widespread and severe vaccinia may arise as we have seen, from the inoculation of the diseased skin. This occurs more readily with the contents of the mature pustule, the virulence of which lasts until about the eleventh day, than it does with the fresh vaccine. The accompanying picture (Fig. 163) is that of an unvaccinated two-year-old child having a slight eczema on the arm and face, which was inoculated with the content of the pustule of a successfully vaccinated brother. This eczema vaccination resembles a severe variola very closely. The child died of a complicating purulent meningitis.

In view of these dangers children, who have unvaccinated brothers or sisters, who are suffering with eczema should not be vaccinated, unless positive assurance can be given, that all necessary protective measures will be scrupulously carried out. It is the physician's duty to protect persons closely associated with the vaccinated child from accidental inoculation. This is most satisfactorily accomplished by covering the vaccinated area with collodion and a bandage and by preventing the scratching of the vaccination papule. Von Pirquet recommends the careful washing of the vaccinated area after twelve hours instead of covering with collodion. We have carefully tried out the collodion method and other physicians also recommend it.

The severe injuries described, resulting from vaccination are, of course extremely uncommon. If they seem to be given greater emphasis than their frequency appears to justify, it is simply because with their very infrequency they are often unrecognized and because vaccination is a procedure, which must be enforced with healthy children. Furthermore, the report of every injury resulting from vaccination is seized by the antivaccinationists as a weapon to be used in their combat against this wonderful agent of prevention.

DIPHTHERIA

Diphtheria is a contagious disease, caused by Loeffler's diphtheria bacillus, the most important symptom of which is a membranous exudation upon certain mucous membranes, especially those of the pharynx and the upper respiratory passages, a disease which produces peculiar toxic effects and resulting sequelæ in the way of paralyses, etc.

Diphtheria has probably been known for a long period: It is certain that it appeared in frightful epidemics in Spain during the sixteenth and seventeenth centuries. The first classical description of the disease is written by the eminent French physician, Bretonneau, who gave it the name Diphtheria (1826). Up to the middle of the nineteenth century the disease appeared in Germany chiefly as a primary affection of the larynx and bronchi, to which the name of membranous croup and membranous bronchitis were

given. The affection of the pharynx became more common in 1860, when numerous German physicians saw it for the first time.

The causative organism was shown by Loeffler, in 1884, to be the diphtheria bacillus. He succeeded also in producing a membrane in rabbits by the inoculation of lesions in the trachea. The Loeffler bacillus was generally acknowledged to be the infective organism when Roux and Yersin succeeded in producing the same paralysis in animals as diphtheria causes in man.

The diphtheria bacillus belongs to the group of coryne bacteria and shows great variation in form, growth and virulence. It is usually a straight or slightly curved, or club-shaped rod, about as long as the tubercle bacillus but much thicker. It stains readily especially with Loeffler's methylene blue. On account of its irregular staining quality it often appears granular. Besides the very short young forms, long club-shaped, degenerative forms are also seen.

The bacillus grows very well on Loeffler's blood serum and moist smoke colored colonies may be recognized in from sixteen to twenty-four hours. The organism is not affected by cold but is rapidly destroyed at a temperature of 56° C. (133° F.). It is also very sensitive to the stronger antiseptics. In dark moist places, especially in membranes or in mucus, it remains alive for months while it dies in a few hours in bright sunlight. On the infected organism it is found in the exudative membrane of the necrotic tissue. Later it occurs in the neighboring lymph nodes and may also be found in the lungs, the cerebrospinal fluid, in the blood and occasionally in the urine.

After the disappearance of the false membrane, the bacillus may usually be found for several days on the surface of the membranes, which have again become normal. At times it may even be present for many weeks. Not infrequently the germ is found in the pharynx of healthy persons and especially in those, who are in close contact with a case of diphtheria. These persons are termed carriers. The short pseudodiphtheria bacillus is closely related (p. 658).

This pseudo form frequently lives in the nose and throat of healthy persons and morphologically and biologically shows gradual transition stages to the true diphtheria bacillus. It is, however, always a virulent for the guinea pig, which is killed by infection with the true diphtheritic type.

Diphtheria is usually spread from one person to another. Its origin is often obscure, since the infection may come from convalescent or healthy bacillus carriers.

Since the bacillus lives almost exclusively in the nose and mouth, the possibility of transmission in the act of kissing is very imminent and the danger of its spread from infected children by contamination of hands, bedding or clothing with the secretions of the mouth or nose is especially great. Indirect transmission by means of handkerchiefs, toys etc., is not at all uncommon. Filth and a lack of personal cleanliness are active agents in the spread of the disease. Accordingly the cleaner children of the well-to-do are less frequently affected than those in poorer circumstances and there is a certain justification in designating diphtheria as a dirt disease. The large, heavy diphtheria bacilli are obviously not very readily diffused and

usually require contact for transmission. Nevertheless droplet infection is quite a conceivable thing in the event of severe coughing and sneezing, and particularly with nasal diphtheria.

As a rule the bacilli reach the port of entry through the mouth, conveyed to it by the fingers or on the food. They develop primarily on the faucial or pharyngeal tonsil.

Cases are most numerous between the ages of two and five. After the tenth year the disease is uncommon and but few adults are affected. In fact adults are affected only in severe epidemics. Cases occurring in infancy are infrequent, probably because the adenoid organs of the pharynx are immature at this period of life. It is not to be supposed, however, that young infants escape entirely. They are affected chiefly with nasal diphtheria.

The general predisposition to the disease is not nearly so great as toward measles or pertussis; many persons never have diphtheria even though they are exposed to infection and have never had the disease. (See page 574.)

Susceptibility depends upon many factors. For some unknown cause certain individuals show a greater predisposition than others. This predisposition usually increases during periods when the mucous membranes most liable to infection are in a catarrhal or inflammatory state, particularly in cold seasons and during transitional periods. This affords explanation of the chief prevalence of affections of the larynx and bronchi during those months, when catarrh is common and of the more sporadic appearance of pharyngeal diphtheria during the summer and fall. The individual predisposition is determined by the presence of irritable mucous membranes and of adenoid vegetations. Children with a lymphatic habitus are affected more readily than those who are free from this constitutional anomaly. These children also offer less resistance to the disease than do normal types.

In certain cases it is often extremely difficult or quite impossible to determine the extent to which either the inherent predisposition of patient or the intensity of virulence in the infective organism is responsible for a given attack of the disease. The character of an epidemic is doubtless determined in a general way by the virulence of the bacillus.

It is not definitely known whether a single attack of the disease confers immunity. It is undoubtedly much less permanent than is generally supposed, for second and even third attacks are not at all rare. It is evidently a question rather of immunity to toxin, than of immunity to bacteria, since individuals recovering from diphtheria often carry virulent diphtheria bacilli for a long time without being affected by their presence. Recurrences, too, often after three or four weeks are especially common in patients who have been treated with serum, taking place at the time when the passive immunity loses its power.

The Schick Reaction.—Schick has published a method by which the natural presence of antitoxin in the blood and tissues can be determined very easily. A standard diphtheria toxin is diluted with normal saline to such a strength, that $\frac{1}{10}$ c.c. or $\frac{2}{10}$ c.c. contains $\frac{1}{50}$ M. L. D. for the guinea pig. This amount is injected intracutaneously on the flexor surface of the arm. The explanation of the test is that when no antitoxin is present,

the toxin acts on the skin and produces in from 24 to 36 hours a circumscribed area of redness and slight infiltration, which measures from 1 to 2 cm. in diameter. It persists from seven to fifteen days and on fading shows a superficial scaling and a persistent brownish pigmentation. This is called a positive Schick reaction and indicates that the individual is susceptible to infection with the diphtheria bacillus. Pseudo-reactions are rather frequent in adults. They appear earlier, are more edematous, usually of greater diameter and disappear in from 24 to 72 hours without leaving the characteristic desquamation and pigmentation. They can be obtained with neutralized or heated toxin and in certain individuals with dilutions of plain broth. According to Park and Zingher the percentage of positive Schick reactions at varying age periods is briefly as follows:

Positive Schick Reactions

Years.	Per Cent.	Years.	Per Cent.	Years.	Per Cent.
1–2	50–70	6–8	21–55	12–14	17–50
2–4	32–60	8–10	22–55	14–16	16–50
4–6	25–55	10–12	21–50	16–30	15–40

Toxin Antitoxin Inoculations.—Theobald Smith made a careful study of the production of an immunity against the diphtheria toxin in guinea pigs by the use of toxin antitoxin mixtures. He suggested the use of such mixtures in children for practical immunization. Behring brought about the practical application of this method. Park and Zingher in this country immunized 4000 cases by inoculating toxin antitoxin mixtures (66-70 per cent. L+ to each unit of antitoxin or 80-90 per cent. L+ to each unit of antitoxin). They obtained the best results by giving three injections at weekly intervals. No harmful after effects were noted and according to their latest reports these toxin antitoxin injections produced permanent immunity in about 90 per cent. of the non-immunes. Based on the Schick reaction 30-40 per cent. became immune three weeks after the first injection, about 50 per cent. at four weeks, 70-80 per cent. at six weeks and 85-90 per cent. at from eight to twelve weeks. The duration of the active immunity as evidenced by continued negative Schick reactions has persisted for over four years.

A pronounced and dangerous predisposition to diphtheria of the respiratory passages is caused by measles. (See page 612)

During recent decades sporadic cases of diphtheria have constantly occurred in most countries. Frequently, however, distinct epidemics appear. In a general way it may be said, that the severity of the disease and the activity of its contagious quality increase with the multiplication of the cases. The origin of these epidemics is still wholly obscure. Over long periods of time, which may indeed cover many decades, the number of cases may gradually increase from year to year, until there develops a widespread and terrible epidemic, after which long periods again elapse in which only sporadic cases appear. In Germany there has been a general decrease of diphtheria during the last twenty years without diminishing the

fact, that epidemics of greater or less severity still occur in single cities or villages, as was seen in Hamburg in 1909 and 1910.

A seasonal influence is usually distinctly noticeable in the appearance of sporadic cases. It has been shown, that the cold months are especially favorable to the disease. Indeed, cold dry winds often cause a marked increase of cases of croup. Epidemics may occur, however, during the summer.

Pathogenesis and Pathologic Anatomy

The growth of the diphtheria bacillus on the mucous membrane first produces a swelling and necrosis of the epithelial cells as a result of the action of the poison extruded by them. The deeper action of the diphtheria toxin causes an inflammation of the capillaries in the immediate neighborhood with resulting infarct formation and the excretion of fibrin. This fibrin passes into the necrotic epithelium and with it forms the white exudate, which constitutes the diphtheritic membrane. In it are also found emigrant round cells. Depending upon the depth to which the toxin penetrates are the macroscopic results. An exudate in the epithelium and a loose pseudomembrane upon the mucosa, anatomically termed croupous, are formed if the process be a superficial one. It includes the deeper layers of the mucosa resulting in a firm inlay anatomically termed diphtheritic if the process is deep. Upon the surface of the mucous membrane are found various saprophytes, staphylocci, streptococci, etc., associated with the diphtheria bacillus and making up the detritus. In the deeper portions of the membrane, which contains larger quantities of fibrin, are found only diphtheria bacilli and markedly degenerated and necrotic epithelial cells. Below this is seen a fibrin layer containing numerous leucocytes and very slightly altered epithelium. Thence, the bacilli reach the neighboring lymph nodes but they very rarely get into the blood, so that most of the disturbance of the general organism must be considered the result of the action of the toxin. If the toxin affects the deeper structures, necroses of the entire mucous membrane may result. Such necroses heal by second intention with scar formation.

Aside from the mucous membranes, the heart and the peripheral nerves are most frequently affected.

At autopsy the heart is found in diastole and usually shows marked myocardial changes. The cardiac muscle is bluish-gray, is friable, containing fat globules, and shows marked parenchymatous degeneration and frequently disappearance of striation. According to Eppinger, a degeneration of the myocardium, resulting in a characteristic dissolution of the muscle fibre, is frequently seen. This finding is denied, however, by others. Serious degeneration of the muscle nuclei is often found following the cardiac death of diphtheritics.

In cardiac death the vagus nerve often shows marked changes, especially in the destruction of the nerve sheaths, in the atrophy of the axis cylinders and in a small cell infiltration. Similar degeneration is demonstrable in the various nerves affected in peripheral paralysis. In the spinal cord distinct changes are usually lacking.

Roemheld was able to demonstrate in a case of diphtheritic paralysis of an adult, an increase of protein in the cerebrospinal fluid, an increase which the writer has found quite regularly in hospital cases of children with similar paralysis. This is evidence, at least, of a toxic irritation.

The General Disease-picture

The course of diphtheria is extremely variable, depending upon its localization, the virulence of the infection and the individual predisposition. The commonest and mildest form is confined to the tonsils. This localized form has a moderate course in the majority of cases, but it may develop a severe toxicity and result fatally. In other cases there is a tendency to involve large areas, the membrane formation spreading to the palate, the pharynx and even to the nose, larynx and trachea. The malignancy of the disease, however, is not always in direct ratio to the extent of its spread, although those cases in which the membrane is confined to the tonsils are as a rule either in themselves or by aid of the antidiphtheritic serum more benign.

In order to facilitate description one may consider a case of medium severity and add to it the peculiarities and complications of the course. The incubation period is not constant. It usually lasts from two to four days; at least this is the length of time which elapses before second cases appear in an infected family.

The onset of the disease is usually attended by general symptoms. The child becomes pale and listless, has a poor appetite, sometimes vomits and complains of headache. The older child may refer to the pain in swallowing, even at this time. The physician, called perhaps the next day, finds the temperature ranging from 38°-39°C. (100°-102° F.), a rapid pulse and pronounced malaise. A slight swelling of the submaxillary nodes, which are painful upon pressure and a marked *fetor ex ore*, which the mother has noticed, lead the physician to inspect the throat, even though the patient may not complain of pain. The tongue has a white coating. Both tonsils are moderately enlarged and reddened. Upon one or both a white spot is seen covering a third or half of the surface. This white deposit cannot be wiped away with a cotton-covered applicator but may be removed with a forceps and is found to be a rather firm elastic membrane. If the physician sees the case very early, he finds merely an ordinary and moderate tonsillar angina, or a fine veil-like exudate over the tonsils. By the next day the reddening and swelling of the tonsils has increased. The membrane has become thicker and more extensive and may cover the entire surface of both tonsils (Fig. 164). Frequently the process stops at this point. Usually however, one or more follicles on the visible or posterior wall of the pharynx become covered with the white exudate at about the same time. From these follicles the membrane gradually spreads until it covers the entire pharynx. Within four or five days the process has often extended so that the soft palate, the uvula, both tonsils, and a part of the pharynx are covered and its maxinal point has been reached.

In the meanwhile the swelling and redness of the tonsils and the soft palate has increased. The tonsils almost touch in the median line and pre-

vent closer inspection of the pharynx. The difficulty of examination is increased by the fact, that the inflamed parts are covered by a mucopurulent secretion.

The swelling of the fauces and pharynx causes difficulty in breathing and snoring and mouth-breathing result. The odor from the mouth, a sickly glue-like smell, is aggravated and may be noticed at a distance. Mucus and saliva flow from the open mouth. The swelling of the submaxillary nodes becomes more marked and may be visible. Palpation reveals separate nodes the size of a cherry. These are moderately sensitive, but there is no inflammation of the perinodular tissue.

The general condition of the patient grows worse from day to day. The temperature varies from 38.5° to 39.5° C. (101°-103° F.). Marked morning remissions may occur, ranging almost to normal or it may drop after the first two or three days. The pulse is rapid, running from 100 to 180 according to the patient's age. It is small and compressible. The face shows an increasing pallor, with deep shadows under the eyes. The child looks tired and ill. His appetite is completely lost and it is extremely difficult even to force him to take any nourishment at all, on account of the pain of swallowing. Sleep is disturbed and often interrupted by the difficulty in breathing, resulting from the swelling and the excessive secretion.

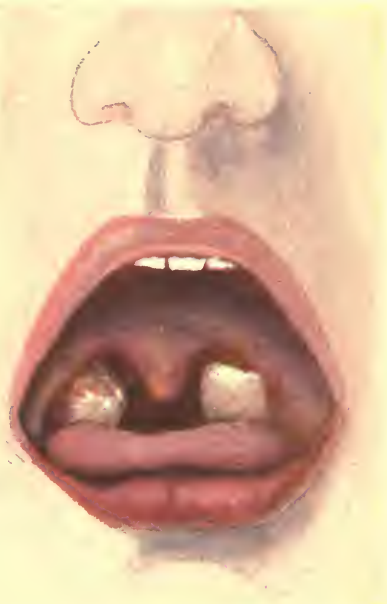


FIG. 164—Diphtheritic exudate on both tonsils. (Right tonsil not entirely covered).

Examination of the internal organs reveals no special changes. The lungs remain normal. In the heart low systolic murmurs are occasionally heard over the mitral and over the pulmonic. The spleen and the liver are slightly enlarged.

The urine frequently contains a moderate quantity of albumen and casts after the third day.

In previously healthy children and especially in those in later childhood, the disease often abates from the fourth to the seventh day. The membrane may have been confined to the tonsils alone or the soft palate and the pharynx may have been included. With the arrest of the disease the fever begins to recede. The membrane begins to loosen around the edges and is separated gradually or in large shreds. The throat is clear within eight or ten days. The swelling of the lymph nodes and the albuminuria disappear within the same period. The resulting anemia requires a little longer for

recovery. Similarly the irregularity of the heart action, the small pulse and the easy exhaustion may persist for some time.

Not infrequently, however, the disease terminates fatally in a week, or two. This outcome may be due to the further spread of the infection to the mucous membranes of the nose, the larynx and trachea and bronchopneumonia and myocarditis may ensue with the increasing toxicity.

Because of these serious dangers, we no longer await a spontaneous recovery from diphtheria, but we proceed at once to the injection of antidiphtheritic serum. This treatment is certain to shorten the course of the disease and to limit its local spread. If a sufficient quantity of antitoxin, say from 5000 to 10,000 units is administered in cases of the degree of severity described, it may be certainly expected, that the local infection will not spread materially. This will be especially true in cases where the toxins have had their necrotic effect, but in which the fibrin has not yet invaded the tissues. Certainly within twenty-four hours after the use of the antitoxin, further exudation ceases and a sharp zone of demarcation appears around the membranous area. The membrane itself becomes spongy and loosens up around the edges. It separates in large lamellæ and is completely disposed of in three or four days. The temperature falls within twenty-four hours and the general condition rapidly improves. Many a child to whom the antitoxin is given early will appear quite well and will sit up and play within a day or two.

In too many cases, however, the disease presents a course quite variant to that of the moderate type described. It may be of even milder, or of far more malignant character, or it may be characterized by peculiar localizations of the disease process.

Mild Forms

Among older children, in ordinary times a large proportion of cases are very mild. The tonsils will show only a small membrane. The temperature does not exceed 38°-38.5° C. (100°-101° F.) The general health is but slightly impaired. The membrane is thrown off within three to five days and in a week or ten days the patient has completely recovered. Occasionally cases are observed that are even more mild than this. In a few crypts of the slightly inflamed tonsils, small, elongated, whitish-gray patches, the size of a pinhead or a little more are formed, giving a picture of simple lacunar angina. There is very little rise of temperature. Nevertheless, these small exudates do not have the soft, cheesy character of the ordinary lacunar angina, but consist of light, but firmly adherent membranes in which diphtheria bacilli may be demonstrated. The diphtheritic nature of these lacunar exudates may sometimes be recognized clinically, particularly when they become confluent and form a single mass membrane which may spread to the uvula and the pharynx. They are also readily recognized when a severe laryngeal croup develops or when paralysis follows the seemingly mild angina. Not infrequently true diphtheria takes a course resembling a harmless, catarrhal angina. In this form the clinical diagnosis is impossible and diphtheria is only suspected, when it occurs chiefly in families or

hospitals where more severe forms appear. A bacteriologic examination reveals the true nature of the disease. These cases are especially dangerous because they are often neglected and in the absence of proper precaution spread the disease.

Severe Forms

Unfortunately one may never be certain, that a seemingly mild case of diphtheria may not and very suddenly pass into a serious one. The *grave forms*, of course, are usually severe at the onset. A child, usually of over three years of age, suddeny becomes ill with high fever, vomiting and headache and wants to go to bed. The older child may complain of sore throat and of pain in the abdomen. In contrast to the usual forms the tonsils are intensely red and swollen. Even on the first day both tonsils are covered with a large, discolored, pasty or membranous exudate. At the same time or by the second day the uvula and the soft palate are covered. The swelling of the tonsils is so great that no room is left for the enlarged uvula and inspection of the pharynx often becomes impossible, a difficulty increased by the excessive secretion of mucus. If the posterior pharyngeal wall can be seen, it will also be found covered with membrane by the second or third day. The extreme swelling of the tonsils and the entire throat makes swallowing almost impossible; the speech becomes difficult, nasal breathing is obstructed, so that the patient is forced to breath through the mouth, from which a terrible fetor arises and from which flows a thin and often sanguineous secretion. The nasal respiration is further obstructed on account of the swelling of the mucous membrane of the posteror nares or of the entire nasal chambers, which are often involved in the diphtheria process. The respiration is labored and deep, even when the lung findings are negative. Should the disease continue for more than five to seven days, large bronchopneumonia foci are often found. The lateral cervical nodes are markedly enlarged and the swelling soon extends to the peri-nodular tissue. This marked infiltration of both sides may extend to the median line, giving the neck an unusually plump appearance.

Cases in which hemorrhage occurs are fortunately rare, for they give a very bad prognosis. While it usually arises from the infected surfaces, epistaxis or even petechial hemorrhages in the skin may be observed.

The general health disturbance is severe from the first. The child is apathetic; his eyes, surrounded by dark circles, remain wide open, while an anxious, drawn expression marks his alarmingly pallid face. At intervals great restlessness obtains. The appetite disappears and although tortured with thirst he is hardly able to swallow fluids. The fever often runs high, ranging from 39°-40.5° C. (102°-105° F.) during the first few days perhaps receding later and often remittent in type. Yet even in very severe cases there may be no fever, so that temperature is no criterion of the gravity of the disease. The pulse from the first is small, frequent, very compressible. Even by the second day, it may hardly be palpable, unless marked improvement can be secured by the use of stimulants. The extremities frequently become cold and cyanotic.

The heart often shows no distinct objective changes aside from quite feeble heart sounds. The first sound at the apex is sometimes replaced by a murmur. After several days cardiac dilatation and a coincident painful enlargement of the liver may appear.

The spleen is usually enlarged also but on account of its softness the swelling is frequently undemonstrable in the living subject. Signs of nephritis are always found in the urine but the symptoms of this complication never become prominent.

In the severe types of diphtheria already described masses of streptococci are sometimes found associated with the diphtheria bacilli in the discolored and at times gangrenous membranes of this so-called *septic diphtheria.* Since streptococci are constant inhabitants of the oral cavity and their increase may be predicated in every inflammatory condition affecting it, it follows that they may be found in large numbers in every case of diphtheria. In this septic form of diphtheria they are not always so very abundant, so that we must still suppose that its serious symptoms are essentially traceable to the diphtheria bacillus. It is better therefore to use Heubner's designation of *malignant diphtheria* rather than the term septic.

If these cases are not treated with large doses of antitoxin from the very onset of the attack, they usually die by the end of the first or the beginning of the second week, after showing signs of cardiac weakness, of intoxication or of severe broncho-pneumonia. The disease does not spread to the larynx and trachea as often as it does in the milder forms, but nasal diphtheria may be present from the very beginning. Early serum treatment may save a large per cent. of the malignant cases. It is true, even then, that the outlook is not very bright for children under five years.

If recovery does set in the membranes will disappear about the end of the first week, but ulcers are frequently exposed, especially on the uvula, which require a longer time to heal. Convalescence is very gradual and may occupy many weeks. During all this time cardiac weakness may still threaten a fatal termination. Sporadic cases, which cannot be saved despite the employment of antitoxin on the first day, are still occasionally seen but fortunately they are now very rare. In such instances, blame for the fatality is to be laid not only to an unusual degree of virulence of the diphtheria bacillus, but also to an extraordinary susceptibility, either local or general, in the individual. When death occurs within the first few days, it is usually preceded by signs of cardiac weakness. When it occurs later, evidences of nephritis are commonly added. The rare cases in which gangrene develops and changes the diphtheritic patches to discolored brownish readily bleeding masses which give a cadaverous odor, are always serious and almost always fatal.

Special Localizations of Diphtheritic Membranes

The most common form of diphtheria begins, as already suggested, upon the tonsils and may extend to the fauces and the pharynx. From these parts the disease frequently spreads to other surfaces, which may indeed be primarily affected. This often results in great diagnostic difficulties.

NASAL DIPHTHERIA

The spread of diphtheria to the nose is quite common and all the more so the younger the child. The exudate, usually appearing first on the tonsils or in the pharynx creeps up along the lateral pharyngeal pillars or over the superior surface of the soft palate to the posterior nares and thence to the nasal chambers. Often, however, the pharynx remains free and symptoms of nasal diphtheria manifest themselves very quickly after the appearance of the disease on the tonsils. This may be readily understood, since the exudate may creep up the posterior surface of the tonsils, where it cannot be seen and since in tonsillar diphtheria the inflamed pharynx always contains diphtheria bacilli.

The onset of nasal diphtheria is not easily determined. At first there is evidence of difficult nasal breathing due to the swelling in the region of the posterior nares. An obstructed snoring breathing may frequently, however, be the result of stenosis of the pharynx resulting from enlargement of the tonsils and the pharyngeal structures. Only when a slightly purulent fluid flows from the nose, at first often of unilateral appearance, may one be certain that there is nasal diphtheria. As compared with that of ordinary coryza the secretion contains less mucus and is often stained a brownish or reddish color; it erodes the nostrils and the upper lip. The nose very soon becomes obstructed. Upon close inspection the alæ of the nose and the surrounding tissue are seen to be markedly swollen, injected and covered in large areas with a dried secretion. Frequently no membrane is visible, its formation usually beginning at the choana and not extending very far forward. Consequently, in the case of small children, it cannot be discerned without the use of a speculum with an attendant to hold the child during the examination. At times, however, the exudate extends so far forward, that it can be detected by simple inspection or with the aid of a reflected light, on the alæ nasi or the septum. Sometimes pieces of the membrane are forced out by sneezing or can be removed with the forceps.

At the onset of nasal diphtheria the submaxillary lymph nodes become more markedly swollen. The parts surrounding the nose may be edematous and reddened by an erysipeloid inflammation.

If nasal diphtheria is consequent upon or accompanies pharyngeal diphtheria it usually causes no new symptoms, but simply aggravates existing conditions and may lead to an increase of fever. It makes the prognosis much more grave and favors the development of such secondary infections as broncho-pneumonia or sepsis.

Not infrequently, however, nasal diphtheria is primary and presents the only localization of the disease. Unless the case occurs during an epidemic the actual condition is easily overlooked and a diagnosis is made only when a pharyngeal membrane or a membranous croup develops or when the serious condition of the child's general health indicates, that the case is other than one of simple coryza. In such an event the use of antitoxin often comes too late, for nasal diphtheria is under all circumstances a very dangerous condition and its early diagnosis is extremely important.

It follows, that every febrile coryza with which there is marked constitutional disturbance, in which there is an enlargement of the submaxillary nodes and in which a thin fetid purulent or even blood-stained erosive discharge is present flowing from one or both nostrils, should arouse suspicion of diphtheria. This is especially true during an epidemic of diphtheria or when there are other cases of the disease in the house.

Primary nasal diphtheria is especially common in infancy. Indeed it is almost the only form of diphtheria observed among infants during their first few months. Great difficulty is often experienced in recognizing it, since one cannot depend upon the formation of visible membranes and the bacteriologic diagnosis presents some special obstacles. It is to be remembered that it is common among infants to find virulent diphtheria bacilli in ordinary coryza or even upon apparently normal mucous membranes. In such cases one must constantly be on guard. Particularly among infants cases of simple coryza are not infrequently to be found, which do not affect the general health, but in which diphtheria bacilli can be demonstrated and upon which a fatal nasal diphtheria develops rapidly after several weeks of gradually increasing fever.

Cases of obstinate rhinitis in children of exudative and scrofulous diathesis often resemble nasal diphtheria at certain points. There is a purulent discharge, the erosive quality of which may lead to the development of ulcers upon the skin or mucous membrane of the alæ nasi and the upper lip. These ulcers may be covered with a membrane similar to that which appears on such erosions in nasal diphtheria. In such cases a bacteriologic examination alone will determine a diagnosis.

In later childhood rare instances are seen of a purely local disease, a membranous rhinitis, which may last for several weeks or even months. In these cases the diphtheria bacillus is usually absent, although the so-called pseudodiphtheria bacillus may be found. Since such a long persisting rhinitis with these diphtheroid bacilli present may produce true serious diphtheritic infections, which may be conveyed by contact to other persons, it is well to treat it as true diphtheria.

AURAL DIPHTHERIA

In young children, diphtheria of the pharynx or more often of the nose, may cause an otitis media. Oftentimes this results in a perforation of the membrana tympani and a purulent discharge from the ear. In these cases one has to deal with a secondary pyogenous infection passing up the Eustachian tube and only occasionally with true diphtheria leading to membrane formation in the tympanic cavity. If the latter does ensue, it may cause deep-seated destruction and permanent deafness while a simple otitis usually leads to no bad results.

DIPHTHERIA OF THE LARYNX, TRACHEA AND BRONCHI

A frequent and much dreaded localization of diphtheria is in the larynx and the deeper air passages. The younger the subject the more frequently does this extension of the disease from the tonsils, pharynx or nose occur.

It may be of coincident development or it may follow the primary onset in from three to seven days. Sometimes the beginning of the disease in the larynx is the first indication, that a slight coryza or an insignificant catarrhal or lacunar angina is of diphtheritic nature. Too much emphasis, however, cannot be laid upon the fact, that *laryngeal diphtheria* is often *primary*, since there are still many physicians, who to the prejudice of their small patients consider the condition an uncommon one and think, that they can exclude diphtheritic disease in cases of hoarseness or laryngeal stenosis, if no membrane is visible in the pharynx, In the cold season during attacks of measles and influenza, indeed at any time when the mucous membranes of the respiratory tract are injured, primary laryngeal diphtheria most frequently appears, while the pharynx and the nares may be and often remain free of the disease. Whether the laryngeal diphtheria in these cases is actually primary as supposed, or is preceded by a slightly diphtheritic throat, which has been overlooked is not important for practical purposes. But it is important to know that in primary diphtheria of the larynx cultures developed from the mucus taken from the apparently unaffected pharyngeal wall, will show diphtheria bacilli.

The first indication of laryngeal diphtheria is hoarseness. This symptom gradually becomes more and more severe and continuous until in a few days the voice, increasingly toneless is entirely aphonic. At about the same time and sooner in young children than in older ones, the inspiration grows increasingly strenuous and noisy without intervals of improvement by day and without benefit by treatment as in pseudocroup. The cough usually becomes very dry, painful and lacking in tone.

The rate at which the disease develops varies greatly. A narrowing of the glottis occurs within a very few days and this leads to inspiratory retraction of the epigastrium and the suprasternal notch. If the lungs remain clear and there is no rickets, the respirations decrease in number and increase in depth. Contrasting with the inspiratory conditions obtaining in bronchitis or pneumonia the retraction of the neck above the clavicles is added to that of the epigastrium, the attachment of the diaphragm and of the larynx being drawn downward with each inspiration.

According to the primary or secondary nature of the laryngeal infection, the throat will appear normal, perhaps slightly injected or will show a membrane upon the tonsils or the pharynx. If a strong narrow tongue blade is placed far back into the throat and firm pressure is applied to the tongue, one may nearly always succeed in seeing the thickened and reddened epiglottis. Not infrequently a whitish membrane at its edge reveals the diphtheritic nature of the condition at once. Rarely is it possible to see the arytenoid cartilages, which are always markedly swollen and reddened. During an attack of coughing one can often see a tenacious yellow secretion pushing up through the glottis. If one finds no evidence of diphtheria either in the pharynx nor on the epiglottis, the appearance upon ordinary inspection is no different than that of a simple laryngitis. In older children it is occasionally possible however, to see the membrane formation

on the false and true vocal cords and even in the upper end of the trachea by aid of the laryngoscope.

Even in those cases in which the membrane cannot be seen by any method of examination, the question of the presence of laryngeal diphtheria does not long remain in doubt. The condition becomes gradually but progressively worse. The aphonia becomes more and more complete, the inspirations more labored and noisy, but not so ringing and audible as in pseudocroup. The increasingly difficult inspiration soon becomes insufficient to supply the oxygen requirement, in spite of the fact, that all the auxilliary muscles are acting and that the head is retracted to assist their effort. In young children the retraction of the epigastrium until it almost touches the vertebral column, the forcible indrawing of the suprasternal notch, with the marked excursions of the larynx are definite signs of a high grade laryngeal stenosis. The incomplete decarbonization of the blood in the lungs causes cyanosis, first to be noticed in the lips and the finger-nails. The air hunger makes the child nervous. Finding no comfortable position, with a frightened look he throws himself about the bed, sits up with head thrown back or clings to the head of the bed, striving to get his breath. From hour to hour the stenosis increases to the point of threatened asphyxiation. In a coughing fit the shortness of breath becomes alarming. No air can be forced through the contracted laryngeal opening with the most forcible respiratory efforts. The face and hands grow pale; cold, clammy perspiration covers the body and the increasing stupor passes into unconsciousness. On a final strenuous effort the patient may throw out the tenacious mucus or even a piece of membrane, which had covered the glottis and gradually recover. Frequently the child succumbs in such an attack although the fatal seizure is usually preceded by a few lesser ones.

Intubation or tracheotomy performed at the proper time may prevent a fatal outcome. If one or other of these expedients is not resorted to in season, asphyxia follows. The pulse grows smaller and more frequent and the heart finally stops during an inspiration. The increasing pallor may deceive the inexperienced as to the immediate need of oxygen supply. The patient's extreme effort to get air gives way to a rapidly deepening apathy, which lapses into somnolence and eventually into deep coma. In a few hours or within a day or two death ends the struggle. At autopsy only the inner surface of the larynx may be found covered with a thick membrane. This may, however, extend far down into the trachea or even into the bronchial tree. While in the pharynx the membrane is of more distinctly diphtheritic type, in the larynx and trachea it is often very superficial, structurally speaking of a croupous form and for this reason as the result either of effort or of mechanical interference may be the more readily expelled in large fragments or as a complete mold. The laryngeal stenoses which leave the bronchi free are especially favorable for operative measures. In these cases the results of tracheotomy or intubation are wonderful. The patient, who severely cyanotic, was threatened but a moment before with asphyxiation, regains his normal color, his breathing becomes quiet and normal and exhausted, he falls into a restful sleep. To-day it is possible to count on

recovery in a large number of cases by the use of antitoxin. Before the advent of the serum treatment, the disease usually continued a day or two after the operation and then with the spread of the process to the bronchi of the first and second order, increasing signs of stenosis followed and soon death. Now, however, this fatal spread of the disease to the bronchi can be prevented by the employment of antitoxin.

Unfortunately cases which the physician sees for the first time in the stage of asphyxia, do not react so favorably. Tracheotomy and intubation give but little relief since the obstructive membranes have already extended to the bronchi. The continuingly labored breathing, clearly recognized even before the operation often evidences the fact. Examination may show no pulmonary signs beyond diminished breathing and a degree of acute emphysema resulting from the obstructed respiration. In other instances besides the formation of membrane in the larger bronchi, extensive foci of broncho-pneumonia may coincidently or subsequently appear and may have their part in aggravating a bad prognosis. Frequently even in severe cases the membrane does not extend beyond the bifurcation of the trachea but, nevertheless, foci of broncho-pneumonia develop. The respiration remains difficult after tracheotomy or intubation and the prognosis becomes more grave.

Further it is to be said that the course of diphtheritic croup depends upon the severity of the disease in general, as well as upon the extent of its spread to other parts. As we have already suggested, the toxicity and malignancy of the disease is by no means entirely dependent upon the area of the affected mucous membranes. In extremely malignant forms of diphtheria the trachea is very often spared. When the trachea alone is affected the mechanical obstruction to the respiration is the most prominent symptom and the evidences of general infection are sometimes slight.

Before antitoxin came into general use, cases of pharyngeal diphtheria were frequently seen in older children in which spontaneous recovery occurred in spite of the fact that incipient laryngeal infection was indicated by hoarseness and stenosis. Sometimes these symptoms were due to a simple laryngeal catarrh spreading from the pharynx but at other times they proved to be true laryngeal or tracheal croup, as shown by the ejection in coughing of pieces of membrane, or as demonstrated in fatal cases at autopsy. At the present time, physicians should refuse the responsibility of delay, in the hope of spontaneous recovery, when hoarseness develops in cases of diphtheria. It is the rule in young children that the onset of hoarseness is the signal of dangerous stenosis.

RARE LOCALIZATIONS OF DIPHTHERIA

Atypical localizations of diphtheria are infrequent. They are almost always secondary to pharyngeal diphtheria. The mucous membrane of the cheeks and lips (Fig. 165) is affected more often than the tongue. These departures show a typical membrane formation. An injury of the mucous surface favors its appearance.

Diphtheria of the conjunctiva is not so uncommon and comparatively

often it is primary. In mild cases it appears as a delicate croupous exudate upon the conjunctiva of the lids, which are moderately swollen and reddened and yield a purulent secretion. Recovery is often seen without any resulting scars. A true diphtheritic form with a firmly adherent membrane on the contrary is dangerous. The lids become intensely swollen and tense; chemosis and a markedly purulent secretion follow. In this type destruction of the cornea often occurs, if treatment with antitoxin is not instituted promptly.

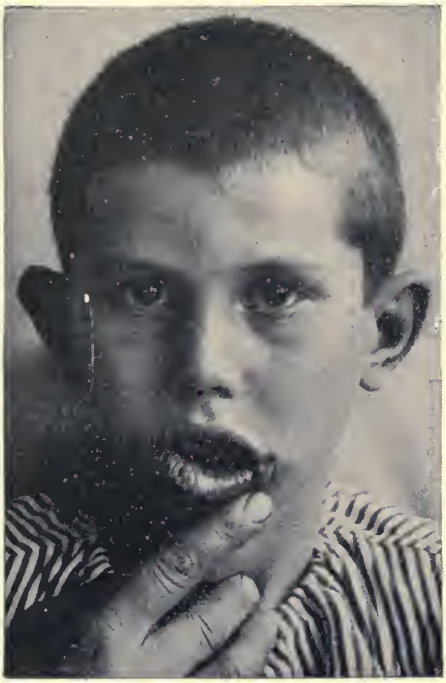


Fig. 165.—Labial diphtheria (together with pharyngeal diphtheria).

Diphtheria of the skin is not very uncommon by way of a secondary infection. It occurs notably when the skin is injured, particularly on the upper lip in cases of nasal diphtheria, in the external auditory meatus following diphtheritic otitis media and over eczematous areas. When there is no affection of the pharynx, the diagnosis may not be readily determined. The membrane may be light and readily removable or thick and firmly adherent upon a reddened base. Its character is easily determined by microscopic or bacteriologic examination. The lesion heals rapidly if antitoxin treatment is given.

Diphtheria of the vulva is uncommon. It is usually secondary to pharyngeal diphtheria as a result of preëxisting inflammatory conditions of the labia or of direct transmission of the bacilli by means of the fingers in the act of masturbation. The inner surfaces of the vulva, the lesser labia, and even the clitoris may be markedly phlegmonous and covered with a thick membrane. The local infection is very often severe and even gangrenous.

PECULIARITIES OF DIPHTHERITIC MEMBRANES

The distinctive feature of the diphtheritic membrane as compared with many other exudates upon the mucosæ is seen in its tough, elastic quality, insusceptible of crushing, dependent upon its large content of fibrin. It may often be torn off in large pieces with a forceps.

There are not infrequently cases of true diphtheria, however, in which these qualities are not recognizable. A cheesy exudate is formed, which on account of its low fibrin content, is soft and pasty and cannot be pulled off in large lamellæ. It resembles the exudate in scarlet fever, Vincent's

angina or even common angina. This soft exudate is found especially in malignant cases, in gangrenous forms and in children, who are seriously ill with some such concurrent disease as tuberculosis or with some cachexia. On the other hand, there are numbers of non-diphtheritic diseases in which membranes are formed, which contain a large element of fibrins.

THE EFFECT OF DIPHTHERIA ON VARIOUS ORGANS

Complications and Sequelæ

A swelling of the lymph nodes of the neck is not usually so marked as it is in scarlet fever. The perilymphatic tissue is only affected in severe cases. Suppuration is much more rare than in scarlet fever.

The Respiratory Apparatus.—Severe bronchitis and broncho-pneumonia are frequent sequelæ of diphtheria alike in its pharyngeal, laryngeal, tracheal and bronchial forms. By confluence of numerous smaller foci, broncho-pneumonia may become very extensive and particularly in young children is very often the cause of death. Diphtheria bacilli may be present in pneumonic foci, but streptococci are more frequent and are apt to be present in large numbers. Since severely toxic cases, even when localized in the pharynx are especially prone to develop broncho-pneumonia it may be supposed, that the toxines of diphtheria probably predispose the patient to this disease.

The pneumonia is occasionally of a hemorrhagic, infarct-like character. Exceptionally it may proceed to gangrene. Pleurisy with large and at times purulent exudate is rare.

The vascular system is often characteristically involved. Since but seldom are diphtheria bacilli found in the blood the circulatory disturbance must be considered purely toxic. In malignant cases a distinct cardiac insufficiency is observed from the beginning, death, in consequence, sometimes ensuing within a few days. The pulse is found to be small, compressible and very frequent, and in serious cases can hardly be felt. The heart itself often dilates suddenly. Systolic murmurs of muscular origin are common. In experiments upon animals profound diphtheria poisoning causes a marked fall of blood-pressure, but in the child the determination of blood-pressure is not of much significance from a prognostic viewpoint.

Cardiac death is characteristic of diphtheria. It may occur in either of two ways. The heart may fail suddenly in grave cases during the second or third week of the disease. Nausea, pallor, abdominal pains and a varyingly irregular, small pulse will often precede the heart failure. A sudden fatality cannot, however, be foreseen from these symptoms.

The so-called postdiphtheritic cardiac death is especially peculiar to diphtheria. It may occur four, six or even eight weeks after the disappearance of the local condition. Here, too, one has usually but not always, to deal with severe forms of the disease, the sufferers from which do not completely recover but remain pallid, weak, without appetite, and often have albuminuria for a long while. The pulse remains persistently small, very rapid and irregular. Forms in which a slowing of the pulse-rate occurs from time to time are particularly dangerous. In these cases the heart is

usually dilated and systolic muscular murmurs are audible. The entire picture is one of marasmus, resulting from a severe general intoxication, which doubtless affects primarily the heart. The least exertion increases the pulse-rate very markedly and may cause alarming weakness. From time to time attacks of exhaustion with sudden pallor, shortness of breath, vomiting and abdominal pain occur without any external cause. The slowing of the pulse indicates special danger. The heart is able to recover after weeks of marasmus with alarming attacks occurring at intervals. Often it does not recover its normal power for months. This is most apparent when intercurrent infections are present. Sometimes after all danger seems passed, sudden cardiac death may result and even so late as six or eight weeks after the onset of the disease. This acute heart failure is due in part to myocarditis and in part to degeneration of the vagus. In some instances embolism of the large cerebral arteries develops. The frequent circulatory weakness appearing at the height of the disease depends largely upon vasomotor disturbances.

The blood does not show any constant or typical changes. Besides a decrease of the red blood-cells the appearance of myelocytes is noted in severe forms of the disease. Leucocytosis is of frequent occurrence.

The Digestive Apparatus.—In grave cases the appetite may be entirely lost. The tongue is covered with sordes. Obstinate anorexia is always a bad sign. Vomiting occurs frequently. Serious diarrhœa often appears, another bad indication.

The Kidneys.—In many cases albuminuria appears as early as the second or third day. The urine contains large quantities of sediment and casts. The latter may be present without albuminuria. An advanced degree of nephritis with epithelial casts is infrequent. The protein content usually remains below 0.3 per cent. The convoluted tubules of the cortex are most seriously involved. The severity of the nephritis is a fair indicator of the measure of the general intoxication. The renal disturbance usually abates as the patient recovers from diphtheria. Its persistence in the form of a chronic nephritis is extremely rare. The urine hardly ever contains blood. In contradistinction to the nephritis of scarlet fever, that of diphtheria hardly ever results in edema and even more rarely in uremia.

The Nervous System.—The toxins of diphtheria seem to have a special affinity for the nervous system as they do for the heart. This reaction manifests itself in the development of pareses and paralyses.

Paralysis of the soft palate is the most common form following pharyngeal diphtheria. The so-called early paralysis should not be confused with it, since it makes its appearance during the first few days of the disease, while the membranes are still present and depends upon the inflammatory infiltration of the soft palate. A true paralysis does not as a rule develop before the second week and from that date on to the fourth week, that is, not until after the membranes have disappeared. In this condition the voice takes on a nasal quality termed rhinolalia aperta. The attempt to swallow thin fluids causes coughing and a part of the food is regurgitated through the nose. Inspection shows the soft palate and the uvula hanging

flaccid and inactive alike in phonation and respiration. Sometimes only one side is paralyzed and usually in those cases no membrane has been formed upon the normal side. Frequently both reflex action and sensation are lost in the paralyzed part. Recovery gradually occurs in from two to four weeks. Often the musculature of the pharynx is also paretic, swallowing, as a result being very difficult or altogether impossible.

Paralysis of accommodation comes next in order of frequency to that of the soft palate. In young children this event is often overlooked and it is noticeable only that fixation upon a finger approaching the eyes is not as rapid as normally. School children may complain of their inability to write and more often to read. Paralyses of the external ocular muscles and especially of one or both abducens are quite common. These result sometimes in convergent paralytic strabismus and diplopia. Diminution or suppression of the patellar reflex is usually associated with these paralyses. This change, whether associated with or independent of these paralyses, may persist for some weeks.

Fig. 166.—Postdiphtheritic paralysis of the abducens and paralysis of the facial. Seven-year-old girl.

Besides these more frequently affected muscles and indeed much more rarely, other muscle groups may be involved in diphtheritic paralysis. Actual paralysis of the lower extremities is uncommon but diphtheria often causes muscular weakness or ataxia. Not so unusual is the involvement of the neck muscles, but extension to the muscles of the face, larynx and back is exceptional. Very rarely almost all the muscles of the body may become paretic. Paralysis of the diaphragm and other respiratory muscles is of course dangerous. Seldom are pronounced disturbances of sensation in the form of paræsthesia, dimunition of the pain sense, etc., to be demonstrated. The nerve roots are not usually painful on pressure. In cases of marked paralysis, incontinence of the urine and feces are often observed. Electrical tests may show partial reactions of degeneration. The pathologic basis of diphtheritic paralysis is a degeneration of the peripheral nerves. Cases in which a membrane forms on only one side and in which the same side of the soft palate is paralyzed, or in which after diphtheria of the skin, its underlying muscles are paralyzed, seem to indicate that the diphtheritic toxin travels from the affected areas to the central nervous system along the nerve paths. Unless the disease proves fatal nearly all of these paralyses recover without leaving any permanent injury.

The Skin.—In the early stages of diphtheria although rarely before the third day, an erythema sometimes makes its appearance. It may be diffuse

but it is usually a narrowly localized matter. Its character is generally that of a rubeolar, scarlatinal or urticarial type. Such an exanthem is transitory and usually disappears within a day or two. As a rule it may be readily distinguished from a serum exanthem by the date of its appearance. Very malignant and rapidly fatal cases show small discrete hemorrhages in the skin of various parts of the body. These are sometimes associated with epistaxis and with bleeding of the pharyngeal surfaces affected by the disease.

THE DIAGNOSIS OF DIPHTHERIA

The diagnosis is seldom difficult where one has to deal with a distinct membrane of the usual physical qualities, situated on the tonsils or in the pharynx. In all doubtful cases one must fall back upon the bacteriologic diagnosis. Nevertheless, an experienced observer will make a correct diagnosis in the great majority of cases upon the clinical findings alone. In questionable cases patient observation will often clear up the problem, since the formation of a membrane together with other symptoms as increasing laryngeal stenosis become more apparent.

Differential Diagnosis

Catarrhal angina can be distinguished from diphtheria only by bacteriologic examination. Tonsillar or pharyngeal diphtheria always has a catarrhal inflammatory prodromal stage, which is followed in a day or two by membrane formation.

Diphtheria quite often begins with lacunar exudates and may so continue throughout its course. Usually, however, the small islands of exudation fuse into a single membrane in a day or two. In simple lacunar angina the exudates in the individual crypts may become confluent and cover the entire tonsil or a large part of it. Usually, the differentiation is not difficult. In lacunar angina, which runs its course without fever more rarely than does diphtheria, the content of the crypts is yellowish, purulent and soft; it is easily removed with an applicator armed with dry cotton; it has usually a bad odor. It is readily broken up into a soft paste when rubbed between cover glasses. The diphtheritic exudate of the lacunæ is more whitish and is adherent. It cannot be removed with the applicator, but if torn off with forceps it proves to be a firm and elastic membrane.

Greater difficulty is experienced in the differentiation of scarlatinal angina and Vincent's angina. In scarlet fever the exudate in the pharynx often presents, at the outset, the same characteristics of a tough fibrinous membrane, so that at this time, if the exanthem is wanting, the clinical differentiation is often impossible. The reddening of the throat, however, is usually much more intense in scarlet fever than in diphtheria. Later the appearance of the rash, the strawberry tongue, etc., aid in establishing a correct diagnosis. The exudate in scarlet fever usually contains less fibrin, is more deeply imbedded in the mucous membrane and more readily results in ulcer formation. A favorite location of the scarlet fever necrosis, from which the diphtheritic membrane is usually absent is on the anterior pillars of the fauces directly in front of the tonsils.

Vincent's or ulcerative angina resembles pharyngeal diphtheria so closely, that it is usually confused with it unless a bacteriologic diagnosis is made (see page 251). This discovers diphtheria bacilli in the one case and spirochætes with fusiform bacilli in the other. In the ulcerative angina the exudate frequently occurs upon one tonsil alone. It is soft and adherent but contains little fibrin. It has a characteristic foul odor and after recovery commonly leaves a distinct ulcer. The fever and the general health disturbances are slight.

With a little care, unusual forms of thrush, aphthous, herpetic, or luetic angina and Bednar's aphthæ covered with membrane, hardly need to be confused with diphtheria. Various bacteria, as pneumococci, streptococci, etc., occasionally cause membranous exudates of the pharynx, the nature of which can be determined by bacteriologic examination alone. Severe parenchymatous tonsillitis sometimes develops a veil-like membrane resembling that which is seen in the early stages of diphtheria.

Differential Diagnosis of Affections of the Larynx

When increasing hoarseness develops coincidently with pharyngeal or nasal diphtheria, or appears a few days later, the diagnosis of laryngeal diphtheria may be definitely made. When, however, there is no other evidence of diphtheria, a prompt diagnosis may be difficult. With a rapidly increasing hoarseness, leading to aphonia and to more and more marked stenosis, with attacks of suffocation, diphtheria is always to be suspected. A streak culture in agar of the mucous from the pharynx or the larynx will usually show the diphtheria bacillus.

Pseudocroup hardly ever causes any serious doubt. This condition commonly develops suddenly as the child is dropping off to sleep and in most cases without any premonitory signs. It leads to a degree of hoarseness and to intense, but rapidly subsiding stenosis of the larynx. The cough, of a ringing, strident quality, and the inspiratory retraction are very intense. The speaking voice is much less affected. The inflammation of the larynx below the glottis which occasions some difficulty in the introduction of a tube beyond the vocal chords, closely resembles true croup.

The retropharyngeal abscess of young subjects causes hoarseness whenever the inflammation of the mucous membrane spreads to the larynx. This, together with the existing stenosis of the pharynx, may simulate croup. The true condition is revealed by careful inspection or more readily still by palpation.

Severe acute laryngitis, often occurring in the course of acute infectious disorders of the upper air passages, as influenza, la grippe and, particularly, measles, offers special difficulties. In measles the symptoms are often absolutely identical with those of diphtheritic croup (see page 609). To this fact is added the difficulty that, combined with measles, true diphtheria usually affects the larynx and the trachea.

Laryngeal and tracheal stenosis caused by papilloma, goitre, hyperplasia of the thymus, tuberculosis of the bronchial nodes, etc., may usually

be excluded very readily by a careful study of the history, with painstaking observation of the case.

Hoarseness and laryngeal stenosis are not uncommon in young children, as results of the presence of foreign bodies, which while located in the larynx cause attacks of suffocation and, later, wedged in a bronchus, lead to a mistaken diagnosis of croup. An inadequate history of an attack of suffocation and hoarseness, coming on suddenly during a meal, or while at play, always justifies the suspicion of a foreign body, which should dictate a Roentgen examination.

Several instances have been observed in which tracheotomy or intubation has been performed for the relief of an intense dyspnœa, with inspiratory retraction and cyanosis, but without marked hoarseness, in which the operation has given no relief nor has it prevented a fatal termination. Autopsy has revealed neither diphtheria nor any other essential condition of disease of the respiratory organs. Simply a status lymphaticus was present, which was necessarily held responsible for the severity of the disturbance and its result.

The postdiphtheritic paralyses are so generally characteristic, that they enable one to make a diagnosis of previous diphtheria, even in the absence of history or with a record of an attack so slight that its nature was overlooked. Their most significant sign is a paralysis of the soft palate.

Bacteriologic Diagnosis.—One cannot sufficiently emphasize the fact, that where there is any doubt whatever concerning the diagnosis, material removed from the throat must be subjected to bacteriologic examination. Usually culture tubes can be sent to municipal or state institutions for diagnosis. In the course of this discussion we have called attention to the fact, that even thick elastic membranes are not always diphtheritic and reversely that diphtheria often runs its course without the formation of a membrane or at least in the absence of any characteristic deposit.

A small piece of the exudate to be examined is removed with the forceps or other instrument; it is washed in pure water and crushed as fully as possible between two microscopic slides. As an alternative, it may be sufficient to wipe off the membrane with a cotton-covered applicator. If there is no membrane, a little mucus removed from the suspected mucous membrane either on the pharyngeal wall or in the suspected larynx, is spread upon a slide. This is dried and stained with Loeffler's methylene blue. If diphtheria is present there are found, beside numerous cellular elements, debris and often threads of fibrin, various kinds of bacteria and among them large numbers of diphtheria bacilli. These may even predominate. They are recognized as thick, almost club-shaped rods, which stain irregularly and are characteristically found in nests or clumps lying at sharp angles to each other (see Fig. 167). This simple technic which any physician can carry out with a little practice is usually satisfactory, so far as the examination of the membrane is concerned. It must be borne in mind, however, that even in true diphtheria, and especially with old membranes, it is often impossible to find the bacillus. In that event and particularly if only mucus has been removed from the throat, it is necessary to make a culture upon Loeffler's blood serum, on which the organism grows very freely. For fur-

ther and absolute identification, an inoculation test upon animals may be made. This gives the most definite differentiation between the true bacillus and the non-virulent forms of pseudodiphtheritic bacilli. This identification cannot be made absolutely with Neisser's double stain of fresh cultures for polar bodies, since this polar nucleation is sometimes absent in the case of true diphtheria bacilli and is definitely lacking in the pseudo-forms. For a more complete description of the bacteriologic diagnosis of diphtheria, the reader is referred to standard text-books of bacteriology.

The prognosis of diphtheria depends to a great degree upon the character of the disease. The type is generally more severe during epidemics than

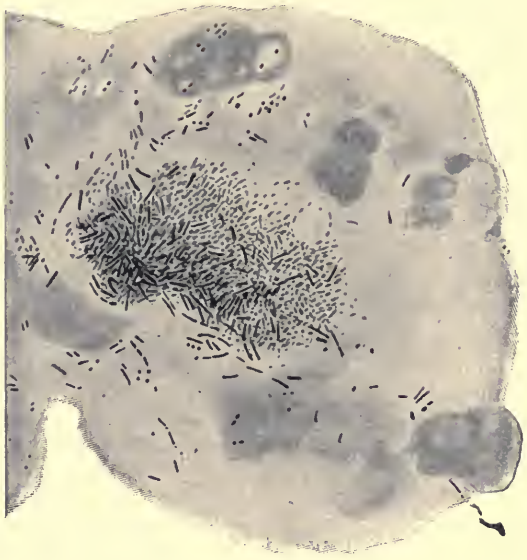


Fig. 167.—Diphtheria bacilli. Smear from exudate on tonsil. Leitz $\frac{1}{12}$, ocular 3. Tube length 16.

it is in sporadic cases. The younger the patient the less favorable is the prognosis, so that infants hardly ever escape unless under treatment with antitoxin. The disease is also especially fatal among tuberculous and cachectic subjects. The spread of the diphtheritic process to the nose or the larynx always reduces the chances of recovery. Localized forms, confined to the tonsils, have a generally favorable prognosis. An intense inflammation in the throat, a gangrenous type of infection, the unusual enlargement of the lymph nodes, an extensive infiltration of the perilymphatic tissue, a severe general toxicity, cardiac weakness, etc., are all indicative of a particularly malignant form of the disease. Even with a seemingly satisfactory course and after recovery from the purely local manifestations, the prognosis must always be made with reserve, since diphtheritic paralyses or serious myocardial changes causing sudden cardiac death may follow later.

At the present time, the prognosis depends chiefly upon the early administration in sufficient quantities of antitoxin. The discovery of antitoxin by Behring may be said to be the greatest triumph and the richest blessing of scientific therapeutics, as a result of which diphtheria has largely lost its terrors. There is hardly another serious disease upon which the physician can look with so great certainty and assurance as he can upon diphtheria to-day. Early treatment is, of course, essential. Cases that receive sufficient doses of antitoxin on the first or second day of the disease recover almost without exception, as is shown in the tabulated statement on page 663. With every day, yes, with every hour that the injection of antitoxin is delayed the chances of the child are diminished. The enthusiastic supporters of this therapy must admit, however, that there are occasional cases in which the individual predisposition is extraordinarily great and which cannot be saved even though antitoxin is given the first day. These exceptions do not alter the fact that early injection will cure almost all cases—a fact which is supported by the history of recent epidemics in Berlin and Hamburg. That there are such exceptions makes the duty to employ the remedy early the more imperative. The failure to use it at the proper time is an inexcusable and often fatal error.

The prognosis of croup during measles, in which the membrane formation rapidly spreads to the smaller bronchi, is relatively bad and demands early and energetic serum treatment.

Prophylaxis.—Prophylaxis requires the isolation of the patient, who must be sent to a contagious hospital if the isolation cannot be achieved successfully at home. After the recovery or removal of the patient the room must be disinfected. The other members of the family should be carefully watched and throat cultures taken. If necessary, a prophylactic injection of antitoxin may be given.

The avoidance of the disease is greatly favored by proper hygiene, by frequent gargling with cold water, to the glassful of which five drops of tincture of myrrh may be added, and by cleanliness of person and dwelling. The patient should not be permitted contact with other members of the family for fourteen days after the disappearance of the local symptoms. If diphtheria bacilli are still found on the mucous membrane, isolation should be continued until they have entirely disappeared. Even a longer time should elapse before the patient is allowed to return to school. If several cases of diphtheria appear in a school or in a children's hospital it is usually possible to weed out the healthy bacillus carriers by systematic bacteriologic examination of all the inmates. In infants special attention should be paid to the nasal discharges. By isolating carriers the spread of infection is often successfully checked.

Treatment.—The serum therapy, the basic principles of which are generally known, is the most important method of treatment to be employed in this disease. The danger from diphtheria is largely due to the toxin produced by the bacilli, although the organisms themselves reach the blood stream or the viscera in very small numbers. Spontaneous recovery occurs as the result of the action of certain protective substances present in the

body and more especially by the action of specific antibodies, the diphtheria antitoxins which the body forms to combat the circulating diphtheritic toxin. If the organism proves able to form sufficient antitoxin at the right time to achieve an active immunity recovery takes place. Otherwise the disease is fatal. This antitoxin, however, does not affect the bacteria, and therefore convalescents and those who have fully recovered from the disease may harbor virulent diphtheria bacilli for a long time. The significance of the serum therapy lies in the fact that it supplies an artificial antitoxin to aid the organism in combating the disease toxin. By repeatedly injecting the horse with the germ-free filtrate of a virulent culture of diphtheria bacilli, the blood-serum of the animal acquires in time a high degree of immunity, that is, it contains a large amount of diphtheria antitoxin. The term immunity unit (I. U.), has been generally adopted to signify that amount of antitoxin-containing serum which will completely neutralize the effects of 100 lethal doses of toxin in a guinea pig weighing 250 grams. We speak of simple antitoxic serum when this unit is contained in 1 c.c. of serum. In late years the sera in use are usually 400 to 500 times as strong as the simple serum, 1 c.c. containing 400 to 500 I. U.

Dose.—For prophylactic purposes, 500 I. U., or, according to Schick, 50 I. U. per kilo of body-weight, are sufficient to give a passive immunity lasting for two to three weeks. In the treatment of the disease in its several forms the following doses may be given by intramuscular injection: For localized pharyngeal diphtheria, 1500 to 2000 I. U.; for nasal or laryngeal diphtheria, 3000 to 4000 I. U.; for malignant diphtheria, 5000 I. U. These doses may be given irrespective of the age of the patient. If no results are observed within twenty-four hours, the dose is to be repeated once more.

American authors are definitely of the opinion that the best results are obtained in the treatment of diphtheria by larger doses of antitoxin. Park recommends the following dosage:

	Mild cases.	Early moderate cases.	Late moderate and early severe*	Severe and malignant*
	Units	Units	Units	Units
Infants (10-30 pounds in weight under 2 yrs.)	2000-3000	3000-5000	5000-10000	7500-10000
Children (30-90 pounds in weight under 15 yrs.)	3000-4000	4000-10000	10000-15000	10000-20000
Adults (90 pounds or over in weight.)	3000-4000	5000-10000	10000-20000	20000-50000
Method of administration advised	Intramuscular.	Intramuscular	Intravenous	Intravenous

*When given intravenously, the smaller amounts stated. When children or adults have been exposed to diphtheria they may be protected from the disease by the subcutaneous administration of from 500-1000 units, a smaller dose being sufficient for infants and young children. The protection does not last longer than from 2-4 weeks.

Recently, Schick determined by experiment that the maximal results of antitoxin are obtained with 500 I. U. per kilo of body-weight. In light cases, he recommends that 100 I. U. per kilo be injected, less than this having no action; in severer cases, proportionately to the seriousness of the attack, he increases the dose up to 500 I. U. per kilo.

A satisfactory effect within twenty-four hours, is announced by a fall of temperature (Fig. 168); a decrease of the pulse-rate, and an improvement in the general condition. During this period the membrane formation ceases or shows but very slight extension in those areas in which necrosis had developed before the injection was given. Similarly, laryngeal stenosis may increase during the first twenty-four hours after the injection, subsequent to which decided improvement is to be expected if the treatment is successful. The favorable influence of the serum is first manifest in a

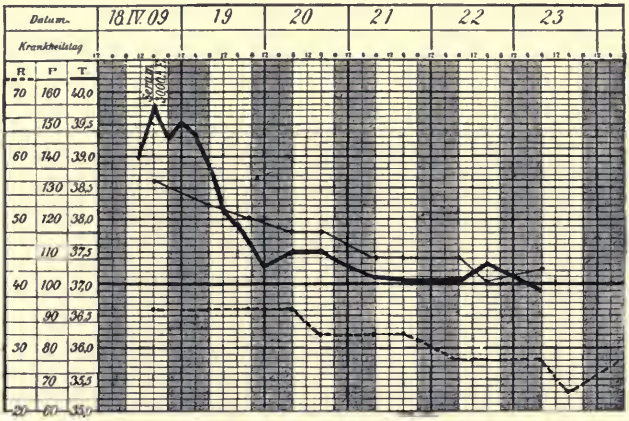


Fig. 168.—Pharyngeal diphtheria in six-year-old girl. Rapid fall of temperature after injection of antitoxin.

distinct red area of demarcation around the membrane, which soon curls up at the edges, becomes loose and soft and in from two to four days is entirely loosened and cast off. If no such distinct results are visible within twenty-four hours a second injection should be given.

In the administration of antitoxin, absolute asepsis must be observed. An ordinary 5 c.c. syringe may be used. The site of the injection may be covered with cotton and collodion.

Since resorption from the muscle is much more rapid than from the subcutaneous cellular tissue, the hypodermatic method may be entirely discarded and all injections be given intramuscularly. The outer side of the thigh or the gluteal region is generally preferred. The intramuscular is less painful than the subcutaneous injection. The weight of experience in animal experiments shows that intravenous injection acts much more rapidly than either, and may often save life where the subcutaneous or even the intramuscular method would be in vain. For this reason the intravenous use of the serum has come into more and more frequent practice

during the last few years. According to all reports supported by the writer's experiences, its results are very favorable and unattended by special danger of anaphylaxis. In this clinic, however, the intramuscular injection is the routine practice; the intravenous method being reserved for severe cases when the veins of the arm can be used without special preparation. Even in these cases the latter method is not employed if the patient has received a previous injection within a few months.

The mode of operation of the serum is not entirely clear. Its action is probably, in the main, one of neutralization, rendering the circulatory toxins inactive. When the toxin has already become anchored to the cell substance as, for instance, in the heart or the nerve tissue, the use of antitoxin will accomplish little or nothing. It follows that the objections of dissenters to the use of antitoxin, who deny its action because it does not prevent myocarditis or paralysis, are not tenable, since these injuries have been suffered before the introduction of the serum. Even the alleged increase of the diphtheritic paralyses, which is claimed to have occurred since the introduction of antitoxin, does not sustain these views, since many severe cases which formerly would have proved rapidly fatal now survive and develop sequelæ. With many other authors, I have gained the impression that paralyses are less common where early injection is had than formerly.

The actual value of the serum therapy is proved conclusively by the reduced fatality of the disease as compared with that of former years.

According to statistics gathered by Deycke in 78,028 cases, the mortality after serum injection is shown by the following figures:

Date of injection	1st day Per cent.	2nd day Per cent.	3rd day Per cent.	4th day Per cent.	5th day Per cent.	6th day Per cent.	After 6th Day Per cent.
Deaths	4.3	7.6	14.7	19.7	31.6	31.3	31.6

Still more convincing is the fact that under serum treatment about two-thirds of the operative cases recover, while without it only one-third escape. Further the canula and the tube need not be left in place as long as they were before antitoxin was used. Another fact which bears upon the benefit derived from the antitoxin treatment of diphtheria is that with its use the disease hardly ever spreads to the larynx and the trachea—an extension which was formerly of common occurrence. Again it is noted, in distinct contrast to earlier experiences, that with antitoxin treatment threatened laryngeal stenosis almost always abates without necessitating operative interference. The opponents of serum therapy, who are not nearly so numerous as they were a few years ago, have but one argument left—the danger of injuries to the child from the injection. These injuries, however, are so uncommon and usually so insignificant that they cannot be considered in face of the wonderful benefits the method has conferred. Nevertheless, serum disease is to be recognized.

Serum Disease.—If the serum of one animal is introduced, either subcutaneously or intravenously, into another animal of a different species

pathologic changes often occur. For human beings this fact has taken on a special significance on account of the frequency of the use of antidiphtheritic serum. Such changes may appear, similarly, when antitetanic or antimeningococcic serum, etc., are used, since they, too, represent sera

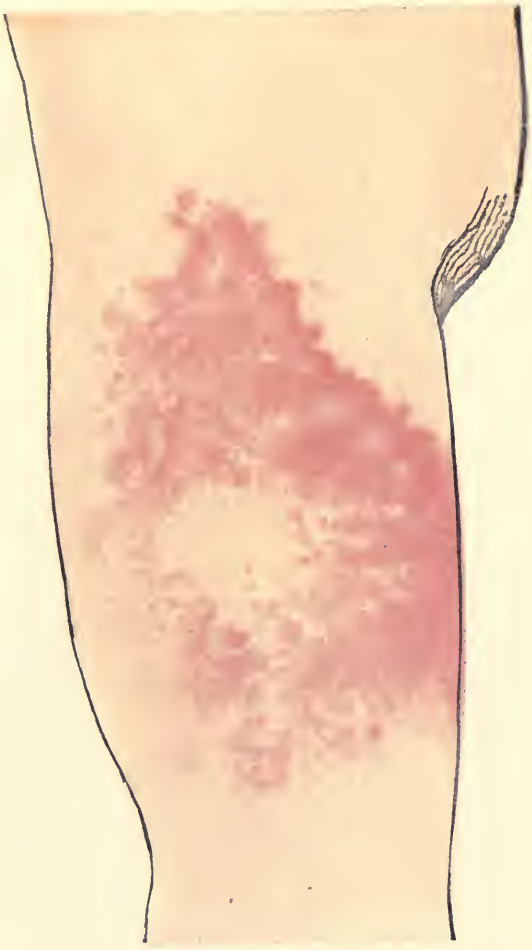


FIG. 169.—Serum exanthem, resembling measles and erythema appearing ten days after injection in the neighborhood of the injection.

from animals of different species and do not merly consist of the specific antibodies derived from them.

Serum disease has been exhaustively studied by von Pirquet and Schick. After the first injection with antidiphtheritic serum, that is, of horse serum containing antitoxin, a painful inflammation develops within a few days in the lymph nodes draining the region tributary to the point of injection. This inflammation disappears in the course of two or three weeks. The

most frequent clinical phenomena of antitoxin therapy are the serum exanthemata, which make their appearance in a varying percentage of cases and usually from the seventh to the twelfth day. The exanthem commonly begins at the point of injection and may be confined to this immediate region (Fig. 169); or it may be scattered irregularly over the entire body. The most common form is an urticarial erythema, causing intense itching. It often appears in extraordinarily large efflorescent patches with measles-like areas lying between them. Sometimes the entire exanthem resembles that of measles which it simulates the more closely in the fact that the conjunctiva are occasionally involved. A most uncommon form is the scarlatinoid exanthem, which is most likely to cause diagnostic difficulties, since the opportunity of acquiring scarlet fever in wards reserved for diphtheria, is often given. The fact that the serum exanthem does not affect the mucous membranes of the mouth is important in the matter of differential diagnosis. It is often accompanied by fever which may continue for several days (see Fig. 170). Arthritic pains occasionally, but less commonly, occur. During the course of serum disease a distinct leucopenia is observed as a result of the decrease of the polymorphonuclear cells. Occasionally precipitins, acting against the foreign serum, may be demonstrated *in vitro*.

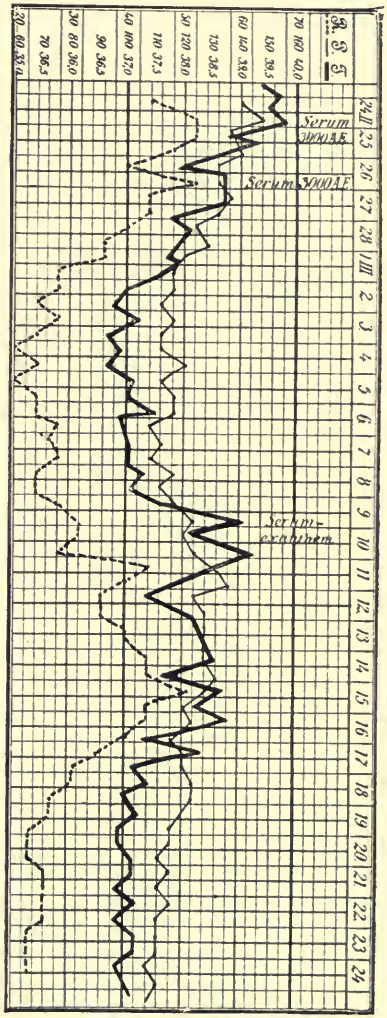


FIG. 170.—Serum fever and serum exanthem beginning twelve and ten days respectively after an injection of serum.

The human organism responds differently to the reinjection of serum. If such reinjection is made within a period of from twelve days to three or even six months indications of disease appear very rapidly. These may develop as an immediate reaction within a few minutes or in some hours after the reinjection, if the first injection had caused anaphylaxis. Usually intense edema and erythema appear at the site of injection. More rarely an intense edema of the face and a general urticaria appear, sometimes associated with dyspnœa and alarming collapse. These manifestations commonly disappear very speedily. If readministration of the serum is postponed later than from three to six months, the symptoms are apt to

resemble those following the first injection, excepting that they appear at an earlier date, generally within three to six days. This idiosyncrasy may persist for many years, so that from the appearance of such symptoms a previous use of antitoxin may be determined. Certain individuals are predisposed to the primary as well as the anaphylactic reaction. Both forms are exceptional.

Serum disease depends upon the formation in the organism of antibodies to the injected serum. These antibodies are ordinarily formed in from seven to twelve days after the first injection and they are found in the circulation for several succeeding months.

Upon subsequent occasion they may be formed more readily, resulting in the more rapid reaction. In view of the small quantities of serum used in diphtheria, serum disease is uncommon and ordinarily light. In scarlet fever, however, in which large doses of 100 or 200 c.c. are required it is often a very serious matter. Consequently scarlet fever serum is used only in severe cases. In some instances the immediate reaction may be very intense and may cause much anxiety. Nor may the possibility of a fatal termination be wholly excluded, extremely uncommon as is the event. The cause of this sudden death is not yet clear. The possibility of its occurrence emphasizes the fact that prophylactic injections should not be advised without very substantial reasons. The danger of anaphylaxis suggests the desirability of the use of the serum of some other species of animal, such as the sheep, for prophylactic purposes.

The question arises whether all diphtheritic cases should be treated with antitoxin. This is not considered necessary. The writer believes that the expectant treatment of slight localized diphtheria of the tonsils, in children of six to eight years, is justified, provided they can be watched carefully. No serious injuries have resulted from this practice. While it is realized that this position will be combated by many, reasons have not been found for withdrawal from it. For those whose experience in weighing the severity of cases is limited, it may be better, perhaps to choose the easier course and inject every patient. Similarly it is not deemed necessary to give prophylactic injections to strong healthy children of over two years of age who have been exposed to diphtheria, but are under constant observation; since the danger of anaphylaxis appearing if a later injection becomes necessary, cannot be overlooked. Since passive immunity disappears after about three weeks a prophylactic injection would need to be repeated frequently in order to prevent infection.

It may be that Behring's diphtheria vaccine will prove of great service in this respect. This causes an active formation of antitoxin, giving an immunity which lasts for a longer time. Furthermore, it is to be remembered that many persons who have not had diphtheria, nevertheless possess specific antibodies and cannot be infected with the disease. These number as high as eighty per cent. among the new-born and from fifty to sixty per cent. among older children. Intracutaneous vaccination with minute quantities of diphtheria toxin produces no reaction in these individuals (Shick), showing that the prophylactic injection of serum is unnecessary.

Some French authors recommend the daily injection of large doses of antitoxin in severe cases of diphtheria and in the event of myocardial or paralytic sequelæ. The experience of other writers accords with our own observations in throwing doubt upon this procedure. This method has been employed, however, in a number of cases, with the injection of 2000 I. U. every two days. Probably it has been useful in some instances and has proved injurious in none. Certain authorities cite good results in severe paralyses with very large doses of serum (20,000 - 50,000 I. U.).

The question as to the time to use antitoxin is to be further considered. It has been already said that the injection should be given as early as possible. In doubtful or indefinite cases it should be employed immediately and even without awaiting the report of the bacteriologist, since very valuable time may be lost and life endangered by delay. Certain cases, in which postponement of the use of antitoxin is justified, have been mentioned.

No other form of treatment, accompanying the serum, is necessary, unless operative interference is indicated for the relief of stenosis of the larynx.

Local treatment of the pharynx by pencilling and insufflation, formerly so common, is an unnecessary annoyance and may do harm because of the fear it excites in the mind of the patient. General hygiene of the mouth, however, is indicated. Older children should be required to rinse the mouth and gargle the throat with a weak solution of boric acid or hydrogen peroxide (1 per cent.). In younger children an attempt should be made to cleanse the mouth by spraying or irrigation with a 2 per cent. solution of hydrogen peroxide, if it can be done without arousing too much antagonism. Forceful measures are justified only in extreme cases of gangrenous pharyngeal diphtheria and even then their benefit is doubtful. In robust children frequently renewed cold packs or ice-bags may be applied to the neck. These may be exchanged later for hot applications.

In nasal diphtheria, also, the local treatment is secondary to the serum therapy. Efforts should be confined to careful removal of the secretions by means of cotton tampons and to the protection of the upper lip from erosion by the use of a bland ointment. If desired, powdered bolus alba may be blown carefully into the nostrils every hour, as described by Trumpp. In ocular diphtheria the phenol-free serum may be instilled directly into the conjunctival sac, combining this treatment merely with frequent cleansing and cold applications.

In early laryngeal diphtheria a moist warm general pack, inducing perspiration, may act favorably. Frequently renewed hot applications may be placed about the neck. Apart from the general quieting effect they produce, there is no special indication for such narcotics as codein or morphin. The constant impregnation of the atmosphere with steam from a 0.5 per cent. solution of sodium chloride is obviously beneficial. Special rooms are equipped for this purpose in hospitals. In the home, resort must be had to the steam atomizer or croup kettle (Fig. 95). If this is impossible a pailful of boiling water may be placed near the bed and the latter covered with an improvised tent. If the laryngeal stenosis increases, despite the use of antitoxin and of these subsidiary measures, the threatening danger of me-

chanical closure of the glottis should be met unhesitatingly by intubation or tracheotomy.

The general treatment of diphtheria even in its milder forms, requires great care. Considering the great liability of the heart to injury, all unnecessary excitement and the infliction of pain must be avoided so far as possible. This should include all unnecessary exercise even in the use of toilet conveniences.

The diet frequently presents great difficulties. Because of the embarrassment in swallowing it must be confined to liquids. It may be better to give small quantities of food every hour or two if there is obstinate anorexia. At the outset, milk gruels, thin farinaceous puddings, cocoa and egg may be given, with a little coffee to serve as a stimulant. In severe protracted cases, freshly expressed meat-juice will prove useful. Fresh fruit juices, such as grape, orange and lemon, for younger children and stewed apple, for older ones, form a welcome addition to the limited menu. Later, the child may be allowed soft toast, finely-mashed spinach, riced potato, chopped meat, etc. Plenty of fresh water should be given. While the laryngeal tube is in place the difficulty of swallowing either fluids or solids is often increased and it is better to give all food in a semi-liquid form. This rule applies, also, in paralysis of the pharynx. By the addition of a very small quantity of gelatin (1-2 per cent.), milk is very readily brought to this consistency.

As long as there are signs of cardiac weakness or of marked lassitude, the patient should be kept in bed and that even though the local symptoms and the fever have been absent for weeks. This is not to say that slight irregularities of the pulse, with an otherwise normal heart, which may persist for months, constitute a reason for confining the patient to bed. During the acute stage stimulants are often necessary. At this period, as well as in postdiphtheritic myocarditis, caffein and camphor may be employed. In myocardial complications the combined use of caffein and digitalis seems to be efficient. In the event of serious vasomotor disturbances, which in grave cases set in rapidly, epinephrin (1:1000), in large doses, will often give satisfactory results. It is given subcutaneously in doses of 0.5-1.0 c.c. (minims viii-xv) in 10-15 c.c. (℥ii-iii) of physiologic saline solution.

The postdiphtheritic paralyses of moderate degree recover without treatment. Injections of strychnia are commonly used but are of doubtful value. They are given from three to five times a week using from 0.5-2 milligrams (gr. $\frac{1}{120}$-$\frac{1}{30}$), according to the child's age. In severe cases repeated daily injections of large doses of antitoxin (2000-4000 I. U.), may be tried as a last resort. There is no objection to the careful use of electricity to the affected nerves.

During the abatement of symptoms and in convalescence, quinine and iron preparations are useful. Removal to the country is to be recommended. The complications of diphtheria are to be treated in the customary manner. Diphtheritic nephritis requires no special attention.

The exact time at which operative interference is indicated cannot be

stated within hard and fast lines. Generally speaking, it should be undertaken whenever the stenosis causes marked retraction of the epigastrium and the suprasternal notch, or when a progressively increasing cyanosis becomes so intense as to induce stupor or attacks of suffocation. Even if the cyanosis is not excessive, but the constant stenosis and the labored respiration threaten exhaustion, the operation should not be delayed. In the home the operation must be undertaken sufficiently early. In the hospital, where the patient is under constant expert observation, greater delay is permissible. For those who crave a more dogmatic rule it may be said, that the proper time for interference is when the sternomastoid is employed as an auxiliary in respiration. The contraction of this muscle is readily recognized by palpation. Placing the thumb and the forefinger along the lower part of the outer edge of the muscle one may feel the contraction during inspiration. This may also be accomplished by holding the muscle between the thumb and the forefinger at various levels.

With the necessity for operative interference arises the question of choice between *tracheotomy* and *intubation*. Intubation is generally to be preferred, without reservation, as soon as the physician has acquired some practice. Tracheotomy has become less and less popular in most clinics. Parents who object to the use of the knife usually give their ready assent to this bloodless procedure. It requires less than a minute in its performance and can be done without anesthesia or expert assistance. Such accidents as asphyxia from the downward displacement of a piece of membrane, etc., are rare, but, nevertheless, preparation for tracheotomy must always be made lest such mishap should occur. Hemorrhage and wound infections do not occur. The tube is often permanently removable from the first to the third day.

A contraindication for intubation is seen in the existence of pharyngeal stenosis, as a result of an extraordinary hypertrophy of the tonsils. Serious embarrassment occasionally results from an obstinate difficulty in swallowing, the repeated coughing up of the tube, and subsequent laryngeal decubitus. But, in comparison, the objections to tracheotomy are many and serious. It demands trained assistance and anesthesia; it involves the danger of hemorrhage and of wound infection; a longer time elapses before the canula can be removed; frequently occurring sequelæ, such as vocal impairment, decubitus and tracheal stenosis result. The effects of intubation properly performed are at least as good as those of tracheotomy, so that it is always to be preferred. Most pediatrists reserve tracheotomy for secondary use in cases where the tube cannot be removed for four to six days, when danger of decubitus ensues. Tracheotomy is advisable also in cases in which intubation does not relieve the stenosis or when the obstruction recurs speedily. In some of the cases the difficulty is due to bronchial croup and tracheotomy, therefore, does no greater good. Occasionally tracheotomy relieves the situation by encouraging the more complete removal and expectoration of the deep-seated membrane.

The method of intubation most frequently employed is after the simple procedure devised by O'Dwyer. A bronze or ebonite tube of appropriate

form is introduced into the narrowed glottis through the mouth by means of a specially devised instrument. The patient is held upon the lap of the mother or nurse. The upper part of the body is wrapped in a blanket, which serves to confine the arms, and the lower extremities are held between the knees of the attendant. The mouth is held open by a mouth-gag, preferably of the Whitehead type, which is self-retaining. The head must be held perfectly straight and must not be bent backward. The physician sits upon a low stool directly in front of the patient. The forefinger of the left hand, introduced into the pharynx, palpates the epiglottis and the arytenoid cartilages and draws the epiglottis and the tongue sharply forward. The intubator, armed with the threaded tube, is held firmly in the right hand and the tube is introduced horizontally between the teeth and exactly in the median line of the pharynx (Fig. 171). The lower end of the tube lies at the radial side of the left index finger and is passed along this to its point, during which movement the handle of the intubator is gradually brought to the horizontal. The left index finger, exerting constant traction on the epiglottis gives way toward the ulnar side, while the end of the tube at its radial side is pushed downward, entering the glottis. This is the more easily accomplished during an inspiration, since the epiglottis is widely opened at that time. The entrance to the larynx may be covered with the tip of the finger for a moment. When it is uncovered the child usually takes a deep breath and this is used to advance the tube. When about one-third of the tube has been introduced, the palpating finger is passed around it to see that it is properly placed. The tube must be entirely surrounded by mucous membrane. Then the intubator is withdrawn, by placing the tip of the finger on the edge of the tube and pressing it completely into the larynx, again palpating the entire area to make certain that the head of the tube is completely surrounded by mucous membrane. The free end of the thread attached to the tube is left projecting from the left angle of the mouth and is fastened to the cheek with adhesive tape. The entire procedure requires but a few seconds. It should be carried out very carefully; no force is permissible. If the tube is correctly placed in the larynx, its presence

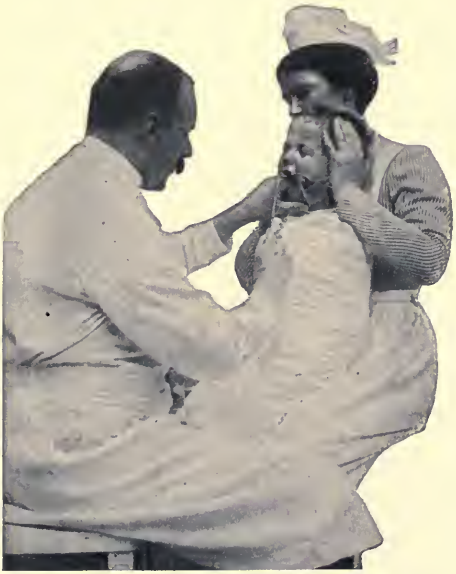


FIG. 171.—Intubation of the larynx. The mouth is held open by means of a Whitehead mouth-gag. The tube is being passed along the radial side of the left index finger which draws the epiglottis forward, into the throat.

there is recognized by the peculiar metallic tone which is given to the breathing and the cough. The beginner sometimes places the tube in the esophagus. In such an event the tube must be withdrawn by means of the thread and the operation should be repeated after a few minutes rest. An artificial passage through the lower constrictors of the pharynx can be made only by the use of brutal force.

The silk thread attached to the head of the tube is fastened, as described, to the left cheek with adhesive tape, in order that the nurse may readily remove the tube if an attack of suffocation should occur. To prevent the child from pulling out the tube its arms may be tied to the bed or be held by stiff cuffs. Any difficulty in swallowing caused by the tube usually disappears within a day or two. It is best, however, to give only semi-liquid food. The discussion of other methods of intubation cannot be undertaken here.

Sometimes the tube is coughed out after a few hours and the absence of further stenosis makes its replacement unnecessary. If it remains in place and the fever goes down the next day, as a result of the action of the antitoxin, the physician may draw out the tube after two days and see whether the patient is able to breathe without it. Severe stenosis often appears immediately or within a few hours. Whether thus removed by the physician or in the act of coughing, the stenosis may suddenly become alarming. For this reason alone, tracheotomy is preferable when the case is to be treated at home. If the tube has to be replaced after the first removal, repeated attempts at extubation should be made every other day. In most cases treated with serum, the tube may be left out permanently after three or four days. If this does not prove true it is well to perform a tracheotomy in four to seven days. Pain in swallowing and the appearance of black spots on the tube indicate decubitus. This is especially to be dreaded in croup occurring in the course of measles. It may develop alike after intubation and after tracheotomy. The technic of tracheotomy is fully described in the text-books of surgery.

Tracheotomy is much more easily performed when the intubation tube is in place. In severe asphyxia, narcosis is unnecessary. Cricotracheotomy should always be avoided, since vocal disturbances are sure to follow. In the presence of goitre, the superior tracheotomy is to be preferred; otherwise the inferior site is to be chosen. If the field of operation is scrupulously confined to the median line and the operator and his assistant each hold the soft tissues on each side of the median line symmetrically with the forceps, cutting and dissecting with the greatest care, tracheotomy is easily performed and with small danger of encountering any large vessels. The first attempt at permanent removal of the cannula is to be made, at the earliest, on the fourth or the fifth day. It is harmful to attempt progressive decannulization with cannulæ of diminished size, since this usually leads to the formation of granulation tissue.

The most efficient relief in difficult decannulization is intubation and, *vice versa*, the most efficient relief or difficulty in extubation is tracheotomy.

PERTUSSIS OR WHOOPING-COUGH

Pertussis is a specific, contagious disease, the most important symptoms of which arise from the respiratory tract and excite characteristic attacks of coughing. The disease occurs in all parts of the world. It was first described in the seventeenth century.

The causative organism has been sought in numerous researches and many different germs have been described as producing the disease. A bacillus recently discovered by Bordet and Gengou is, however, the only organism that appears, with any degree of probability, to be the true cause of the disease. These authors have consistently found a peculiar polymorphous bacillus in the sputum of patients with pertussis. This is a weakly staining bacillus, the centres, in particular, taking the stain very lightly (Fig. 172). It is very abundantly present, and even in pure culture, in the sputum of recent cases. Advanced cases show them less numerously but exhibit large numbers of saphrophytes. This bacillus is found in no other disease. It is agglutinated by the serum of individuals convalescing from pertussis. Its viability is not great and it does not resist high temperatures. Inaba has produced pertussis in the monkey by inoculation with its pure culture

FIG. 172.—Pertussis bacilli (Bordet-Gengou) after a preparation by Prof. C. Trauken in Halle.

Anatomy and Pathogenesis.—Since deaths hardly ever occur in pertussis, unless complications or secondary infections develop, the opportunity to study the pathology of the disease is very meagre. The pathologic changes are evidently very slight and, aside from alterations in the blood, consist merely in catarrhal conditions of the upper air passages. Broncho-pneumonia, foci of which are found in almost every case that comes to autopsy, is the most frequent cause of death. It must be regarded as a secondary infection. The tenacious, glairy mucus is probably the cause of the paroxysms of coughing, acting as an irritant as it passes the glottis, the posterior angle of which is the most sensitive point.

The contagion in pertussis travels almost wholly from one person to another and is probably carried in the sputum, that is by droplet infection. The secretion of the upper air passages seems to be extraordinarily infectious, since contact with an affected patient for but a few minutes suffices for the conveyance of the disease. The vast numbers in which the Bordet-Gengou bacillus, is present in the sputum of recent cases satisfactorily explains the rapid spread of the malady. Indirect transmission of the disease by healthy persons or by means of clothing is extremely rare, to say the least. It is usually supposed to have taken place when, as a matter of fact, transmission has occurred through the agency of masked cases.

Its contagiousness is marked from the very beginning of the catarrhal stage, when it still requires a week or more to determine the true character

of the disease. This is the period in which transmission generally occurs since it is the time when no precautionary measures are taken. Furthermore, the contagion is at its height at this stage. It continues during the convulsive period, but its virulence lessens rapidly. In the stage of decline its contagious quality is very slight and often seems to disappear entirely. It is safe, however, to suppose that there is still danger of conveyance as long as the child coughs.

At times the prevalence of the disease amounts to an epidemic. This happens most frequently in small communities, while in large cities where cases can always be found and large numbers of children are immune, it hardly ever assumes epidemic proportions.

The predisposition to the disease is very general, so that nearly all children who have not had it, take it, if exposed. No age is exempt, but the greatest liability is between the first and the third year. In no other contagious disease is infancy so frequently affected as in pertussis (see page 573). Many children are attacked even within the first few months and not infrequently within the first week. Indeed, even congenital pertussis has been reported when the mother has had whooping-cough before delivery; the new-born infant having a characteristic cough on the first day. The author's youngest patient was ten days old. The mother had pertussis. It may be well supposed that the predisposition is as great during the first year as it is later. If fewer children are affected with the disease during this period than in the second or third year, it may be fully explained by the slighter danger of infection—slighter because young infants are more carefully isolated than are older children and because many first-born are not exposed in infancy at all.

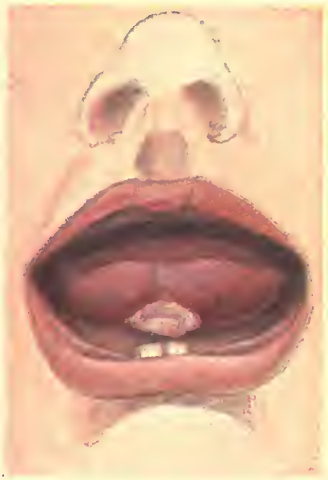


Fig. 173.—Ulcer of the frenula of the tongue in pertussis.

One attack of the disease confers a high degree of immunity, so that a second is extremely rare. When second infection does occur, it is usually in the adult, who had the disease in childhood and is later exposed to reinfection by close contact with children suffering with whooping-cough. The markedly greater predisposition of females is quite noticeable at all ages—a fact not yet accounted for.

The constitutional quality of the child exercises an influence upon the course of the disease. The attack is more severe and lasts longer in nervous, neuropathic and spasmophilic individuals.

The cold season causes an increased sensitivity of the respiratory tract and thus favors the spread of pertussis.

The disease-picture is ordinarily quite monotonous and varies chiefly in the intensity of the individual case. For purposes of description a case of medium severity is outlined.

The incubation period is of variable duration. On the average it covers about one week, but it may be shortened to three or four days or extended to two weeks. It is safe to conclude that a child who has been exposed and does not develop any catarrhal symptoms within fourteen days has not been infected.

Since its very first recognition, three stages of pertussis have been noted; a catarrhal stage, a convulsive stage and the stage of decline.

The *catarrhal stage* begins with slight coryza and cough, possibly a little hoarseness, and a reddening of the conjunctiva. In those predisposed to laryngeal symptoms the disease may be ushered in by an attack of pseudo-croup. These manifestations last from one to two weeks, rarely for a shorter or a longer period. During the first few days slight fever may develop in some few cases, even before distinct catarrhal signs are present. At the outset there is nothing to distinguish this stage from ordinary acute catarrh. A suspicion of pertussis is aroused only upon information of exposure. Occasionally, a case never gets beyond the catarrhal stage and recovers without a diagnosis. Usually, however, the cough becomes more and more severe without the development of any bronchitic signs. Most noticeable are the facts that ordinary remedies fail to relieve the cough; that the coughing occurs at night as well as by day and gradually establishes a certain periodicity.

The catarrhal phase gradually passes into the convulsive stage. The cough is rather less frequent but it occurs at regular intervals, even during the night. The attacks assume a violent, compellant, intense character and proceed, with varying rapidity, toward the typical quality so readily recognized. The spasms of coughing are often ushered in by restlessness and discomfort. The child senses a tickling in the throat, a degree of pressure in the thorax; it becomes frightened and runs to the mother, or clutches any convenient object. In some cases initial vomiting occurs. After a deep inspiration a succession of intense coughs follow each other so rapidly that inspiration is arrested. The face reddens, the conjunctiva becomes injected and the tongue protrudes. The coughing fit is followed suddenly by a forced, loud inspiration termed the "whoop," produced by the violent indrawing of air through the glottis, which is still in a state of spasmodic contraction. But the attack does not end with this temporary relief, as may be seen from the persisting restlessness of the child. Repeated spasms of coughing terminated by the characteristic whoops follow. These attacks recur several times, while the lips and tongue become more and more cyanotic and in severe instances quite blue, until the child seems threatened with suffocation. The series of attacks usually end when the patient expectorates, with difficulty, the accumulated mass of tenacious glairy mucus. In very young children this material frequently remains in the pharynx. Should the attack be very severe it is followed by the vomiting of all food recently taken. Robust children will recover immediately from serious attacks and will continue their interrupted play with apparent unconcern. The younger and weaker are exhausted; they perspire freely, and it will be some time before they recover from the immediate effects.

In number and intensity the attacks are extraordinarily variable. They increase progressively during the convulsive stage, which usually lasts from three to six weeks. Many children have only five to ten attacks in the twenty-four hours; in others the number will run to thirty, fifty or even more. The individual attacks vary similarly; the series of staccato coughs and the reprisal may be repeated twice to five times, as a minimal, and from ten to thirty times as a maximal range. In rare cases a spasmodic sneezing takes the place of the coughing. The lungs, in uncomplicated cases, present normal auscultation sounds or scattered coarse râles which disappear for a time after each attack. Frequently, however, a distension of the lungs develops during the convulsive stage. This becomes an especially marked feature in infants. In severe cases, the heart shows dilatation of the right ventricle. The pulse-rate is increased. Edema of the face is common at the height of the disease. Fever always indicates the presence of complications.

When whooping-cough has once reached its maximal intensity, it continues for several days or weeks at its height and then enters the stage of defervescence and declines rapidly. The attacks at first become less numerous but remain as intense as ever. Soon the intensity likewise decreases; the vomiting stops; the cough occurs but rarely and loses its typical character. A simple catarrhal cough may persist for a long time.

The duration of the entire illness varies in the average case from four to ten weeks, provided there are no complications. Even with a favorable termination, there is considerable loss of weight. Convalescence is commonly rapid in the absence of pulmonary complications which may lay a foundation for tuberculous infection. If bronchitis sets in during the stage of decline, it usually causes a recrudescence of the disease or the reappearance of frequent and intense attacks, which again disappear speedily with the relief of the bronchial affection. If a child develops any ordinary bronchial catarrh, weeks or months after a complete recovery from whooping-cough, the characteristic quality of the cough of pertussis again appears.

Variations in Course

Sometimes the course of pertussis is completed in two or three weeks. But few attacks occur and often these are not typical. At other times an irritative cough persists for several weeks without the development of distinct spasmodic attacks. The diagnosis of this mild form is possible only when definite cases of pertussis are to be found in the immediate neighborhood. Such marked cases are quite common in late childhood and among adults and naturally favor the spread of the disease.

On the contrary, extraordinarily severe cases are comparatively few. Apparently they depend either upon marked predisposition or upon exceedingly virulent infection. These cases in the beginning show a distinct fever. The temperature may rise as high as 39° C. (102° F.). The general well-being is seriously impaired by restlessness and loss of sleep. The pulse is rapid. Dyspnœa and frequent exhausting attacks of coughing and vomit-

ing soon develop. So severe a type of the disease, without any marked complications, may lead very speedily to a fatal termination.

Pertussis in young infants often presents peculiar characters. The attacks of coughing occur with such severity as to cause cyanosis and at times persisting apnœa with ensuing loss of consciousness, eclamptic attacks and general atony. The crowing inspiration at the close of the paroxysms disappears and is replaced by a gurgling and choking with accumulated mucus. Vomiting in these cases is comparatively uncommon. In the absence of the usual symptoms a diagnosis often fails, particularly in the early months of life.

Special Symptoms and Complications

In ordinary cases the general well-being is not greatly disturbed. At times an irritable and peevish disposition is noted. Fever, after the first few days, almost always announces complications most frequently found in the respiratory tract. In a few cases, however, simple pertussis is attended by slight persistent fever. Complications nearly always set in during the convulsive stage. Many of these are the direct consequences of the intense paroxysms of coughing and the ensuing venous congestion.

Dilatation of the right ventricle of the heart is often the result of the severe coughing spasms continued for a long period. This may be demonstrated by percussion, but it is often veiled by the distension of the lungs. In these severe cases it is impossible to determine to what extent a persistently increased pulse-rate is due to over-exercise of the heart, or in what measure it should be attributed to toxic influence. Actual myocardial disease, endocarditis and pericarditis are rare complications of whooping-cough. Persistent congestion of the lungs causes an accentuated pulmonic second sound. Sudden cardiac death is rare.

The blood often shows an increased number of leucocytes, ranging up to 20,000 or more. This is usually due to a multiplication of the neutrophiles and still more definitely of the lymphocytes.

Through a congestion of the cervical veins severe paroxysms of whooping-cough cause a reddening of the conjunctiva and an edema of the eyelids, frequently spreading to the entire face and incompletely disappearing in the intervals between the attacks. On this account, children suffering with pertussis often show a peculiar puffiness of the face, especially marked about the eyes, and coincident with a swelling of the thyroid.

During the paroxysms the frenum of the protruded tongue is injured by pressure against the lower central incisors, causing the formation of a transverse white ulcer (Fig. 173). While this injury to the tongue may appear in the event of coughs of other origin, or even independently of this symptom altogether, it is especially common in pertussis and most often occurs in children, who have only the two lower incisors, since the irritation of the frenum is then most severe.

The abdominal strain attendant upon the spasmodic coughing, not infrequently develops in those predisposed, either an inguinal hernia or a prolapse of the rectum. Involuntary defecation and micturition often occur with severe paroxysms.

An intense venous congestion often leads to a rupture of the delicate capillary walls, injured possibly by the disease toxins, and cutaneous hemorrhages result. Epistaxis is a very common occurrence, but is rarely alarming. At times bloody expectoration from the pharynx or the bronchi is observed. Semilunar extravasations in the bulbar conjunctiva around the cornea are comparatively common. Hemorrhages in the eyelids are rare.

The most frequent and the most dangerous complications are those of the respiratory tract. It is doubtful whether even coarse, bronchial râles can be considered as incident to pure pertussis, or whether, as one would prefer to believe, they are to be regarded as evidences of complication. The fact that many healthy children throughout the entire course of the disease never present catarrhal breathing sounds and that these alterations are especially observed in the cold season and in weak, rickitic children, supports this view. As long as only occasional coarse râles are heard without change in the respiratory rhythm and without fever, no harm results. Nevertheless, the presence of these râles should prepare the clinician for more serious developments, since they are often but the precursors of a severe bronchitis or of a fatal broncho-pneumonia. If a severe bronchitis develops, the glairy sputum takes on a yellowish-green color and an ordinary catarrhal cough more or less frequently occurs in the intervals between the paroxysms of whooping-cough. In children of three or four years or over, the bronchitis eventually disappears with no more serious results than the protraction of the pertussis. Associated with coryza and bronchitis, a catarrhal or purulent otitis media, of usually benign character, may appear.

In young children, and particularly in those who are rickitic and weakly, bronchitis has a great tendency to lead to broncho-pneumonia. At the onset this transition is often unrecognized, on account of the presence of only small scattered foci of invasion. It is sooner suggested by dyspnœa and by a more intense and remittent fever than by the demonstration of dulness. The paroxysms of coughing frequently lose their typical character when a broncho-pneumonia or an accidentally intercurrent lobar pneumonia develops. They grow shorter and the whoop is weakened or is entirely lost, while the cyanosis remains. After the pneumonia is over the paroxysms reappear in typical form. Capillary bronchitis, as a complication of pertussis, is a less common event.

A progressive and extremely dangerous broncho-pneumonia is particularly frequent when measles occurs coincidently with pertussis. Extensive changes in the lung favor the occurrence of convulsions.

Broncho-pneumonia, in these cases, follows a very protracted course and is extremely obstinate as long as intense paroxysms of coughing occur. Etiologically, the condition is generally due either to the strepto- or pneumococcus. The latter gives the more benign form.

Pertussis often causes pulmonary emphysema, revealed by the prominence of the thorax and the low position of the lung border anteriorly on the right side. The increased expiratory pressure leads to a diffuse bronchiectasis, giving no clinical symptoms but often determined at autopsy.

Permanent bronchiectasis following pertussis is rare, nor does pulmonary emphysema very often remain, and generally only in those cases which show an asthmatic tendency. Intense coughing may cause rupture of the alveoli which exceptionally leads to an interstitial, a mediastinal, or even a subcutaneous emphysema and, in turn, to severe dyspnœa and death. This protracted form of broncho-pneumonia resembles the type of the disease associated with tuberculosis. The differentiation is all the more difficult, because a latent tuberculosis frequently becomes active during an attack of pertussis and generally takes the form of tuberculosis of the bronchial lymph nodes or of broncho-pneumonia arising from the hilus. The possibility of tuberculous infection must always be considered when a young child who has, or has recently had whooping-cough, shows emaciation, anemia and an irregular fever. A positive cutaneous reaction to tuberculin must be accepted, even in the absence of adequate lung findings, in such a case. In older children simple broncho-pneumonia is uncommon and, therefore, positive pulmonary findings become more suggestive of tuberculosis. In younger children a negative tuberculin reaction is often an aid to the exclusion of tuberculosis.

Next to the respiratory tract, the nervous system is apt to be involved in pertussis. Its previous condition has a determining influence upon the course and gravity of the disease. In the neurotic or neuropathic the paroxysms of coughing are more frequent and more severe than they are apt to be in the normal individual. Excitement or pain to which they may be subjected readily brings on an attack.

When a number of children suffering with pertussis are together in a single room and one of them goes into a paroxysm, all the rest may join in the chorus. Older children are often able to suppress an attack by sheer force of will and may even be stimulated to do so by threats. It is recorded of the wife of a certain general that she cured her children of whooping-cough with the rod. An anxious or restless demeanor upon the part of other members of the family has a bad influence upon the patient, while his recovery is favored by quiet and sensible behavior. It would be a dangerous perversion of facts, of course, to argue that the nervous constitution of the patient is any more than an aggravating influence, or to regard pertussis as a nervous disease or as merely the reaction of the neuropathic to various catarrhal infections of the respiratory tract.

In very young children the spasm of the glottis which causes the crowing inspiration at the close of the paroxysm of coughing often becomes very severe and may lead to a long continued closure of the aperture. This may induce apnœa, a profound degree of cyanosis, and the loss of consciousness. Quite frequently slight twitching of the facial muscles is added to the spasm of the glottis. Even a general eclamptic condition lasting for several minutes, with prolonged loss of consciousness, has been observed. Not infrequently general convulsions follow a severe spasm of the glottis in the young infant. These convulsions, indeed, may appear independently of and in the intervals between the attacks. The very great majority of children in whom the spasm of the glottis is accompanied by eclampsia of

very intense degree, and even threatening life, are spasmophilic. In fact the prognosis of pertussis in the spasmophilic is always grave. In infancy, sudden death, in the midst of these severe convulsions, is not uncommon. Some writers assert that eclamptic convulsions in pertussis are always indicative of this constitutional disturbance, but with this view the author is not agreed. The possibility of cerebral irritation as a result of venous congestion and often as a result of deeper organic changes is to be considered. Neurath and others have found that edema and true infiltration of the pia mater occur in pertussis. Occasionally, severe cerebral disturbances occur which must arise from an organic lesion. Sudden hemiplegia, appearing during a severe paroxysm is only occasionally due to cerebral hemorrhage. Central blindness, deafness, flaccid paralyses, imbecility, confusion, paresthesias, conditions which are fortunately of rare occurrence and usually transitory, are supposably traceable to the same cause.

Aside from the edema and the subcutaneous hemorrhages already noted, the skin is rarely affected in pertussis. Erythemata are very uncommon.

The digestive system is but little disturbed in the majority of cases. Very often, however, vomiting occurs at the close of a paroxysm. In nervous individuals this may be very obstinate and may lead to actual inanition, especially when it is combined with anorexia. Diarrhœa is uncommon. In children of one or two years of age it is to be dreaded because it may impair the nutrition seriously.

Enlargement of the liver occasionally occurs in pertussis as a result of congestion. Enlargement of the spleen is rare. Albuminuria is demonstrated in some severe cases. True nephritis is a great exception.

The diagnosis of pertussis is very easily made in advanced cases, if the physician is present during a paroxysm. It is especially easy of recognition when several children of a family are simultaneously affected. If necessary, a paroxysm may be brought on if some interval of time has elapsed since the last one. This is most readily done by introducing a tongue depressor well back in the mouth and pressing the tongue down forcibly. If this does not suffice, compression of the larynx or trachea from without, will often bring on an attack.

If the physician cannot observe a paroxysm, the diagnosis may often be made from the history of attacks of coughing occurring at regular intervals and even during the night, ending in a "whoop," and followed by the expectoration or hawking up of a tenacious mucus, causing vomiting. In coughs of other origin, children of less than eight or ten years are not apt to bring up sputum, so that any young child who does may be suspected of whooping-cough. The regular appearance of the paroxysms, and their continuance through the night, together with the lack of objective lung findings, speak definitely for pertussis. Frequently signs of congestion about the face, the presence of an ulcer on the frenum of the tongue and indications of hemorrhage in the eyes, etc., assist the diagnosis. A decision becomes very difficult or even impossible when there is only an irritative cough without distinct paroxysms, and particularly in the catarrhal stage before their paroxysmal quality has developed. Without a knowledge of

the disease in the immediate neighborhood, or without the discovery of a known source of infection, diagnosis must often be reserved to a later period.

In the differential diagnosis of pertussis, certain diseases of the upper air passages, tuberculosis of the bronchial lymph nodes, and hysteria must be considered. Adenoid vegetations and recent pharyngeal catarrh often causes a severe cough, which may occur during the night when secretion flowing into the throat causes some irritation. Such coughing spells are, however, irregular and frequent, but they do not increase in severity, nor assume a paroxysmal form. Certain forms of la grippe produce a severe and persistent cough, which may excite vomiting. The occurrence of marked and frequent paroxysms of coughing at the outset of the disease, with the development of fever and râles, argue against pertussis. The cough which results from enlargement of the bronchial lymph nodes is very similar to that of whooping-cough. Frequently it has a paroxysmal character. As a result of pressure of the enlarged glands upon the vagus it may become sufficiently severe to cause the expectoration of mucus and vomiting. The crowing inspiration, however, is lacking. Such a cough may last for many months without the noticeable increase or decrease of severity seen in pertussis. Further, there are usually other signs which point to the basic disease. Among these are fever of irregular course, emaciation, and dulness in the intra-scapular space, while characteristic shadows are observed in the Roentgenogram. Imitative neuroses, due to hysteria, occur only in older children and even with them are exceptional. These cases are differentiated by the absence of paroxysms during sleep.

The prognosis is determined, primarily, by age. In children of three or four years, or more, it is usually favorable, but in younger children the possibility of broncho-pneumonia is always to be considered. The younger the patient the greater the danger. Nevertheless, young infants often weather the attack surprisingly well. The greatest mortality is found between six months and two years of age, in a word, during that period when spasmophilia and rickets are at their height. These two disorders increase the mortality of the disease materially; the former on account of the intensity of the spasm of the glottis it induces and the liability to eclampsia it carries with it; the latter on account of its characteristic tendency to severe bronchitis and broncho-pneumonia.

There is danger of a fatal termination in debilitated or tuberculous infants or in those suffering with disturbances of nutrition. The prognosis also depends in large measure upon the environment and the kind of care the patient receives. Frequently bronchitis or a tendency to it persists for months. It is a notable fact that in many cases an attack of pertussis seems to determine an improvement of the general health.

Prophylaxis.—The heaviest task in the prophylaxis of pertussis is in the protection of children from infection until after their third or fourth year. This is not impossible with the carefully tended children of the well-to-do, since the disease is directly transmitted in practically all cases. Children who have pertussis or are under suspicion of it should be isolated both within and without the family. This isolation should be scrupulously

applied in day nurseries and kindergartens. During an epidemic every child suffering with a coryza or cough should be considered a suspect. If a case of whooping-cough has appeared in a family, the separation of the patient from others in the home who have not yet had the disease is usually in vain. Should the isolation be attempted, the possibility of the spread of the infection remains for the lapse of fourteen days from the date of last exposure. After this period, the children who are well may be permitted to associate with other children if they show no sign of coryza or cough. Children suffering with pertussis should not be permitted to appear on the public streets or playgrounds. Ordinarily they should not be allowed to associate with others until the cough has entirely disappeared. School children may be readmitted to school, however, after a period of three months, even though some cough remains.

The sputum and vomitus should be carefully removed and disinfected with a 3 per cent. solution of lysol. Since the organisms die very rapidly outside the body, disinfection of the patient's room is unnecessary.

According to a large number of workers, pertussis vaccine is definitely effective, as a prophylactic measure, if the following requirements are met. First, the use of a freshly prepared vaccine and second, the administration of of sufficiently large doses. The following four doses are recommended at forty-eight hour intervals; one-half billion, one billion, two and one-half billions and five billions.

Treatment.—*As long as there was no specific* therapy, hygienic measures were the mainstay of treatment. The most important of these is fresh air. Patients without fever should be kept in the open as much as possible. Older children may be permitted to play about in the garden, in the woods, or in other places that are free from dust. Smaller children should be wheeled or carried about. In the open air the paroxysms are less frequent and less violent. During rough and cold weather, however, the patient should be kept in the house, but even there, all possible provision for fresh air should be made. In artificially heated rooms the atmosphere must be kept at the proper degree of humidity. The patient with fever must be kept in bed, although in pleasant weather he may be placed on a veranda or in the garden.

The value of a change of environment, so highly recommended, has been greatly over-estimated. It is of value only when it means transfer to a more genial climate—that is to a warmer one in winter or to a dust-free, woody place during the summer.

In the selection of a dietary the irritability of the mucous membranes must be taken into account. Highly spiced, coarse foods should be avoided, since they may provoke paroxysms of coughing. When the attacks are followed by vomiting it is well to give a small amount of food immediately after a paroxysm. Semi-liquid food is to be preferred.

If the paroxysms are very numerous a moist pack, tepid at first, and later at room temperature, applied to the chest for two or three hours, has a quieting affect even in cases in which there are no bronchial râles.

The number of the medicinal agents recommended in pertussis is legion,

the most definite indication that no certain remedy has been found. Cases are so extremely variable in severity that the influence of drug therapy is hard to estimate. Nevertheless, there are certain remedies which have an action more definite than that which rests upon mere suggestion. In judging the results obtained by medication, one must not forget that while it is very difficult to influence the disease at the onset of the convulsive stage, it is very easy to obtain results at the close of this period.

In recent cases quinine is often given. To infants 0.05-0.1 gm. (¾-1½ grs.), of the hydrochlorate may be given three times a day; to older children 0.15-0.5 gm. (2-7½ grs.), twice a day. Young children take the chocolate tablets containing the quinine tannate very well. In later childhood, the quinine may be given in gelatin capsules. Euquinine may be substituted if the child refuses other salts of quinine on account of their taste. Of this preparation infants may be given 0.1 gm. (2 grs.), two or three times a day, and older children, 0.15-0.5 gm. (3-8 grs.), three times daily. Antipyrin may be tried in doses of 0.03 gm. (½ gr.), for each month of age; or 0.3 gm. (5 grs.), for each year of life. These doses of antipyrin appear to be rather large and if administered are worthy of very careful supervision.

The value of pertussis vaccine in the treatment of the disease has been fairly well established. Its absolute harmlessness and the disagreeable chronicity of the illness justify its use especially if it can be administrated before the paroxysmal stage has begun. For this purpose a fresh vaccine is absolutely essential and the dose should be at least as large as that advised under prophylaxis and no objection can be raised to giving a fifth injection of ten billion organisms.

If no results are obtained from this treatment after eight or ten days use and if severe paroxysms continue, it may be well to employ narcotics which never wholly fail of benefit. For the purpose codein and the bromides are most highly recommended. An infant in the latter half of the first year may receive 0.001 gm. (1/50 gr.) of codein; a child of two years, 0.002-0.003 gm. (1/30-1/20 gr.); a child of five, 0.005-0.006 gm. (1/12-1/10 gr.), each three times daily. These doses may be doubled if they do not have the desired effect. Of the bromide preparations, the sodium salt is preferred and may be given, in aqueous solution, to infants in doses of 0.3-0.5-1.0 gm. (5-8-15 grs.), each day, and to older children in doses of 3 gms. (45 grs.). Bromoform, in large doses, given three times a day, is also highly recommended. This heavy oily liquid must be kept in dark bottles. The dose beginning with two drops for each year of age, may be increased a drop, at a time in similar progression. Thus a child of two and a half years would receive, initially, five drops three times a day, increased, later, to five drops four times a day and finally to a maximum of seven drops four times daily. Infants may be given two to four drops, three or four times daily, while older children may be given as high as forty drops in the course of a day. The required amount of the remedy is carefully dropped into a teaspoonful of syrup or sugar water. It is usually efficacious, but it does not act rapidly. On account of the great liking which children sometimes acquire for bromoform, its use should be guarded. They have been known

to drink an entire bottleful at once, with resulting fatal poisoning. The remedy should be placed in the hands of careful parents only and must be kept away from the children. Often it has been useful when other medicinal agents have failed. The child's appetite frequently shows marked improvement in ten to fourteen days.

In very severe and threatening paroxysms a dose of morphin may be given. With proper precautions an infant may be given one milligram (1/60 gr.), two or three times a day, and children of two to four years, two or three milligrams (1/30-1/20 gr.), twice a day. Large doses of morphin given subcutaneously have been recommended recently, but the writer has not witnessed any favorable result from their use.

In severe spasm of the glottis or in eclamptic attacks large doses of the bromides often save the patient. From 0.5-1.0 gm. (8-15 grs.), a day, may be used in infancy.

Of innumerable other remedies, belladonna, which sometimes has a very distinct influence may be mentioned. Probably it serves its purpose by reducing the excess of secretion. Doses of from 1-3 milligrams (1/30-1/20 gr.) of the extract of belladonna, or doses of 0.05-0.2 milligram (1/1000-1/300 gr.) of atropin sulphate may be used in infancy; from 30-50 milligrams (1/2-1 gr.) of the extract or from 0.3-1 milligram (1/200-1/60 gr.) of the atropin with older children. The smaller doses should be given at first, and the appearance of dilated pupils or of an erythema of the skin should be accepted as indications for the immediate reduction of the amount. It is well to combine this remedy with the bromides. If the secretion is very tenacious the croup kettle or the evaporation of the ethereal oils often gives relief. In very severe and alarming attacks a combination of bromides with codein and atropin is frequently effective.

During attacks it is well to lift small or weak children and to support the head.

It is extremely important that complicating conditions of spasmophilia should receive antispasmophilic treatment, by way of a scant diet, with but little milk, the use of the calcium salts and if necessary, phosphorus and cod-liver oil. Temporary underfeeding seems to be beneficial in strong non-spasmophilic children with severe paroxysms.

When eclamptic attacks are alarming and persist in spite of treatment, lumbar puncture sometimes gives relief. Transitory narcosis may also be useful. In extreme cases with persisting spasm of the glottis intubation may be tried.

Bronchitis and broncho-pneumonia are treated in the usual manner (see pages 370-377). With excessive bronchial secretion, the question often arises whether expectorants or narcotics should be used. Cardiac insufficiency is combated with caffein and camphor (see page 415).

Guaiacol preparations are useful in pulmonary involvement during the stage of decline. From 0.05-0.15 gm. (1-3 grs.), of guaiacol carbonate may be given three times a day.

If convalescence is delayed and fever persists for any length of time, arousing a suspicion of tuberculosis, the child should be sent to the country,

to the seashore or to the mountains for a time. Institutions for the treatment of pertussis, located in the suburbs of large cities, fill an actual need and may save many a child in indigent circumstances, who might otherwise succumb to chronic bronchitis or tuberculosis.

MUMPS; EPIDEMIC PAROTITIS

Mumps is a contagious disease, the chief symptom of which is, commonly, an acute swelling of the parotid gland. The causative organism of this disease, a malady recognized since the time of Hippocrates, has not been discovered. Primary and idiopathic parotitis occurs sporadically and in epidemics of variable spread. The latter may extend over entire localities and invade, particularly, schools, institutions, barracks, etc. These epidemics usually last for several months. Sometimes they are remarkable for their special intensity, sometimes for the prevalence of unilateral expressions and, again, for the appearance of complicating diarrhœas.

The contagion is commonly conveyed directly from the sick to the well. According to the writer's observations, the contagious period antedates the appearance of the typical swelling of the gland by one or two days; it usually decreases rapidly in intensity during convalescence, although it may persist for weeks after healing. The spread of the disease is favored by the occurrence of numerous light ambulant cases. It is possible of indirect spread through the medium of healthy individuals and even by means of infected utensils, etc. It is commonly supposed that the germs enter the mouth and pass from thence through Stenson's duct. This conception, however, seems doubtful when the atypical forms and the vagrant localizations of the disease are taken into account.

The predisposition to the disease is general, although it is not universal. Persons between five and fifteen years show the greatest liability. The infection of children less than two years of age is uncommon and cases in infancy are exceptional. A few instances of the disease have been reported among infants and even congenital infections, derived from the affected mother, have been described. One attack gives positive immunity. Second attacks, however, have been not infrequently reported in adults.

The pathologic basis of the disease, as determined in the few examinations made, consists in edema and congestion of the interstitial tissue of the parotid gland and the surrounding structures. A round cell infiltration may be added. The parenchyma of the gland is not directly involved.

Mumps has a very long incubation period, averaging two and a half to three weeks, so that an epidemic invading a family often lasts a long time.

Clinical Picture.—After the incubation period, without symptoms, light prodromes are often noticed, continuing for twelve to thirty-six hours. The child is languid, irritable, without appetite, and suffers with chills and slight fever. Sweats, epistaxis, and pain and roaring in the ears may develop. The swelling of the parotid gland of one side immediately follows, or appears as an initial symptom. A slight thickening is first noticed in the part of the parotid that lies directly over the ramus of the jaw. This swelling is often more readily discovered in a view of the full face than it is by

palpation. It is especially evident upon comparison of the two sides of the face. The tumor has a doughy quality, about which no sharp borderline can be recognized. Its characteristic location, immediately beneath the lobe of the ear and in front of the tragus, indicates that the parotid gland is involved. In the majority of cases there is no sensitiveness to touch and when present, it is slight. It is actually painful only in exceptional cases. The skin over the swelling shows no change. If the tumor is very large, the skin may become glossy, but is scarcely even reddened or warm. The increase of the swelling is observed for two or three days. It fills the space

Fig. 174.—Epidemic parotitis. The swelling fills the hollow between the ramus of the jaw and the sternomastoid and forces the lobe of the ear outward.

between the mastoid process and the ramus of the jaw and forces the lobe of the ear outward quite characteristically (Fig. 174). The peculiar plumpness of the cheek has made the disease familiar to the laity and has caused various popular names to be applied to it, which testify to the general harmlessness of the disease.

The tumor may become very marked and the edema may extend far beyond the parotid gland up to the orbit and down to the horizontal portion of the lower jaw. The patient often complains of the tenseness of the cheek. He experiences difficulty in opening the mouth and pain on mastication. The pressure upon the external auditory canal may cause lancinating pains in the ear and deafness. The swelling remains at its height

for some two days, after which it goes down rapidly. At or before this climax, the parotid gland of the other side becomes affected with the accompaniment of another rise of temperature.

Occasionally, the other salivary glands are involved simultaneously with the parotid. They are affected, at times, instead of the parotid and may swell to large size. The submaxillary gland is much more commonly infected than the sublingual (Fig. 175). If the submaxillary and sublingual glands alone are involved, or if the swelling of the parotid has been slight and has escaped recognition, the diagnosis is almost impossible unless, indeed, the case occurs during an epidemic.

The mucous membrane of the throat and mouth are often slightly reddened. A more marked degree of inflammation in the form of a catarrhal

Fig. 175.—Simultaneous illness of brother and sister with mumps of the submaxillary gland.

or lacunar tonsillitis is rare. The saliva flowing from the parotid duct shows no change in quality but is sometimes increased in quantity.

The accompanying fever shows no characteristic features. With, or even before, the initial swelling of the glands the temperature usually rises for two or three days. It commonly varies between 38° and 39° C. (100°-102° F.). With the subsidence of the swelling, or even earlier, it falls rapidly, but no characteristic curve can be determined. Fever above 39°-40° C. (102°-104° F.) is rare in children, but not necessarily so in adults. The fever, sometimes, is of so brief duration and so insignificant as to escape notice.

The course of the disease covers five to seven days when one gland is involved, and ten to twelve days when both sides are diseased. The attack almost always terminates in complete recovery. Very exceptionally, in cachectic cases, a secondary infection causes a further enlargement of the gland. Relapses after a period of ten to twenty days are very rare.

The disease, with extremely few exceptions, takes the described course without serious symptoms and without leaving any permanent injury. As

a rule, the malady is a light one in children under ten years of age, so that it is hard to keep them indoors and but few cases are seen by the physician.

There are rare cases, however, of atypical localization and very occasionally with severe complications. The localization of the disease in the testes was recognized as a peculiar feature by Hippocrates. This accident is confined almost exclusively to youths and adults, in whom it occurs in perhaps one-third of the cases. Usually about a week after the onset of the parotitis and with the disappearance of the swelling in the cheeks, a high fever, attended at times by delirium and with serious general disturbance, sets in. Coincidentally a painful, inflammatory swelling of one or both testes is observed. After this interstitial inflammation has subsided, an atrophy of the testes may develop, which, if both sides are affected, causes sterility. In children such an orchitis is exceedingly rare. It occurs only after the thirteenth year and very seldom in advance of the development of the sexual function. Sometimes it appears before the parotitis and may even be the only local expression of the disease. An analogous inflammation of the ovaries and the mammary gland is said to occur in females. The occasional syndrome of vomiting, abdominal pains and tenderness upon pressure in the region of the pancreas has been taken as evidence of pancreatitis.

In common with other infectious diseases, mumps is now and then followed by nephritis. This is usually of a hemorrhagic and transitory type. As exceptional symptoms may be noted a swelling of the thyroid, of the lachrymal gland or of individual joints, and variant eyrthemata, sometimes of a rubeolar and again of an urticarial character.

The nervous system is materially affected in but very few cases. Certain French authors state that the development of a slight serous meningitis is not rare. Its manifestations, of somewhat indefinite order, are fever, headache and a slow pulse. The cerebrospinal fluid is said to show the objective evidence of these indistinct symptoms in an increase of its protein content and its lymphocytes. Within a few days these conditions disappear. Very rarely they go on to the development of a well-marked form of meningitis, still of a serous type, evidenced by rigidity of the neck, Kernig's sign, delirium and convulsions, and even leading on to death. The occasional appearance of paralyses of the ocular muscles or of monoplegia show that meningoencephalitic changes may occur, while polyneuritic paralyses suggest that the peripheral nervous mechanism is not always spared. A sudden acute labyrinthitis, causing deafness upon the affected side or even complete deafness if both ears are affected, has been known, but fortunately is extremely rare. A harmless otitis media is more common. A transitory facial paralysis may be due to compression of the nerve branches by the swelling of the parotid gland. Acute mental confusion and rapidly passing psychoses are occasionally described.

The diagnosis of epidemic parotitis is ordinarily easy and is often made by the laity merely from the typical swelling of the cheeks. Its epidemic occurrence and the sequence of its development first on one side of the face and then the other side, is of assistance in doubtful cases. The disease is

most likely to be confused with lymphadenitis or with alveolar periostitis. Mumps, however, gives a doughy non-circumscribed swelling in front of the tragus, where enlargement of the lymph nodes is uncommon. The well-rounded extension downward of the primary swelling, but without redness or pain, is typical of mumps. In lymphadenitis the swollen node can be easily palpated and its phlegmonous painful character is quite clear. Metastatic parotitis appears among the complications of severe infectious disease, as in diphtheria, typhoid fever, etc., and is seldom an occasion for error. Moreover, it always tends to suppurate. Isolated instances of mumps of the submaxillary glands or of the testes are discovered only in the midst of epidemics. Finally, it may be noted that some persons react to iodin medication with a fluctuating swelling of the parotid.

Prognosis.—It is apparent from the related history that the prognosis is not always as favorable as it is generally believed to be, even though these serious complications are rare. The prognosis is always better in children than in adults, so that it may appear to be a mistake to protect healthy children from the disease. Still, considering the possibility of permanent deafness it is doubtless the part of wisdom to guard the child carefully from infection.

The treatment may be expectant and confined to rest in bed and to liquid diet during the febrile stage. The swelling may be covered with warm oil or with bland ointment and be protected with dry cotton. For the care of the mouth and for accompanying angina, irrigation and gargling with solutions of borax are recommended. It is well to consider the secretions of the mouth as contagious and to render them harmless by disinfection; as it is, also, to prevent the spread of the disease by avoiding kissing the patient, etc. In hospitals, institutions, etc., it may be necessary to fumigate the place, after the infected inhabitants have been removed, in order to bring the epidemic to an end.

TYPHOID FEVER

Typhoid fever is a specific acute infectious disease, in which the intestinal tract and its lymphoid system are especially affected, the clinical picture of which is largely dominated by the general symptoms.

The causative organism is the bacillus typhosus, an organism closely related to the colon group. It may be demonstrated, almost always, in the blood and in the invaded organs of the infected subject from the beginning of the febrile period. It is found in thirty per cent. of the fecal specimens and in fifty per cent. of the urinary specimens of patients up to a late stage of convalescence. Healthy typhoid carriers are found among children, although they are much less common than among adults. This relative scarcity is probably due to the fact that disease of the gall-bladder, which favors the continued growth of the micro-organism for a long time after the subsidence of the disease, is infrequent in the young.

Direct transmission of the disease is more common than was formerly supposed. The lack of cleanliness in small children favors the infection of attendants or of persons living in the same house. Typhoid fever in infants, usually a matter of late diagnosis, is especially dangerous in this respect, as

the writer's experience in several cases has shown. Almost without exception the infection is brought about by bacilli which escape in the feces and urine and which, failing of destruction, contaminate drinking water, milk, or other food materials. In this way large epidemics arise through the use of infected water supplies, milk, etc. The food probably serves only as means of entry. Cases in which the infant, fed at the breast of a mother with typhoid fever, is infected are doubtless traceable to a lack of cleanliness upon the part of the mother in the care of pacifiers, bathing materials, etc., rather than to the passage of bacteria through the mammary gland. Many cases, indeed, have been observed in which the children of mothers suffering with typhoid have not been infected.

Typhoid fever often occurs in several members of a family who come down with the disease in rapid succession. Thanks to the advances of public hygiene, large epidemics have become infrequent in late years.

Predisposition in the child is practically the same as in the adult after the fifth year. In children of two to five years it is decidedly less, while infants are but rarely attacked and cases within the first six months are exceptional. No doubt cases of light and atypical form may occur during the first year and escape diagnosis. This very fact may give the opportunity for some epidemics. A few definite cases of congenital typhoid fever, with the disease present in the mother, in which the typhoid bacillus had passed through the placenta have been reported. Typhoid in the mother during pregnancy usually causes the abortion and death of the fetus. The child proves viable in but very few instances.

Predisposition to typhoid seems to be quite general. The question is determined rather by the virulence of the infection than by the individual resistance. In localities where typhoid is endemic new arrivals usually take the disease more readily than old inhabitants, even though the latter have never had the disease. It is doubtful whether one attack of typhoid confers an immunity which lasts for any considerable period. Second attacks are not uncommon.

Pathologic Anatomy.—Pathologic study, as well as clinical signs, go to show that the typhoid processes in the child's intestine are, generally speaking, more superficial and less serious than in the adult. In cases within the first year, both the agminated and the solitary nodes usually show only moderate swelling and small discrete patches which heal quite rapidly. The large, deep-seated patches frequently found in the adult, in whom they tend to extensive ulceration and even to perforation, are observed in childhood only after the seventh or eighth year, and even then are less common than in the adult. Swelling of the mesenteric lymph nodes is occasionally very marked even in the infant, but similar enlargement may occur in numerous other intestinal infections.

The clinical picture of typhoid fever in childhood is generally that of a mild type, frequently termed gastric fever in the past, a quality which is the more pronounced, the younger the child. Shorter periods of fever, lower temperature, abortive forms, less prominent nervous symptoms, and a rarity of intestinal hemorrhage, are typical of the typhoid of childhood.

The typical form of typhoid fever in childhood presents a well-marked clinical picture. The patient suffers at first with indefinite symptoms, the time of onset of which it is difficult to determine. These prodromes consist of lassitude, diminished appetite, vomiting, restless sleep and, in older children, headache. In spite of a high temperature, which is not in accord with the slightly disturbed health, it is often impossible for some days to determine the existence of any organic disease.

In many cases the fever is the most important symptom of the disease. This commonly shows the classical curve; a step-like rise, a stage of continued high fever, and then a period of marked variations. In the child these several stages are often shorter than they are in the adult. The temperature reaches its maximum after four or five days, the extreme peak of the curve is maintained for about one week, and in the ensuing three to five days great variations of fever are observed. The entire febrile period is often completed in two weeks (Fig. 176). The stepladder rise of the fever is often absent in the child. A high temperature, sometimes preceded by a

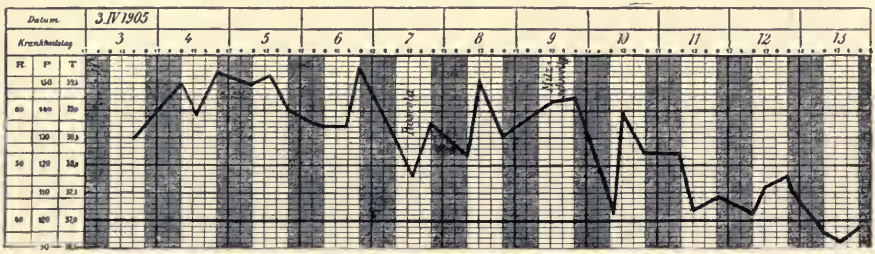


FIG. 176.—Typhoid fever in infant of seven months.

chill, may be seen in older children on the first day of actual illness. This onset, however is only apparently acute. It merely means that in strong, robust children the initial symptoms are overlooked, sometimes for want of careful observation on the part of the parent. In children of less than five years the temperature during the stage of continued fever is usually not above 39.5°- 40° C. (103°-104° F.), but in later childhood it may run higher even in ordinary cases. The range of morning and evening temperature is between 0.5° and 1.2° C. (1° to 2.° F.). The fever is of a remittent character in children more frequently than it is in adults, so that a classical curve cannot always be expected.

The clinical diagnosis usually remains doubtful throughout the entire first week. The probability of typhoid becomes greater with each day of persisting or increasing temperature, for which no organic cause is to be found. Frequent vomiting, a thickly coated tongue, the moderate reddening of the throat and headache are symptoms too common to justify, in themselves, a diagnosis.

The *tongue* is usually thickly coated and dry. In older children it is often clean around the edges and at the tip, where the clean area has a triangular form. The coating and dryness are scarcely ever as marked as in

the adult. Discolored sordes are seen only in severe cases. Frequently the lips become dry and cracked, leading the patient to pick at them.

The *enlarged spleen* is usually palpable at the end of the first or the beginning of the second week. It is of diagnostic value only when the enlargement is marked and develops rapidly. Even then, allowance must be made for the fact that in children, an increase in the size of the spleen occurs in very many infections. If the routine examination is not embarrassed by meteorism, the enlargement of the spleen is hardly to be missed during the second week of the disease.

Rose spots commonly appear at the beginning of the second week and often they are very few in number, so that the surface of the abdomen, etc., must be searched for them. In young children they are not infrequently absent throughout the entire course.

Meteorism, as a rule, is very slight in children, but they complain of abdominal tenderness comparatively often. This pain is increased on pressure over the region of the appendix, but there is no sense of muscular resistance.

The *bowel movements* are usually normal or constipated during the first week. In the young child an early tendency to diarrhœa is not uncommon. After the first week the stools are generally watery and very often have the well-known pea soup consistency. There are seldom more than four to six bowel movements in the course of the day. In a large proportion of cases, possibly in from one-fifth to one-third of them, normal stools are passed throughout the illness. This has been observed even in patients in whom intestinal hemorrhage occurs later.

In many cases a slight *bronchitis*, announced by a cough, develops. It almost always appears in feeble children and in those who fail of proper care. In severe cases of long duration it may lead to broncho-pneumonia. Rickitic children and those who suffer disturbances of nutrition are especially liable to this disease. Broncho-pneumonia is the most common cause of death in the typhoid of childhood.

The *heart* usually shows no distinct changes. Its integrity and great power of resistance during childhood contribute largely to a favorable termination. The slowness of the pulse relatively to the temperature rise, an important indication in the typhoid adult, is distinctly evident in children only after the sixth to the eighth year. The same age factor affects the development of a dicrotic pulse.

In non-complicated typhoid cases, the *blood* shows a distinct leucopenia, a reduction of the neutrophiles, and a disappearance of the eosinophiles. Later the lymphocytes often exceed the leucocytes. During the period of high fever the urine contains traces of albumen and a few casts. Distinct nephritis rarely occurs. Pyelitis is more common. In very nearly every case attended by marked fever, a strong diazo-reaction is present from the end of the first week and persists throughout the stage of high temperature.

The *nervous system* is but slightly affected in young subjects if the fever is not too intense and does not continue for too long a time. Headache, restlessness, apathy and, more rarely, a moderate or noisy delirium, occur in ordinary cases and particularly in those who are not given required treatment.

The entire febrile disease often lasts for only one and a half to three weeks. After the third or fourth week children are commonly convalescent and even from a severe attack recuperate with surprising rapidity.

A rarer and distinctly more *serious type*, however, stands in sharp contrast to this essentially mild form of the disease. It is seen with greatest frequency in later childhood *i. e.*, in children over five years of age and chiefly in the course of prevalent epidemics. It resembles the graver forms of typhoid fever in the adult. Long continued high fever, running up to 40° C. (104° F.), or more; a violent and excited delirium, persistent headache, deafness, and hyperesthesia of the skin over the abdomen, are its principal early features. Oftentimes at the beginning there is a condition of profound apathy, which may deepen into somnolence and coma. Food is refused; the tongue becomes dry and covered with sordes; the pulse is small and very slow. Within a few days bronchitis, attended by cough, makes its appearance and may lead in turn to extensive broncho-pneumonia. Diarrhœa aggravates the increasing exhaustion. Intestinal hemorrhages are not uncommon. Deep-seated ulcers may cause perforation. Evidences of meningism, and particularly Kernig's sign, rigidity of the neck, hyperesthesia of the skin and spasm of the jaw muscles, occur more frequently than in adults.

Peculiarities of Course; Complications

While in the young the course of typhoid fever resembles that in the adult more and more closely as the years advance, the picture in *infancy* is oftentimes not a very characteristic one and frequently remains unrecognized. Even in cases that prove fatal the intestinal lymph nodes are but slightly involved. On this account the familiar findings are often missed at autopsy and the disease is essentially an acute septicemia. The course is commonly short, the fever remains moderate and lacks the features of the typical curve (Fig. 177). The accompanying vomiting, diarrhœa, meteorism and coated tongue give occasion for a diagnosis of gastro-enteritis. Notwithstanding the persistent fever, the absence of colitic stools and the early palpable enlarged spleen should arouse suspicion of the true character of the disease. In the majority of cases a few rose spots also appear. The tendency to apathy, rigidity of the neck, and the tensity of the fontanelle often suggest a meningitic character. Frequently a correct diagnosis is dependent upon the presence of other cases in the neighborhood. Typhoid fever occurring in infants and in children of two or three years is frequently transmitted to the attendants or to other persons in the home, a circumstance explained, in part, by an often late diagnosis and, in part also, by a lack of cleanliness in the habits of these small patients. The writer agrees with the contention of Fischl that the prognosis of typhoid fever in infancy is not bad.

The Digestive System.—Typhoid sometimes begins with a catarrhal or lacunar angina. In a few instances a thin veil-like exudate is seen on the tonsils. In severe cases, when the mouth is not properly cleansed, aphtha-like ulcers may develop and cheesy exudates appear on the gums. Occa-

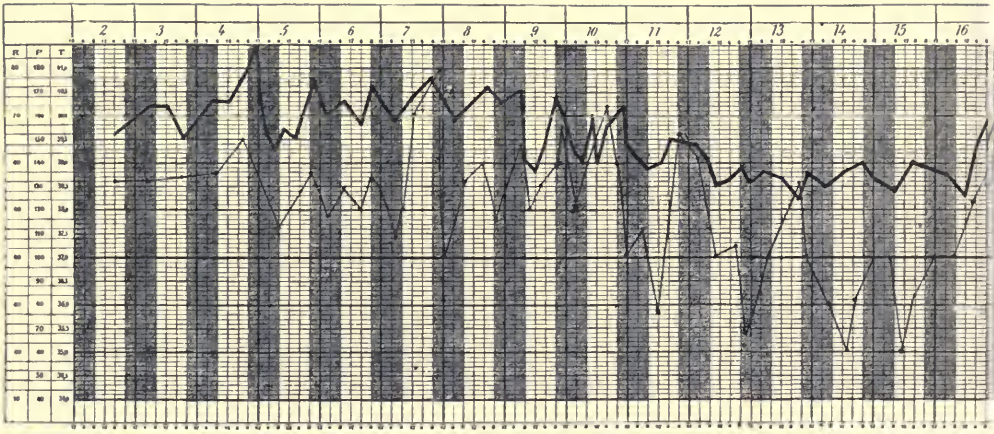
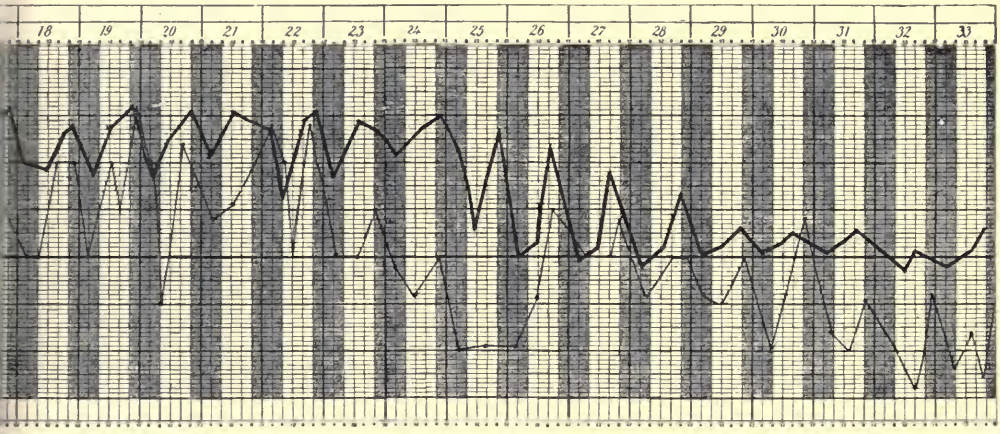


FIG. 177.—Severe typ



ever in six-year-old girl.

sionally thrush is seen. A secondary infection of the parotid gland, leading to suppuration, may ensue. In neuropathic patients the vomiting may continue for days, while in others it occurs only at the onset. In serious and often fatal forms, a marked swelling of the liver may be evident. A long continued infection of the bile ducts, a cholecystitis, or a peritonitic exudate is very exceptionally seen and only in late childhood.

The Respiratory Tract.—The nose is usually dry, and this leads the child to do injury to the mucous membrane. Epistaxis is rather common in older children, but it requires no treatment.

Otitis media accompanies the disease more often in childhood than in adult years. It often leads to a benign suppuration which does not particularly affect the course of the disease. Young children with severe bronchitis tend especially to this complication. Marked laryngitis with necrosis of the cartilages is rare.

Broncho-pneumonia hardly ever develops before the second week. Frequently it begins without any distinct symptoms in the hypostatic paravertebral form. It may extend to other large areas very rapidly. A rare, but serious complication is the appearance of an exudative pleurisy which often becomes purulent.

The heart suffers to a great extent only in severe cases of long duration. Heart failure is much more uncommon than in adults and sudden unlooked-for cardiac death is still less frequent. Toxic myocarditis often manifests itself in diminished clearness of the first sound of the heart, in marked tachycardia and, at times, even in systolic murmurs and enlargement of the heart. Bradycardia and irregularity during convalescence have no serious significance. Endo- and pericarditis are comparatively rare.

The *function of the kidneys* is very seldom disturbed to any serious degree. Even in severe cases distinct nephritis is rare. It seldom becomes a prominent feature in the disease-picture and almost always disappear when convalescence sets in. It is a remarkable fact that children who have acquired habits of cleanliness hardly ever lose control of the bowels and bladder even though they may be very ill. Even without the development of albuminuria, typhoid bacilli are occasionally demonstrable in the urine up to the period of convalescence. The diazo-reaction reappears in the event of relapse, but not in such febrile complications as broncho-pneumonia.

The Nervous System.—In late childhood aphasia is now and then observed and may be independent of apathy or stupor. During convalescence a transitory mental disturbance or depression may be noted. In severe cases, some degree of paresis of the extremities and particularly of the legs is occasionally recorded. Meningitic symptoms are more common with children than with adults. They may be significant of a serous meningitis as a phase of typhoid infection, a diagnosis supported by the increased pressure of the cerebrospinal fluid which contains an increased number of lymphocytes. From a prognostic standpoint, these symptoms are unfavorable.

The Skin.—At the onset of the disease toxic erythemata, commonly of a scarlatinal type, are not uncommon, or are at least decidedly more common than in the adult. In the later stages of the severer grades of infection,

polymorphous erythemata will sometimes appear. Dark red cheeks and cyanotic mottled extremities are unpleasant indications and evidence paralysis of the vasomotor system. After the fever has subsided profuse sweats and the consequent formation of sudamina are frequent. Later, extensive desquamation of the skin of the trunk and limbs is often seen. The surface of the face and of the hands and feet is not affected. Decubitus is much less common than in adults. During convalescence, it is not unusual to find, even in mild cases, multiple skin abscesses. These are especially common in certain epidemics.

The Osseous System.—In contrast to the slight liability of the adult the skeleton is very often affected in typhoid children. During convalescence, and sometimes within a subsequent period of three to six months, small circumscribed periostitic foci are occasionally found. The most frequent seat of these is on the tibia. As a rule they do not cause fever. Upon incision pus containing typhoid bacilli is drained from these areas. A thickening of the periosteum remains after healing. Larger areas of osteomyelitic infection are uncommon.

At times, the convalescent from typhoid exhibits a rapid gain in height. In the young this growth may be so marked as to cause transverse separations of the cutis, similar to the linea albicantes of pregnancy, on the extensor surface of the thighs

Recurrences of the disease are rather common and seem to depend more upon the character of the epidemic than upon external conditions. As in the adult, they usually appear from three to ten days after the subsidence of the first febrile period.

Diagnosis.—As the foregoing history suggests, the diagnosis of typhoid fever in children is beset with greater difficulties than it is in later life. It is oftener impossible to make the diagnosis from the clinical data alone.

A severe febrile disturbance lasting for several days or weeks, without any demonstrable local cause, presents always the probability of typhoid fever. Other obscure organic diseases must be excluded by repeated careful examinations. Into this differentiation come, among other possibilities, cystitis, certain forms of acute rheumatism, the gastro-intestinal form of influenza, and lobar pneumonia. There are central pneumonias which do not produce distinct pulmonary symptoms for perhaps two weeks. Rapid respiration, with wide expansion of the alæ nasi, suggests pneumonia. The Roentgenogram shows a shadow at the apex or at the hilus. A marked swelling of the spleen indicates typhoid. The differentiation from appendicitis occasionally causes some difficulty. Cases of typhoid occur in which vomiting and pain, localized in the region of the appendix, persist for days. On the other hand, cases of peri-appendicitis are seen in which the local inflammation and tenderness are subordinate and the fever is the most prominent symptom for some time. In these instances the discovery of a leucopenia points to typhoid. An ordinary gastro-enteritis cannot simulate typhoid for more than a few days, since its initial accompaniment of fever does not persist. Miliary tuberculosis, so common a disease in childhood, causes much diagnostic difficulty for some time, and this is equally true of

that form which is confined to the meninges. Either type commonly begins with fever, which continues for a week or two before pulmonary or cerebral symptoms become apparent. Cryptogenic sepsis, and ulcerative endocarditis, in particular, is less often a matter for differential diagnosis among children than it is with adults. It is rare in that period of childhood when special predisposition to typhoid exists. A suspicion of purulent or cerebrospinal meningitis may disturb the diagnostician for a very short time in studying the most severe forms of typhoid.

Apart from the high temperature, the most important diagnostic points, in the absence of evidence of organic disease, are the splenic enlargement, appearing at the beginning of the second week, and the presence of rose spots. In doubtful cases the diazo-reaction is very helpful and in the author's judgment is very much underrated. A strong diazo-reaction is present in almost every case of typhoid fever from the beginning of the first week and during the entire period of high temperature. If this reaction is absent, typhoid may be excluded with some degree of certainty. On the contrary, the presence of the reaction is by no means positive proof of typhoid, since it occurs in a number of other diseases and among them in miliary tuberculosis, lobar pneumonia, etc. In these diseases, however, it is rarely as distinct or as constant. In doubtful cases leucopenia often establishes the diagnosis. A definite diagnosis is often impossible, even in the second week, if splenic enlargement and rose spots are lacking, or when broncho-pneumonia is the most marked feature in the clinical picture. At this point, the laboratory diagnosis serves to clear up the situation. As in the adult, so in the child, with the beginning of the second week, the blood causes the agglutination of typhoid bacilli in fresh culture. The agglutinative power of the normal blood of the child is less than in the adult blood and agglutination, therefore, in a suspension of 1:50 may be considered positive for typhoid in the young.

The demonstration of the typhoid bacillus in the blood, usually possible within the first few days with adults, is often a very difficult matter in children, since the required volume of blood can be obtained usually only by venous puncture and veins suitable for this purpose are hardly ever available in children under five years of age. On this account, greater reliance must be placed upon the agglutination test and it should be employed in all doubtful cases. The demonstration of the bacilli in the stools and the urine is, of course, reliable proof, but the organisms are not always present in these excreta.

The prognosis is, on the whole, more favorable in childhood than in adult life, but it depends greatly upon the violence of the epidemic. Meningism, persistent coma, profuse diarrhœa, all decrease the chances of recovery. In most instances the patient convalesces with surprising rapidity.

Prophylaxis is the same at all ages. Isolation of the patient is desirable. Careful removal and disinfection of the bedclothing, etc., apt to be soiled with urine or feces, are especially important. For disinfection the linen may be placed in a 5 per cent. solution of lysol, in which it should be kept until it can be boiled. The nurse should wear a large enveloping apron in

the sick-room and must wash her hands scrupulously after touching the patient or any object in contact with him. Upon the appearance of typhoid fever in a family, well children should be given only cooked food. Fruit must be peeled. Water for washing and bathing purposes and for the cleansing of nursing bottles must be boiled. Raw milk and butter may be dangerous vehicles for typhoid, when bacillus carriers come in contact with it, either at its source or in its distribution.

The value of antityphoid vaccination has been repeatedly demonstrated. The protection is not absolute; some cases occur among the vaccinated, but their number is small. Antityphoid vaccine, as usually administered in the United States, is a suspension in physiologic sodium chloride solution of killed typhoid bacilli together with their soluble products from young agar cultures. One cubic centimeter of the suspension contains 1000 million bacteria. At present a triple vaccine is used, which contains 1000 million typhoid bacilli and 750 million of each of the paratyphoid A and B bacilli, in each cubic centimeter. The vaccine is regularly given in three doses, two days or a week or ten days apart. The best time of day for administration is about four in the afternoon, because if a general reaction supervenes, the greater part of it will be over before the next morning. The vaccine must always be administered subcutaneously and never into the skin or into the muscles or veins. A local reaction is almost invariably present, while the general reaction may be absent, mild or severe. These reactions ordinarily subside in a few days and leave no sequelæ.

The duration of the immunity is difficult to establish, but it seems to be a matter of years rather than months. Revaccination among nurses, attendants and physicians should be carried out annually.

Children, two years old or more, bear the vaccination better than adults, and should be revaccinated at two to three year intervals. Either the regular vaccine dose, namely one injection of 500 million, followed by two injections of 1000 million or quarter or half amount of the above doses (100 million and 250 million or 250 million and 500 million) can be used. As a rule only local reactions are noted.

Treatment.—The treatment is chiefly dietetic and physical, since no specific remedies are available. The value of the various sera is still too doubtful to justify their recommendation for general use. The patient and his immediate attendant should be confined to one room and complete changes of bedding should be reserved for their use. A rubber sheet may be placed over the mattress. If decubitus threatens, air or water cushions may be provided. Proper care of the skin and the frequent washing with alcohol of the parts liable to pressure are important. To this may be added, later, suitable attention to the mouth. The nourishment should be liquid or semi-liquid in form throughout the entire course of the disease. Very young children may be given mixtures of milk and gruel, or flour soup made with milk and water. For somewhat older children, heavier gruels, thin brown flour soup with milk, cocoa, etc., may be provided. If a marked tendency to diarrhœa is shown, the quantity of milk must be reduced and the use of flour, dextrin and maltose preparations, etc., increased. For still

older children a welcome addition to the diet may be prepared as follows: Cornstarch, 80 grams (2½ ounces), is stirred into a little water and brought to a boiling point. To this is added one litre (one quart), of fresh fruit juice, made from currants, raspberries, etc., and the whole is boiled for a few minutes. This pudding is eaten with milk and sugar. Gelatin puddings and finely mashed and strained apple sauce may be given also. In long and serious cases the addition of fresh meat-juice to the gruels [10-30 grams (⅓ to 1 ounce) a day], may be recommended. It should not be given undiluted. Food should be given every three hours or oftener, and even every hour, according to the condition of the patient. Very heavy feeding is objectionable. A liberal quantity of fluid is important. Water or weak tea and, if there is no diarrhœa, water with fruit juices should be offered to the patient frequently.

As the fever subsides the liquid diet may be replaced gradually by solid foods; but for diplomatic reasons it should be continued for two weeks after the disappearance of the fever—that is until all danger of recrudescence is passed. After this, thick gruels, milk toast, mashed potatoes, spinach, and chopped meat may be given. Within another week or so, rice, macaroni, and roast meats are permissible.

Medicinal therapy is of minor importance. If a cathartic is required at the onset of the disease it is as well to replace calomel with the milder castor oil. It may be given in two doses, of one or two teaspoonfuls each, at an interval of three hours. If the stools are very frequent and watery, bismuth subnitrate, in doses 0.5-1.0 gm. (8-15 grs.), or tannalbin, in similar quantities, three times a day, may be useful. For severe headache or for persistently high fever, a dose of phenacetin, from 0.1-0.4 gm. (2-6 grs.), or pyramidon from 0.05-0.2 gm. (1-3 grs.), according to the age of the child may be given. The continued exhibition of antipyretics is useless and unless the dosage is very carefully regulated is harmful. In fact their popularity has properly yielded to the virtues of hydrotherapy.

Hydrotherapy is by all means the most important method of treatment in children. Tepid baths, given twice to four times a day, as soon as the temperature rises beyond 39° or 39.5° C. (102°-103° F.), are very agreeable. For children over five years, the temperature of the bath should be initially 33°-34° C. (91°-93° F.), and should be cooled rapidly to 30° C. (86° F.). With younger children the initial temperature should be maintained. During the bath, which should last from five to ten minutes, the limbs and back should be rubbed energetically. If the patient shows apathy, somnolence, or delirium, if broncho-pneumonia or hypostasis and defective circulation are present, the tepid bath may be followed by a douche of cold water, given by means of a bath-spray or a sprinkling can, and poured over the body. This may be repeated a number of times, at intervals of half a minute. This treatment causes repeated deep inspirations. The combined bath and cold douches stimulate the nervous system and the circulation and act favorably upon the respiration. It is the most satisfactory weapon for combating the typhoid state and the development of broncho-pneumonia. The lowering of the body temperature by this

means is a secondary and less important matter. If the fever in young children is persistently over 40° C. (104° F.), cold sponges of the entire body may be given several times a day. With older patients, packs moistened with water at room temperature may be used morning and evening. The patient may be left for fifteen minutes in the pack, which may be repeated if necessary. An ice-cap may be applied alternately to the head and over the heart.

In younger children who are cyanotic and whose circulation is poor, warm baths at 37° C. (98.6 F.), rapidly increased to 40° C. (104° F.), continued from three to five minutes and followed by the cold douche, act more favorably than tepid baths. If there are signs of collapse the cold douche should be omitted. If the heart is weakening, a little coffee with milk may be given as a stimulant before the bath. Frequently repeated stimulation is required and for this purpose caffein is very useful (page 415).

Intestinal hemorrhage, of rare occurrence, is to be treated by absolute rest and the administration of gelatin subcutaneously or by mouth. Immediate surgical interference may save the patient if perforation occurs.

During the period of defervescence, quinine is a good tonic, but during the febrile stage of the disease the indication is rather for the use of an acid mixture. The following is recommended:

		Grams	
℞	Acidi hydrochlorici diluti	2.0-4.0	(ʒss-i)
	Syrupi rubri idæi	30.0	(ʒi)
	Aquæ destillatæ ad.	150.0	(ʒv)

M. Sig. Two teaspoonfuls in water three times a day.

Even in mild cases the patient must not be allowed to get up until ten to fourteen days after the fever has completely disappeared. If the convalescence is slow the patient may be given a preparation of iron and quinine and, if possible, should be sent to the country.

PARATYPHOID

Bacteriologic research has shown within recent years that occasionally a typhoid-like disease is caused by organisms of the same group as the typhoid bacillus. Sometimes the disease is due to the bacillus paratyphosus A., which resembles the typhoid bacillus in many respects and produces a similar disease-picture, but milder in character.

A disease caused by the paratyphoid bacillus B. is of more common occurrence than that due to the A. type, but it is still much less frequent than that due to the typhoid bacillus itself. It is contagious and occurs in epidemics. It is supposedly carried by food. Usually its disease-picture resembles true typhoid very closely. Frequently its course is more acute. The rose spots and the diazo-reaction are less constant than in actual typhoid. The fever often has a remittent character and the remissions are quite frequent. Intestinal hemorrhages may occur in this form, but it is not of serious degree. Its gastro-intestinal and septic features are less marked.

A diagnosis can be made by bacteriologic examination alone. This

should be directed particularly toward the agglutination test in the blood which is strongly positive for paratyphoid bacilli and persistently negative for the typhoid bacillus.

INFLUENZA AND GRIPPAL DISEASES

In 1889-91, influenza, coming out of Asia, presented a disease new and unknown to most physicians at that time. It spread rapidly as a great pandemic over all of Europe and to other civilized countries. Up to the onset of the recent pandemic, cases have been infrequent during the last few years. The laity and the profession alike have encouraged the use of the term influenza, which now is applied with freedom to the group of febrile respiratory disorders, occurring so commonly during the cold season, which formerly went under the designation of la grippe. This indiscriminate use of the term is a disadvantage, because it leads many to believe that we still have to deal with true influenza; whereas the term la grippe had come to represent a quite miscellaneous group of conditions.

It seems preferable therefore, to reserve the name influenza for that particular pandemic disorder which in our generation was first recognized in 1889-90, and to apply the term la grippe to this large number of other contagious diseases of the respiratory tract. La grippe may occur pandemically, epidemically, or in endemic form (Filatow).

The influenza with which I had an opportunity to become acquainted, as a young physician in 1889-90, recurred in 1918 as an extensive pandemic and traveled over the entire European continent in a much more malignant form than that previously observed. Most of the victims were young adults, but children of every age succumbed.

Most authors designate the influenza of 1918 as la grippe or Spanish influenza.

In discussing this subject, contrary to the custom of most text-books, to which those of Filatow and Finkelstein are notable exceptions, it seems proper to consider not influenza alone, but distinctively those conditions which are represented as la grippe. In la grippe we do not have to deal, as in influenza, with a specific infectious disease; but rather with etiologically different diseases and probably with mixed infections which produce similar symptom-complexes. The separation of these diseases into distinct groups is not yet possible, since their clinical and bacteriological peculiarities have not been studied sufficiently. As suggestive of the variant etiology of the group, it may be better to speak of grippal diseases, rather than of la grippe which in time will probably be resolved into a number of distinct disorders. At present it is only possible to separate pandemic influenza, as a definite entity, from among this general group of diseases. Even this is not clinically possible as it was upon the first appearance of the disease twenty-five years ago. It can be done only by the aid of bacteriologic examination. The points of similarity and difference will be brought out most clearly if we attempt to describe influenza and grippal disease side by side.

Etiology.—The causative organism of influenza was shown by Pfeiffer in 1890 to be a specific cocco-bacillus found abundantly in the mucus of the nose and of the bronchi. These extremely small bacilli, occurring in groups and resembling a school of fish, may be grown upon blood or upon media containing hemoglobin or two vitamins. They stain readily with carbol-fuchsin and die rapidly outside the human body. Since the bacillus does not penetrate the body, many of its disease manifestations must be considered as the result of toxic action. Latterly it has been found as an invader of the body cavities and in bronchiectasis, etc. It is not infrequently a common saprophyte.

The views dealing with the etiology of *influenza* during 1918 are still very much divided. Most of the investigators believe that the influenza bacilli, streptococci and pneumococci which are found in this disease can only be considered secondary invaders. Based on the observations of two cases of meningitis caused by a pure infection of influenza bacilli, the author of this treatise is inclined to accept the view just mentioned.

Olsen found in more than two-thirds of the cases of influenza the influenza bacillus in the lungs (166 times in 222 cases). In recent cases these organisms were present practically in pure culture.

The causative organisms of the grippal diseases vary in different epidemics. The most common of these is the pneumococcus and next in order of frequency, the micrococcus catarrhalis, Friedlander's bacillus and the streptococci; in a word, those germs which live in the mouth and the pharynx of healthy persons as parasites and which appear in large numbers during attacks of la grippe. These diseases are probably due less to an increased virulence of the organism than to an increased local sensitivity of the mucous membranes. This increased local disposition may be developed by the influences of a rough and changeable climate, by exposure to cold, etc. Children suffering with disturbances of nutrition, or with lymphatic or scrofulous diatheses, are especially predisposed. It is not impossible that the bacteria mentioned may represent in part merely secondary infections and that the specific organism actually causing the disease is still unknown.

The method of transmission of influenza and of the grippal diseases is identical. It is accomplished by direct or by droplet infection in coughing, sneezing, etc. Indirect transmission seems to be very rare, particularly in influenza. The contagiousness of these diseases is extremely great and therefore very brief contact is sufficient for infection. Characteristic of both groups of diseases is their constant appearance in epidemic form.

Influenza has appeared at great intervals of time, arising in the East and spreading through Russia to Europe where it has developed great pandemics. These major outbreaks soon faded, but smaller epidemics appeared for a number of years after each pandemic. In the smaller communities, influenza usually spreads so rapidly that the entire population is affected within a few weeks. Seasonal and meteorologic conditions do not always influence its spread.

The grippal diseases occur regularly during seasons of inclement weather; the first cases usually appear with the onset of winter. Frequently

large epidemics develop in the wake of some sudden change of weather. These epidemics take a slower course than those of influenza and may persist for many months.

Influenza and grippal diseases show some difference in respect to predisposition at various ages of life. In general, influenza affects adults and older children most frequently. Younger children are less commonly affected and if they suffer it is generally with a milder form of the disease. The infants of affected mothers or wet-nurses often escape the infection. The grippal diseases, on the contrary, are most common in early childhood and infancy and occur in very severe form in these periods of life.

Symptoms.—In the clinical picture of the individual case, it is usually impossible to distinguish between influenza and the grippal diseases. As in Asiatic cholera and cholera nostras one must depend upon the bacteriologic examination. Influenza, occurring in 1891–92–93, caused disturbances of the respiratory tract which developed very early in the attack, while in the early history of the pandemic 1889–90 these symptoms were rarely characteristic of the disease.

The points of similarity in the two disease-pictures are the following; The incubation period is very short, covering from one to four days. After the fever sets in, it may rise rapidly to 40°-41° C. (104°-106° F.). It often lasts only one to three days and falls by crisis, followed by perspiration. The fever is frequently of a remittent, or even intermittent type. Occasionally, it persists for an entire week and in rare instances for two or three weeks. Very commonly a prodromal stage occurs in the grippal diseases, with coryza, slight cough and a rise of temperature preceding the appearance of the more severe symptoms. In influenza, however, a sudden onset is the rule.

With the rise of the temperature there is often vomiting, reddening of the conjunctiva, and swelling of the mucous membrane of the nose and throat which at first is without increased secretion. An intensely livid injection of the tonsils, the uvula and the palatal arch, sharply circumscribed at its anterior border, is noticeable. The usual nasopharyngitis is often overlooked in younger children since it does not cause very marked nasal discharge. Enlargement of the cervical lymph nodes follows which in turn causes a reflex rigidity of the muscles of the neck, suggesting meningitis. In infants this inflammation occasionally leads to retropharyngeal abscess. Quite frequently in individuals of the lymphatic type the infection of the lymph nodes becomes especially prominent and may occasion an irregular fever for weeks after the original disease has disappeared—a sequel which is suggestive of tuberculosis. An annoying dry cough, often of hoarse tone, is soon in evidence. In children of lymphatic diathesis, pseudocroup is not uncommon. During the influenza epidemic of 1918–19, severe forms of laryngitis were quite frequently noticed. The cough may strongly resemble that of pertussis and may even provoke vomiting. Coughing spells often occur during the night and may present serious diagnostic difficulties. It is important to note that these short but intense paroxysms of coughing occur much earlier in grippal diseases than in pertussis, but,

as in the latter, the lungs and bronchi show no physical changes during the first few days. The tendency of the catarrhal inflammation to spread from the throat to the middle ear is very marked. Children, who are old enough, often complain of earache even on the first day of the disease; in younger ones restlessness and persistent crying call for an examination which elicits pain upon pressure at the tragus and shows reddening of the tympanic membrane. A hemorrhagic myringitis, with blood filled vesicles, is frequently seen. The accompanying otitis media often leads to suppuration and perforation of the drum. During the pandemic of 1918–19 the ears were very slightly affected.

The general health may be slightly or severely affected with the varying conditions of the disease. On account of the cough, the headache and earache, sleep is often restless and broken.

If the fever subsides in from one to three days a rapid recovery follows, the cough becoming free and the otitis healing speedily. Very often, however, the fever persists and the inflammation extends to the mucous membrane of the respiratory tract, as a whole, involving the bronchi and the lung alveoli. This is an especially common result in young children. Dyspnœa, expansion of the nostrils, and other signs of broncho-pneumonia, become apparent. The downward spread of the infection may be so rapid as to lead one to suspect a lobar pneumonia. The physical signs are often surprisingly like it. Commonly the gradual extension of the inflammation, the numerous râles accompanying it, the remittent fever, and the delayed resolution are evidences of lobar disease. In la grippe due to a pneumococcus infection, the similarity of the picture to that of primary pneumonia is easily understood and even the pathologic findings show identical conditions. In other cases, again, autopsy shows mixed conditions which indicate that lobar pneumonia and broncho-pneumonia may exist side by side. Sometimes yellowish foci are found surrounding the bronchi, while associated with these is a dense, varicolored infiltration, with hemorrhagic areas, which is essentially cellular. The pandemic of 1918–19 was characterized by a predisposition to acute pneumonia of hemorrhagic and necrotizing character.

Occasionally, a fibrinous pleurisy or even an empyema is associated with these pneumonic changes. This, in turn, may lead to purulent metastases in the bones or joints, to septic meningitis or to pulmonary abscess. In the early years of life pneumonia is the most common and the most important complication or rather accompaniment of grippal disease and in the subjects with cachexia or nutritional disturbances it is often fatal. In the ordinary forms of grippal disease the catarrhal inflammations of the respiratory tract, from coryza to pneumonia, are the commonest symptoms. In influenza, however, this is not always true. In the pandemics of 1889–90 and 1918, constitutional disturbances were much the more prevalent. Aside from slight catarrh of the nose, throat and conjunctiva, the air passages were often unaffected, particularly in young children. If they were affected, the involvement did not occur for several days.

The toxic symptoms, particularly of the nervous system, are always much more in evidence. Younger children are unusually weary; older

patients complain of severe headache and backache and of photophobia. Infants cry continually and are exceedingly restless. An extreme desire for sleep may deepen into stupor. Hemorrhagic encephalitis, meningitis, hallucinations, dementia, neuralgias and neuritic paralyses, all indicate how seriously the nervous system may be involved. In late childhood convalescence in such cases is long and tedious, even though the attack be of short duration. This nervous type is much less common in the grippal diseases. Nevertheless, severe eclampsia and meningism are not infrequent, but they are associated with spasmophilic diathesis and, therefore, they occur only in those of early years.

Gastro-intestinal symptoms may be prominent alike in grippal disease and in influenza; they represent a form of attack which is recognized also in adults. They are much more common, however, in children. In fact, the younger the patient the more frequently does the disease take this form. During an epidemic of la grippe or influenza the older members of a family may have a febrile bronchitis, while the younger children vomit and have diarrhœa. The stools may be watery or mucopurulent. If the fever is long continued, the disease may present a picture which resembles in some respects that of typhoid; a resemblance may be further supported by the appearance of an exanthem like the rose spots of typhoid fever, but of more general distribution over the body.

It is not at all uncommon to find other forms of exanthemata in the course of the disease. These are usually of a scarlatinoid type and may create much confusion. At times the exanthem resembles either measles or rubella.

In influenza, marked disturbance of the heart indicates the toxic influence of the influenza bacilli. This disturbing influence is more marked in older children; in whom, as in adults, it may cause arhythmia, a small and very rapid pulse, dilatation, and even heart failure at any time during the course of the disease and even in convalescence. Occasionally, in older children, during convalescence of an uncomplicated case, the pulse may be slow.

The spleen is always enlarged but not prominently so. Albuminuria is common in all the diseases of this group, but definite nephritis is rare. Hemorrhagic nephritis is occasionally seen. The diazo-reaction is but rarely present. During the first days of the attack in 1918-19 epidemic, the blood showed a distinct leucopenia.

The course of these diseases is extremely variable. It may be concluded, with coryza and ephemeral fever, in two or three days. It may be so mild that it is entirely overlooked. It may last for eight to fourteen days as a general febrile attack, with bronchitis or broncho-pneumonia. Again, it may terminate fatally after an illness varying from a few days to some weeks, death resulting usually from some secondary purulent infection, as meningitis, empyema, etc. There are probably no afebrile forms of the disease, but in frequent ephemeral cases, the physician is often unable to verify a rise of temperature. Fever may be entirely lacking in infections of atrophic or premature infants in whom collapse speedily occurs. In

some cases a chronic bronchitis develops or a latent tuberculosis becomes active. In older children true influenza is followed by a slow convalescence, with neuralgia and persistent weakness.

Diagnosis.—Influenza appears as a general infection in which toxic and nervous symptoms stand out prominently. It does not always involve the respiratory tract or often only as a later stage of the disease. In fact, the pandemic of 1918–19 was characterized by the frequent occurrence of very severe pneumonias. In the grippal diseases the picture is definitely one of febrile respiratory disease. These differences, however, very often disappear, and a diagnosis can be clearly made only by the coincidence of an epidemic or by a bacteriologic examination at the hands of an experienced bacteriologist.

In every instance, a number of febrile infections must be considered in making a differential diagnosis. In typhoid, the fever is more continuous. The diazo-reaction, the rose spots, and the enlargement of the spleen appear at the end of the first week. In true lobar pneumonia, also, a more continuous fever, as compared with the marked remissions of the grippal diseases, is noticeable. A severe accompanying bronchitis, the extension of the inflammation to other lobes, the delayed resolution and the absence of the crisis contraindicate pneumonia. Intense pain in the limbs may suggest acute rheumatism, but in this disease the involvement of the respiratory organs is wanting. In doubtful cases, conjunctivitis and otitis indicate grippal disease. The fever, conjunctivitis, coryza and cough resemble the initial stages of measles. Very often a differential diagnosis can be made in the prodromal stage from the Koplik's spots alone. In the grippal diseases, the scarlatinoid exanthem is transitory and often appears only with the subsidence of the fever. The angina and the strawberry tongue of scarlet fever are lacking. The suspicion of meningitis frequently causes anxiety in the grippal diseases. In influenza, this suspicion is aroused by a marked somnolence, and in other grippal diseases by an accompanying otitis and a swelling of the cervical lymph nodes, causing reflex rigidity of the neck. Meningitis is also simulated by a spasmophilic meningism. The differentiation is all the more difficult because a true meningitis in either the serous or the purulent form, and due to either the pneumococcus or the influenza bacillus, is not so very uncommon as a sequel of the grippal diseases.

In grippal bronchitis, as compared with simple bronchitis, particularly intense cough, conjunctivitis, high fever with slight constitutional symptoms, disturbed sleep, marked malaise and nervous irritability are more or less distinctive.

The differentiation of pertussis, in which the fever is very slight or entirely wanting, has been detailed in the discussion of that disease. Very often the peculiar cyanotic reddening of the throat in la grippe determines a diagnosis.

The prognosis must be made with caution even in the mild cases, since the coryza with which the disease begins is very often only the introduction to a severe and even fatal bronchitis or pneumonia and since the pos-

sibility of empyema and other complications must be remembered. In infancy, the grippal diseases, often taking the intestinal form, come next in fatality to the primary disturbances of nutrition. Even from the second to the fourth year many weak and rickitic children succumb to them.

Prophylaxis.—As a matter of prophylaxis, therefore, we cannot neglect the slightest coryza or cough and the greatest possible care should be exercised upon the appearance of even the milder affections of the respiratory tract. It is extremely important that young children, and especially those suffering with rickets or disturbances of nutrition be safe-guarded, so far as possible, from infection. Kissing, the use of common handkerchiefs, etc., should be strictly avoided even when the child is well. He should be kept away from adults who have coryza, cough, etc. If any member of the family shows symptoms of fever or of nasal or bronchial irritation, from any cause, it should be a rule of ordinary hygienic behavior to keep such a one isolated from the child as fully as possible. Very often, of course, the household environment makes the observance of such a rule impossible.

In hospitals, at least, children with febrile disease of the respiratory tract should be strictly isolated. Never should they be allowed in the same ward with infants.

Treatment.—At the beginning of the fever, or the catarrhal symptoms, diaphoretics often seem to bring the invasion to a halt. The patient should be placed in a warm bed, well covered and be given two doses, at intervals of one hour, of acetyl-salicylic acid of 0.1-0.5 gm. (2-8 grs.), graduated to his age. Large quantities of hot drinks should be given. After a profuse perspiration a hot bath may be given and the bed and body clothing changed. In spasmophilic or lymphatic children these diaphoretics cannot be employed.

Rest in bed should be continued for several days after the fever has subsided. The diet should be liquid and of scant quantity at first, especially with spasmophilics, in whom the development of convulsions and meningism dictate a brief period of starvation, which together with catharsis often gives surprisingly good results.

Upon the appearance of febrile toxic symptoms a mild course of hydrotherapeutic treatment, with warm baths, followed if necessary by cold douches, is useful. For the rest, the dominant local conditions, in nose, ear or respiratory tract, may be treated as they arise.

Disturbances of nutrition should be managed as detailed in the chapters dealing with these conditions.

Threatening cardiac weakness demands rest and stimulants, caffein, at the proper time. The use of large doses of camphorated oil (twenty per cent.) in doses of 5 c.c. twice daily, or in older children 10 c. c. subcutaneously or better intramuscularly, produced, in the experience of the author, beneficial results.

Following severe cases of long duration, tonics, preferably of quinine and iron, may be given during convalescence. Later a visit to the country or the mountains is beneficial and is a preventive of tuberculous conditions.

ACUTE ARTICULAR RHEUMATISM

(Polyarthritis Acuta)

Acute articular rheumatism is a febrile disease, which is marked by transitory, non-suppurative inflammation of several joints and frequently produces inflammatory lesions of the heart.

Verified knowledge of the nature of the disease is still limited. Doubtless processes differing in their etiology are grouped by means of similar symptom-complexes. This is especially true in the chronic forms growing out of the acute disease. Many facts suggest that in the major number of cases coming under this head, we are dealing with an infectious disorder. Some authorities hold that it is a matter of a feeble form of sepsis. It becomes, however, more and more apparent that in pure types of inflammatory rheumatism, the blood, the joints, and the heart valves are sterile. Hence we must conclude that the causative organism is still unknown (Jochmann).

The fact that several cases of the disease appear coincidently in a single house and that the disease is often preceded by angina seems to indicate an infectious agent.

The *predisposing factor of age* shows great variability. The disease is very rare between two and five years; it is more common between five and ten and very frequent between ten and fifteen. A few cases have been described in which a mother with articular rheumatism has given birth to a child who has shown symptoms of the disease at birth. These instances are very doubtful, as is the occurrence of any case in infancy.

The attempt has been made, again and again, to determine the influence of heredity, but without positive result. The relationship of cold-taking, or of residence in damp dwellings, has been suggested but it is difficult to attach any significance to these factors. The marked tendency of certain persons to repetitional attacks in the course of several months and years suggests the importance of individual predisposition, the basis of which is not as yet clear. Sometimes a familial tendency is apparent. The disease as it occurs in childhood demands special attention, since it offers a number of very marked differences from the typical forms seen in later life. These peculiarities consist in the minor importance of the joint affections as compared with the constitutional disturbance, in the imminence of serious and permanent heart complications and in its frequent association with chorea minor (see page 549).

As in the adult, the disease usually begins with evidence of inflammation in the joints, sometimes of sudden and intense and, again, of gradual and insidious development. Comparatively often, however, general disturbances, by way of lassitude, anorexia and slight fever, take a primary place in the clinical picture in the first few days. Frequently the attack begins with a catarrhal or lacunar angina, justifying the assumption that the organisms causative of the disease find their port of entry in the tonsils; as they do, in great measure, in infection of the adult with rheumatism or sepsis.

The *joint manifestations* are usually so slight and so transitory that

they are often overlooked. This is particularly apt to be true when they are of late appearance and when the attention of the physician has been centred upon the angina and the general health disturbance. Hardly ever do they become as severe as in the adult. They appear most commonly in the knee, foot, or shoulder joints and do not persist for any length of time in any one joint. The number of joints affected is usually less than in the adult. The cervical vertebral column is involved with relative frequency. The swelling of the joints is usually insignificant and is apt not to attract attention unless it is specifically sought. The oversight is the more likely since reddening of the surface occurs only in exceptional cases. Palpable exudation in the joints is ordinarily lacking. Very often no swelling can be discerned and the affection of the joint is apparent only from the tenderness which leads to fixation and in young children to the maintenance of eccentric postures. Frequently the tenderness is discovered only by systematic examination of all the joints and by the exercise of pressure and passive motion. Indeed cases are recorded in which cardiac lesions, or chorea are discovered and a carefully elicited history relates that the child had complained of slight transitory pains in one foot or knee some few weeks or months previously.

The fever is usually moderate, varying between 38° and 39° C. (100°-102° F.), rarely more and often even less. While the temperature may fall to the normal range for a few days it rises again as some other joint is newly involved.

No such constant relation, however, between the temperature and the joint manifestations always obtains; and it may well be supposed that the general rather than the local infection upon which the implication of other organs, as the heart, depends, may readily escape observation and yet play an important part.

Sweats frequently occur, but are not usually so profuse nor of so acid a quality as in the adult.

A certain degree of *involvement of the heart* is so common, occurring in from eighty to ninety per cent. of all cases, that it may be said to belong to the regular course of the disease and to dictate frequent examination of the organ. Examined daily from the very beginning of the attack, a slight systolic murmur will be heard at the apex, in very many cases, toward the close of the first week. For a long time it is impossible to say whether the murmur is functional or is the sign of endocardial infection of the mitral valve.

Observation shows that these murmurs of acute rheumatism and chorea are extremely difficult to interpret. Considering, however, that a definite valvular lesion appears sooner or later in the large majority of cases, it is fair to suppose that the remaining number which develop no dilation of the heart and no accentuation of the pulmonic second sound and in which the murmur disappears within a few weeks or months, suffer, nevertheless, a slight endocarditis. In many instances the heart lesion becomes apparent even during the attack of rheumatism, but in other cases it is not evident until after recovery from the acute disease. Endocarditis is most apt to appear at an early date when pericarditis is associated with it. Not infre-

quently it appears coincidently with the onset of the rheumatism and may even precede the latter, an indication that the joint affections are merely symptoms of a general infection.

The duration of acute rheumatism is rarely over fourteen days, saving in certain unusual forms. Indeed, cases are observed in which the joint manifestations permanently disappear within a week; but again, as in the adult, severe cases with complications may drag on for many weeks. Relapses are not uncommon. They may occur immediately after the initial attack, the same or other joints being affected or they may be postponed for weeks. Repeated attacks may be observed in the course of months or years. Rheumatism, endocarditis, and chorea may appear in rotation in the one individual; *e. g.*, endocarditis may occur six months after an attack of rheumatism; within the year, chorea may develop; and again later another attack of rheumatism, etc. In such rotary cases rheumatism may, at one time, appear first; on another occasion, chorea is the first manifestation; or, again, endocarditis may take the precedence. Evidently each and all are expressions of one and the same infectious disease. In severe and threatening cases, the triad of symptoms is simultaneously present. Occasionally, the rheumatism disappears suddenly and is replaced, in a degree, by a definite chorea.

Peculiarities of Course With The Implication of Various Organs

Heart complications take a prominent place in the clinical picture of the disease, not only on account of their frequency but also because of their importance. Rheumatism is the cause of more valvular lesions in childhood than it is in adults. Endocarditis may occur at any time. Very often a child is supposed to have survived an attack of rheumatism without permanent injury. Upon casual examination, however, months later and when no other illness has supervened, a distinct valvular lesion is found. The lesion is almost always that of mitral insufficiency. Aortic insufficiency is rare. The observant parent may report that the child has been habitually tired and pale in the interval since the attack of rheumatism, and his temperature may be found as high as 37.5 or 38° C. (99.5-100.5° F.). Frequency of pulse-rate and cardiac dilatation may be wanting. On the other hand, the endocarditis may be apparent even during the acute attack of rheumatism and may lead rapidly to the development of symptoms. A marked systolic murmur at the mitral, an accentuated pulmonic second sound, cardiac dilatation, an increased pulse-rate, dyspnœa, precordial oppression, a renewal of fever due to further involvement of the joints, etc., will ensue. Fortunately the inflammation extends to the myocardium and the pericardium in only a small number of cases.

Pericarditis is almost always associated with endocarditis and is a severe and extremely dangerous affection. It is often fatal, even in the acute stage, either in consequence of the cardiac insufficiency, resulting from the accompanying myocarditis, or from compression of the heart by the large accumulation of serous exudate. Very frequently it passes into the chronic form. In small circumscribed areas of infection, local oblitera-

tion of the pericardial space occurs, while with more general involvement, obliteration often takes place after the exudate has been resorbed. This results in death after months, or even a year or two, of extreme suffering. Pericarditis is, in fact, the most common cause of death in either acute or chronic stages (see pericarditis page 407).

Fibrinous pleurisy is not uncommon and the exudative form is even more frequent. It is usually coincident with pericarditis. The exudate is generally completely absorbed if the pericarditis does not cause death.

Severe cerebral symptoms are rare, but they give a very grave prognosis. In very serious cases attended with high fever 41° C. (105° F. or more), they take the form of delirium, with other evidences of meningitic irritation, followed speedily by coma and death.

Fig. 178.—Rheumatism nodosum. Six-year-old child.

Various forms of erythemata occasionally appear. These are either of the multiform or marginate exudative type, or of the papular variety. It must be remembered that an endocarditis often develops in the course of a traumatic erythema. Rarely have pale or erythematous areas of painful edema been described.

An unusual form of disease, seen almost exclusively in children, is rheumatism nodosum (Fig. 178). In the course of extreme cases, with cardiac complications, nodules, varying in size from that of a pinhead to that of a cherry, and painful upon pressure, appear upon the surface of the joints, most frequently of the elbow or along the course of the large tendons, or on the scalp. The nodules may be very numerous, but they usually disappear after a short time. They are of fibrous structure.

The diagnosis of inflammatory rheumatism is readily made in advanced cases. In children, however, mistakes are very common, especially when there are neither objective nor subjective manifestations of joint infection or when the cardiac symptoms dominate the disease-picture.

Differentially a number of diseases which cause pain and inflammation in or about the joints must be considered. Syphilitic osteochondritis is readily excluded, since it occurs only in young infants and will be associated with other symptoms of syphilis. This is equally true of the painful joints of florid rickets and infantile scurvy. In addition, a number of specific and septic infections which cause metastases in the joints must be taken into account. These constitute the so-called rheumatoid diseases. Among

these, pneumococcic arthritis is the most important. This is a frequent sequela of pneumonia or empyema in young children, often affecting several of the large joints, and usually proving benign in spite of a purulent exudate. The rheumatism of scarlet fever is especially innocent. It often develops in the second week of the disease and is most commonly located in the wrist. It soon disappears without leaving permanent injury. A severe pyemic inflammation of the joints which may occur in scarlet fever or in any form of septic disease must not be confused with it. Gonorrhœal arthritis is less common after vulvovaginitis than it is after ophthalmia. It occurs within two to four weeks after the primary infection. It generally affects the knee and the hip-joint, and often only one of them. It causes a painful red swelling. Its prognosis is usually good. These various possibilities must be weighed in every case of rheumatism, especially of a monoarticular form and the vulva and the conjunctiva should be carefully examined. The exudate in the joint contains gonococci. It must be remembered that hereditary lues not infrequently causes chronic inflammation of the joints in older children. This disease is remarkable for the absence of pain and for the fact that it most frequently appears in the form of bilateral hydrops of the knee. Often it is associated with a painful hyperplastic periostitis of the tibia.

Tuberculous rheumatism occupies a peculiar place. It has been described most fully by such French authors as Poncet and others but has not been fully accounted for. Tuberculous individuals, or rather persons inclined to tuberculosis, are met with in whom affections of the joints and of the vertebral column appear in their early history and these cannot be distinguished from ordinary rheumatism. Only by their obstinacy to ordinary treatment, by the fact that the involved joints are few, by their chronic course, and the coincidence of tuberculosis in other parts is their etiology recognized. The reader should be reminded that tuberculous coxitis, in its onset, may very closely resemble rheumatism of the hip or knee.

The prognosis of acute articular rheumatism must be made very cautiously. The joint affections almost always recover without leaving any permanent injury and only in exceptional instances do they pass into the chronic form.

From one-half to two-thirds of all cases are left with a chronic heart lesion. In a few cases death results from the severe infection during the acute stage; more frequently the patient succumbs to pericarditis in the course of years. Even patients who pass through the acute stage well are liable to later recurrences or to chorea or cardiac lesions.

The treatment is the same as in the adult. Rest in bed, a carefully warmed room and the avoidance of draughts are essential. The salicylates should be used in every case. Sodium salicylate may be given to infants in doses of 0.25 gram (4 grs.); to children of from three to five years, 0.5 gram (7½ grs.); and to children of from eight to ten years 1.0 gram doses (15 grs.), three times a day. Doses of acetyl-salicylic acid (aspirin), should be a little smaller. After the fever and the inflammation of the joints have subsided, medication should be continued for at least a week, but during

that time the doses may be reduced to one-half, and then to one-third of the original quantity. Many cases react favorably to the salicylates. If they fail of results, antipyrin may be tried in doses of 0.2-0.7 gm. (3½-10 grs.), three times a day. The joints should be fixed and wrapped in cotton. Later, warm baths with massage are desirable. Hot baths from the beginning are often recommended. The heart complications are treated according to the usual measures, elsewhere described in this volume. Even when all the joint symptoms have disappeared, if fever still persists the patient must be kept in bed, because of the possibility of endocarditis. In such cases and in those of advanced endocarditis small doses of the salicylates are probably useful even though it is, generally speaking, impossible to prevent endocarditis or valvular lesions by their use.

The diet should be light and very little meat should be given. Alcohol must be avoided.

After the rheumatism has subsided, careful efforts should be made to increase the resistance of the patient. At first, dry rubs with rough flannels should be given, these should be followed with alcohol rubs and later by sponge baths with water at room temperature. In the cold season the patient should wear woolen undergarments. As after treatment, if all the joint symptoms have not disappeared, sun-baths or sulphur-baths may be recommended. In chronic cardiac conditions, the mild Nauheim treatment may prove beneficial.

CHRONIC RHEUMATISM

Chronic rheumatism is even less a specific disease than is the acute form. Its customary subdivisions may be dismissed with a word.

Two forms are readily distinguished. First, a secondary form arising in cases of acute rheumatism which do not recover completely; in which new attacks develop from time to time, and which lead to permanent joint changes. These cases often begin acutely and show intercurrent febrile attacks. The exacerbations often seem to spread from centres of infection, as from the large joints of one extremity to the smaller joints of the hands and feet. The frequent appearance of endocarditis, or rather of valvular lesions in these cases may be readily understood. They rarely react to the salicylates, but may recover after many months under treatment with hot baths, massage, mud-baths, etc. Certain forms of so-called tuberculous rheumatism develop in a similar manner.

The second type is that of primary chronic articular rheumatism. This includes a group of diseases, which are as variable and as obscure in childhood, as in the adult; although they are much less frequent. A certain number of these cases begins insidiously either without fever or with a very slight rise of temperature. The joints of the toes and fingers or of the wrist and ankle are first affected, and then one joint after another is gradually, but with gruesome certainty, involved. Cardiac changes are of rare occurrence and this fact alone casts doubt upon the true rheumatic origin of the disease. This is also indicated by the fact that it frequently appears between the second and the fourth year. Fixation of the vertebral column

is relatively common. In many cases, a knob-like thickening of the phalangeal joints is observed, which in the secondary form of chronic rheumatism appears later and is hardly ever so noticeable a feature. No other very distinctive differences are determinable, at least in the more advanced cases. Subsequently, the capsule of the joint often becomes thickened and shrinks. The cartilages are eroded and undergo fibrinous changes. The joints are ankylosed. The bones and even more markedly the muscles atrophy. In a case which eventually recovered, spontaneous dislocation of one hip-joint has been seen (Fig. 179). The disease progresses slowly through a term of years and yet much more rapidly than it does in the adult. With the fixation of the spine severe contractures develop which are especially painful. Arthritis deformans of the hip-joint is not uncommon in older children and is often treated for a long period as tuberculous coxitis.

Fig. 179.—Chronic articular rheumatism. Girl three and one-half years old. Marked swelling of all the larger joints of the extremities and of the joints of the fingers.

A peculiar form of the disease was described by Still, in which the several joints of the extremities and the cervical vertebræ were gradually ankylosed. The process was practically painless. It developed in a series of attacks, accompanied by fever and by an enlargement of the spleen and the lymph nodes. The joints themselves were not destroyed.

The prognosis of the secondary form is undoubtedly better than that of the primary chronic type, from which, however, recoveries have occurred. Death usually results from general exhaustion or from secondary infections.

In the differential diagnosis of these conditions tuberculosis and syphilis, traumatic injury to the epiphyses, and occasionally joint changes incident to hemophilia, must be considered.

In the treatment of the group, the salicylates should be tried in every case. They may have a favorable action in the secondary forms. Potassium iodide and arsenic may be given. In some instances thyroid preparations have been found very useful. Recently beneficial results have been reported by the use of non-specific protein shock therapy (injections of milk, etc.). The best results, however, are obtained by physical measures, in the way of massage, passive motion, hyperemic stasis, and the various baths. Sun-baths, mud-baths, sand, and sulphur-baths, in addition to hydrotherapeutic methods, have been employed. If but few joints have been affected, the results may be improved subsequently by tenotomies, orthopedic apparatus, etc.

ERYSIPELAS

Erysipelas is an acute inflammation of the skin, spreading by way of the lymph channels and featured by circumscribed raised margins between the inflamed portion and the normal skin. It is accompanied sometimes by mild and sometimes by severe general symptoms. It is almost invariably of streptococcic origin. The disease is much more rare in children than in adults. In the new-born it plays a special rôle and is comparatively frequent. In later childhood it takes a very minor part, but toward puberty, again, it grows more common.

Etiology.—Excepting in the first few days of life, when the umbilicus forms a special port of entry, true wound erysipelas is rare in this period, and especially so since two other factors, formerly of frequent influence, *viz.*, vaccination and circumcision, are now of minor importance, thanks to the improvement of surgical technic. Formerly erysipelas often appeared in the vaccination area and may have been primarily due either to infection of the virus with streptococci, or to carelessness and want of cleanliness in the operation itself; or it may have been secondarily due to the scratching of the pustule with dirty finger-nails, an accident which still occasionally happens. In older children erysipelas arises in a large number of instances from the nose, as it does in adults, the excoriations of anterior rhinitis sicca affording a ready port of entry. In younger children a number of other points of possible invasion, such as eczematous skin lesions, fissures of the lips, ears, and genitals, scratched chicken-pox pustules, etc., present themselves.

Frequently, the disease arises by auto-infection, since streptococci are often found in the mouth or nose, upon the unclean skin, in eczematous sores, etc., for reasons not yet understood, the streptococci in these areas suddenly become virulent. In children, the disease probably begins in the throat less frequently than it does in the adult. The development of erysipelas is doubtless due also to a reduced power of resistance. This is particularly true in weak, premature infants or in those suffering with disturbances of nutrition. It is really strange that, barring the new-born, erysipelas is so rare among infants, in whom sepsis is so common and especially since their extreme liabilities to intertrigo, eczema, impetigo, etc., give ample opportunity for the entrance of streptococci. It rather gives one the impression that the lymphatic system of the child's skin does not readily admit superficial infection.

Erysipelas is generally a matter of indirect transmission. Formerly, when cleanliness was not strenuously enforced in hospitals and the knowledge of asepsis and antisepsis was scant, infection with erysipelas was a common occurrence, as is shown by the records of old lying-in hospitals, foundling asylums, and even surgical wards. Now-a-days the conveyance of erysipelas from one case to another in hospital is extremely uncommon. Even when a mother has the disease the infection of her infant is rare.

The clinical course of erysipelas in the child is similar to that in the adult, excepting in the new-born or very young infant. Generally, however,

it is relatively mild and benign. The initial chill, frequently noted in adults, is usually wanting in young children. The general health is often not markedly disturbed, even when the local manifestations are distinct. Albuminuria may be expected, but true nephritis is rare.

In anemic and feeble children the local expression is often slight. The redness may be very indistinct and the marginal elevation small; so that, in the absence of these characteristic features, the eruption is discernible by palpation rather than by sight. Severe forms are much less frequent than in adults. When they do occur, they commonly involve an extensive area of the skin, a high fever, great restlessness, delirium, somnolence, and possibly, death within a few days.

The enlargement of the spleen is usually very marked but difficult of palpation on account of its softness. The blood shows a distinct leucocytosis.

The fever may be entirely wanting in young and cachectic children. The small frequent pulse and the general prostration indicate, nevertheless, the severity of the disease.

Complications.—The most frequent complication, especially in young and rickitic children, is broncho-pneumonia. As in old people this is often the direct cause of death. Excepting in the new-born and in weakly infants, general sepsis is rare. Sometimes a widespread post-erysipelatous edema develops after the erysipelas has disappeared. This occurs most frequently on the limbs and may have an independent distribution. Subcutaneous abscesses are common sequelæ.

Relapses are fairly common in older children. Individuals with chronic eczema, rhinitis, blepharitis, etc., are especially predisposed to the disease.

Erysipelas of the new-born is hardly ever congenital, even in children born of septic mothers. Under these circumstances general sepsis in the infant is more common. Erysipelas of the pregnant mother often causes abortion or premature labor. The viable infant, in such an event, is rarely infected. Usually erysipelatous infection does not occur until after birth. The common route of infection is by way of the umbilical wound, which has been subject to neglect and want of cleanliness. Formerly, in the old type of lying-in hospitals all sorts of umbilical infection resulting in ulcer, lymphangitis, periumbilical phlegmon, and general sepsis were common occurrences. Premature and debilitated infants have the greatest liability to infection. The disease is discussed in its clinical relations on page 149.

The diagnosis is usually easy. Difficulties are most likely to arise in its differentiation from acute eczema of the face, phlegmon, and lymphangitis, but in none of these disorders do we find the characteristic raised margin. The doubt will ordinarily be cleared up by the second or third day; but there are true intercurrent forms of erysipelas and phlegmon, occurring commonly on the scalp. The prognosis, in strong healthy infants or in older children, is better, as a rule, than in the adult. In the new-born, in whom erysipelas often appears as a feature of general sepsis, the prognosis is nearly always bad. This is equally true of feeble, artificially-fed infants during the first months.

Prophylaxis depends upon the protection of the new-born by efficient asepsis and antisepsis. The isolation of a person suffering with erysipelas in the home is necessary when a new-born infant, a recently delivered mother, or any member of the family with a recent wound is in the house. In the hospital, isolation, even from the medical wards, should be complete.

Treatment.—If the disease attacks the artificially-fed infant or the new-born, every effort must be made to procure mother's milk. Without this there is hardly any hope for the new-born babe. In infancy, and even with older but feeble children, local applications of ice are contraindicated. The painting of the surface with a twenty-five per cent. mixture of ichthyol with vaseline or collodium, and the use of compresses moistened in a solution of aluminum acetate are recommended. Camphophénique may be applied with a brush once to twice daily. The use of ultra-violet rays, also injections of polyvalent antistreptococcic serum are recommended.

GENERAL SEPSIS

Under this caption it will be desirable briefly to discuss the septicemic diseases with reference to their special significance and peculiarities in childhood, although the details are considered in several divisions of this work.

Fortunately the time has passed when the majority of new-born infants in lying-in hospitals and foundling homes succumbed to puerperal fever. Nevertheless, even to-day, septic disease in infancy is not a rarity and is frequent in direct ratio to the youth of the child, so that it is most frequently met with in the new-born and in young infants. In these children the symptomatology of sepsis presents a number of peculiarities, the special consideration of which is desirable. The sepsis of the new-born which is consequent upon puerperal infection in the mother, and septicemia resulting from umbilical infection are not included in this discussion (see page 144).

The causative bacteria of sepsis in children are the ordinary pyogenic bacteria and chief among them are the streptococci, and particularly certain intestinal organisms. Relatively frequent is the colon bacillus, the pneumococcus, the staphylococcus, the bacillus of influenza, etc.

The frequency of the septic affections in early childhood is partly explained by the fact that the ordinary ports of entry—the skin and the mucous membranes, are more delicate and more easily injured than they are in older children or in adults. The tender thin epidermis is frequently the seat of fissures, erosions, intertrigo and eczema, in which the organisms producing sepsis readily find a fertile soil, and all the more fertile when its proper care and cleanliness are neglected. The organisms cause abscesses, ulcers, phlegmon, etc., which, often small and comparatively unimportant in themselves, nevertheless may serve as the focus of a fatal general infection. Similarly such a result may follow from small multiple abscesses which appear numerously in the skin of the occiput, the back and the nates of infants suffering with disturbances of nutrition. Very often the mucous membranes, imperfectly developed, permit the easy entrance of infective organisms to an extent uncommon after infancy and altogether unknown

in the adult. The permeability of the delicate epithelium of the mouth is increased by stomatitis, thrush, etc. The well-intentioned but thoughtless scrubbing of the mouth of the infant which, unfortunately, is still practiced to a great extent, increases the injury to the delicate mucosa and opens wide the door to invading organisms. Similarly, Bednar's aphthæ, often covered by a fibrinous pseudodiphtheritic exudate, or an inflamed nasal mucosa, as in syphilitic rhinitis, or adenoid growths, give easy access to the invaders. The tonsils, which play an important part in later years, are comparatively harmless in infancy; in fact, all forms of tonsillitis are rare at this age. The pulmonary affections, so often found at autopsy, following pyemia, and sometimes demonstrable during life, must usually be considered as of secondary relation. While the intestinal epithelium of the new-born is permeable to bacteria and to proteins, and while its permeability may be increased by the injuries worked by a number of intestinal disorders, we do not know what relation the normal and abnormal factors bear to each other. It is certain, however, that among these forms of intestinal disease, a streptococcic colitis often leads to general sepsis.

A very important port of entry is opened in the urinary tract by cystopyelitis (see page 439). This disorder is extremely common among young infants and must be looked for in all cases of fever of indefinite origin. In infancy, a sepsis from colon bacillus infection often arises from this source, or, to be more exact, occurs coincidently with it; since, obviously, a cystitis due to the colon bacillus must be considered as a probable metastasis from the blood infection. These infections are not infrequently the result of an alimentary intoxication.

The tendency of infancy to septic disease depends, in part, upon an initial want of protective bodies in the blood and, partly, upon the inability to form them, in reaction to the infective organism. This inability is especially marked in artificially-fed children, while the breast-fed infant is always better protected (Moro). In the artificially-fed, frequent and exhausting disturbances of nutrition favor the infection. The significance of these influences can hardly be over-estimated. The number of breast-fed infants with sepsis is extremely small, if cases occurring in the new-born in poorly conducted lying-in hospitals be excepted. Furthermore, premature and syphilitic infants show special tendencies to septic infection. In this connection, the reader is referred to the chapter upon Sepsis of the New-born (see page 144).

The bacterial diseases of infancy are peculiar in their very acute course, their rapid progress, their active spread to many organs, and their tendency to general infection. This tendency to wide distribution is seen, also, in other diseases which, in the narrow sense, are not of septic character, as, for instance, tuberculosis and syphilis. Similarly, with typhoid fever in the infant, the phenomena of general infection are more conspicuous and the local or intestinal manifestations less so than in the adult. The pneumococcus shows a peculiar tendency in childhood, and especially during the first two or three years, to widespread metastases and to the development of suppuration.

The *course* of sepsis is often a turbulent one. Following a comparatively mild onset, extremely threatening symptoms of severe infection or intoxication suddenly appear, often leading to collapse and death with surprising rapidity. This swift course is responsible for the fact that far less frequently, than in later years, does the development of the disease give sufficient time for the formation of distinct pyemic metastases. These are most often seen in the slower progress of pneumococcic sepsis. The low resistance of the infant is responsible for the frequent rupture of the primary focus of infection into the circulation, undelayed by an interposed lymphadenitis.

In the symptom-complex of sepsis, the rapid toxic action is reflected in the general condition by an apathy, alternating with restlessness, jactitation and tremor. The anxious expression, the sunken eyes, and the sharp-pointed nose indicate the severity of the disease. In the infant a general hypertonicity of the musculature is often observed. Convulsions without cerebral infection or septic meningitis are rare.

The fever is irregular and remittent, but is hardly ever absent at the onset of the attack. Later, however, collapse, suddenly appearing, may bring the temperature down to a normal or subnormal range; a result which is more common in general sepsis than it is when pyemic foci exist.

Chills seldom occur; in fact, this symptom is scarcely ever seen under any condition in small children.

The pulse is always small and extraordinarily rapid. Cyanosis and coldness of the extremities are common. The heart rarely gives any clinical evidence of organic change. Ulcerative endocarditis, which in the septic adult is of so frequent development that it may be considered the most important symptom, is only exceptionally found in young children. This is equally true of simple endocarditis. Fibrino-purulent pericarditis is more common and is usually either the accompaniment or the result of a pleural empyema. The diagnosis of pericarditis (see page 409), is seldom clinically possible. The dyspnœa and cyanosis would tend to arouse a suspicion of pneumonia or miliary tuberculosis.

In infantile sepsis certain groups of symptoms, and particularly those of the air passages, the gastro-intestinal tract, or of the skin, may dominate the disease-picture so completely, that the physician easily falls into the error of their exclusive diagnosis, while he overlooks the basic disease.

Oftentimes respiration acquires a toxic type and suggests an immediate diagnosis of pneumonia in spite of the failure to demonstrate any signs of infiltration by physical examination. A distressing dyspnœa may be misleading, but the definitely deepened respirations, the slight participation of the auxiliary muscles, and the coolness of the expired air, conditions not proper to pneumonia, will aid the clinician in reaching a correct diagnosis.

Septic disease is much more readily mistaken for severe gastro-intestinal disorders, especially in young infants, since vomiting and diarrhœa may be major symptoms. It must be admitted that as yet we do not know how frequently the primary focus of sepsis may lie in the digestive tract itself, or to what extent gastro-intestinal disturbances may appear as merely the toxic symptoms of a sepsis arising in some other part of the body. It may

only be said that in a majority of instances the gastro-intestinal symptoms are secondary to the sepsis. Since cases of severe streptococcic infection of the bowel, producing ulceration and sanguino-purulent stools, do occur, it must be admitted that a true primary intestinal sepsis may exist. When neither primary nor metastatic foci can be found, the real causative agent may be hard to discover. This is particularly true when icterus and cutaneous hemorrhages, which are characteristic of sepsis and are not associated with primary gastro-intestinal disease, fail to appear.

The spleen, as a rule, is markedly enlarged but is often impalpable, on account of its softness. Enlargement of the liver is so common a symptom in infancy that no conclusions can be drawn from it alone, but if it is accompanied by icterus, fever and serious disturbance of the general health, the possibility of sepsis must always be considered. Nephritis of variable degree is of very constant occurrence.

The skin is very commonly involved. Primarily, an erythema of varying form, scarlatinal, rubeolar, or urticarial, usually appears. No great significance can be attached to this since it occurs in innumerable minor diseases, in intestinal disturbances, etc. Vesicular, pustular, or pemphigoid eruptions, however, are more serious and more suggestive of sepsis. *Cutaneous hemorrhages* are of very grave importance. They are extremely common in sepsis and are rarely seen in infancy under other than septic conditions. They are considered to be due to capillary bacterial emboli. They vary in size and number. Often no larger than a pinhead, they may extend, in some cases, with frightful rapidity, to include large areas. Frequently these hemorrhages are not of altogether spontaneous occurrence, but arise in areas of the skin exposed to slight pressure, as over the patellar ligament, after tapping with the pleximeter for the knee reflex, or at the site of camphor injections, etc. Their significance under such circumstances is not diminished, for they often give the first signs of sepsis.

Hemorrhages from the mucous membranes of the conjunctiva, the nose, the stomach, or the intestinal tract, frequently occur. At times these hemorrhages, whether in the skin or from the mucous membranes, or simultaneously from both, so govern the entire clinical picture that the inexperienced observer is likely to make a diagnosis of primary hemorrhagic diathesis. The benign forms, however, of the purpura of Werlhof are extremely uncommon during the first year. In the very nature of the condition a distinct differential diagnosis is often impossible, since many cases of hemorrhagic diathesis are of bacterial origin. Nevertheless, careful observation in most of the septic forms soon reveals their true character, either by the consequent disturbance of the general health, by the development of icterus, nephritis, etc., or by the clear indication of a primary focus of infection. In later infancy and in older children, cutaneous hemorrhages occasionally appear under conditions of atrophy or chronic disturbance of nutrition. These usually appear in the skin of the abdomen. They must be regarded, in all probability, as an expression of injury to the vessel walls, which is susceptible of recovery.

The diagnosis of sepsis often meets with great difficulties, when no distinct port of entry, no primary focus, and no metastases can be found. The demonstration of bacteria in the blood is extremely difficult in infants, from whom it is hard to get sufficient blood for examination. Then, too, bacteriemia is not synonymous with sepsis. In even mild cases of lobar pneumonia, for instance, it is nearly always possible to obtain a culture of pneumococci upon proper media. Clinically, severe gastro-intestinal disorders often resemble sepsis in many respects. In such cases, differentiation is readily made if cutaneous hemorrhages and icterus appear.

The prognosis is generally bad. In advanced infancy it is not entirely hopeless. Pneumococcic sepsis is the most favorable form and recovery from it may take place in spite of numerous bone and joint metastases.

Prophylaxis accomplishes much more than treatment. Scrupulous care and extreme cleanliness give great protection to the young infant. Breast feeding must be considered the best prophylactic and the best remedy. Artificially-fed infants with disturbances of nutrition succumb rapidly. The smallest rhagades, or the most minute pustular eruption demand careful or antiseptic treatment.

Treatment.—Up to the present time the only treatment for developed sepsis is symptomatic, since no specific serum therapy can yet be said to give any certain results. Abscesses must be opened as soon as possible. Hemorrhages are often entirely beyond control a characteristic, indeed, of the hemorrhages of sepsis. For epistaxis of long duration tampons should be used. Recently, coagulin has been employed. Some clinicians recommend non-specific protein shock therapy. In persistent anorexia very concentrated food should be given. For further details consult the paragraph dealing with the Treatment of Hemorrhagic Diathesis Page 190. External hemorrhages may be treated by compression, by applications of gelatin, or by the galvano-cautery. The subcutaneous injection of 10 to 20 c.c. (⅓-⅔ ounces), of a 10 per cent. sterilized solution of gelatin, may be tried. In gastro-intestinal hemorrhage, gelatin may be given internally. Liquor ferri chloridi may also be used in doses of two or three drops, in milk or gruel, every few hours.

IX.
TUBERCULOSIS

BY

C. Frh. von PIRQUET,

Vienna.

REVISED AND EDITED BY

HENRY DIETRICH, M.D.,

Attending Pediatrician Children's Hospital, Los Angeles, Calif.

At one time tuberculosis was looked upon as essentially a disease of adults, because the cavernous pulmonary phthisis or consumption was supposed to be its main form. Not until Koch discovered the tubercle bacillus in 1882 and showed that a large number of diseases of different systems of organs were due to this same organism did the clinical conception of tuberculosis begin to embrace a much larger field. In recent years it has been shown, by careful postmortem study and by the use of the local tuberculin reaction, that tuberculosis not only occurs frequently in childhood, but that it is the most important chronic disease among the children of the wage-working classes of the great cities.

In the large majority of cases, the causative organism is the human type of the tubercle bacillus. In but a very small per cent. is the bovine type of bacillus demonstrable. Furthermore, the latter does not produce so severe a form of disease as does the human type.

Every human being is probably predisposed to tuberculosis in the sense that infection with virulent tubercle bacilli causes disease. An individual difference of predisposition may lie in the fact that the same infection may produce disease of greater degree of severity and destructiveness in one person than another. In this respect the age at which the infection occurs is especially significant. It is the more dangerous the earlier its invasion in childhood. For this reason alone the hereditary transmission of tuberculosis is a matter of little inportance in later life. Children infected during fetal life, die during the early months of infancy. Such transmission in utero is extremely rare. Indeed, it hardly needs to be considered from a practical standpoint. Infection from a tuberculous placenta during birth, which is accepted by Rietschel, may occur in some of the cases that die during the first year of life. Prolonged latency of such an infection, causing tuberculosis at a later period of life (Baumgarten) is not probable, even though, according to Bartel's findings, it cannot be wholly excluded.

Since it is clearly shown by a comparison of autopsy records with the history of tuberculin reactions that the frequency of tuberculosis in childhood increases from year to year, coincidently with the percentage of children who have survived acute infectious disease, it seems quite evident

that the tuberculous infection is not congenitally transmitted, but is acquired from external sources in later life. During the last few years there has been much discussion concerning the mode of infection. Certain it is that the first manifestations are nearly always to be found in the lungs and bronchial lymph nodes. Since the regional lymph nodes are always the first to become diseased in infections of the skin (Cornet), we may accept the simplest explanation that in disease of the bronchial glands, also, the infection comes from the lung. The tubercle bacilli have probably reached the lung by the inhalation of droplets containing them (Flügge). From the reports of Parrot (1876), Küss (1898), Albrecht (1909), and especially from the excellent studies of Ghon (1912), we know that in carefully made autopsies of tuberculous children a port of entry, a primary lesion, can always be found. In the great majority of cases this primary focus lies in the lung and most commonly not in the apex but in various other parts.

In several cases in which the primary focus was in the lungs, Ghon found the various lobes affected as follows: upper right lobe, fifty-seven times; middle right lobe, fourteen times; lower right lobe, thirty-nine times; upper left lobe, fifty times; and the lower left lobe, forty times. In other words, the frequency of the primary affection in the various lobes corresponds to the size of the lobe.

According to Heinrich Albrecht, the primary lesion in the lung varies in size from that of a millet-seed to that of a hazel-nut. It is a round focus and shows a small fissure at the centre corresponding to the line of the bronchiole. The focus is usually isolated. More rarely, several foci are found in the same lung or in both lungs. The centre is at first caseated; later it goes on to sclerosis and subsequent calcification or fibrous contraction which usually begins by encapsulation at the outer margin. Finally, the focus may be reduced to a small scar with a granular particle of calcium salt at its centre and can be found only by extremely careful examination of the entire lung. In other cases, the focus does not heal and a small cavity communicating with a bronchus is formed. Miliary nodules, which gradually enlarge, appear around it.

The tubercle bacilli may, however, invade the body by some other route. The frequency of primary infection of the gastro-intestinal tract has been much debated. It seems that such mode of infection is more common among infants in countries where cow's milk is fed without boiling, as in England or America.

In the carefully conducted autopsies made by Albrecht and Ghon, of Vienna, very few definite primary foci in the intestine were observed. In 1060 autopsies on tuberculous children, Albrecht found but seven with primary intestinal tuberculosis. Ghon found three in 189 cases.

Other mucous membranes and the skin, also, may occasionally serve as a port of entry for the tubercle bacillus. As compared, however, with primary lung infections these cases are extremely rare.

In his series, Albrecht found one primary focus in the nose, one in the mucous membrane of the cheek and one in the tonsil. Ghon, in his series, found one primary focus in the skin and one in the tonsil.

The spread of tuberculosis from the primary lesion to the regional nodes, occurs by way of the lymph channels. The small nodes at the bronchial branches and the nodes at the bifurcation of the trachea, enlarge and caseate, From these the infection spreads to the nodes lying along the trachea.

With this course the spread of the process of infection in late childhood usually terminates. In early childhood, however, a further spread from the primary focus to the surrounding tissue commonly occurs. The focus enlarges in all directions, reaches the pleura, where it causes serofibrinous inflammation, and finally occupies an entire lobe, which may become cavernous.

The bacilli from the primary focus having reached the bronchi, extend from there to the mouth, nose and conjunctiva and infect the regional lymph nodes of these parts. From the mouth and pharynx the bacilli are swallowed and reaching the intestine develop intestinal ulcers, caseation of the mesenteric nodes and peritonitis.

Distribution by way of the blood is especially dangerous to life. This occurs when a large number of bacilli reach the blood from the primary focus directly or by rupture of a caseated node into a vein and are thus spread broadcast over the body. The tendency of the infection to spread by the blood channels decreases from year to year. This is indicated by the statistics of tuberculous meningitis, which is most frequent at the end of the first year.

In the large study of the disease, however, we must not consider the parasite alone as though it were the only factor controlling the situation. As a result of the infection, an allergy—a change in the reactive power of the organism, occurs. Specific substances of the nature of antibodies, or ergines, which have a digestive action upon the parasite are formed. The products of this digestion seem to be the cause of such effects upon the general organism as fever, etc. They probably also cause the manifestations of inflammation around the tuberculous focus, as well as the scrofulous catarrh of the mucous membranes. In the acute diseases, such as chickenpox and measles, we may suppose a similar formation of antibodies which digest the infective organisms and thus form substances causing the inflammatory conditions. In the latter infections, however, the digestion destroys all the invaders and the disease ends with crisis. In tuberculosis, on the contrary, all the micro-organisms are not destroyed. The bacilli at the centre of the foci remain alive and are merely encapsulated. If occasion arises they may again get into the circulation and cause new foci.

The opportunity for reinfection is also afforded in another way; when the antibodies become, for some reason, inactive and the bacteria get beyond their power. This may occur in such anergic periods as are observed during measles. In this disease a reduction of the intensity of the tuberculin reaction always takes place when the rash appears. For about a week the reaction power is reduced to a minimum, after which it gradually reappears. Clinical experience shows that tuberculosis frequently spreads very rapidly at this time. It is probable that other acute infectious diseases act in a similar manner and that disturbances of other kinds, such as pregnancy, or underfeeding, hard work and the like, may have similar effects.

Chronic cavernous pulmonary phthisis, rare in early childhood, but the most important form of tuberculosis in later life, may probably be traced back to latent tuberculosis, acquired early (Behring, Hamburger, Römer); especially since animal experiments have proved that an intravenous injection of tubercle bacilli, obtained from the animal itself, results in cavity formation (Römer).

According to F. Hamburger, we must recognize three distinct stages of tuberculosis: (1) The primary lesion with infection of the regional lymph nodes and the surrounding tissue. This may mark the termination of the disease; or (2) a secondary stage with hematogenous and lymphogenous extension to the various organs may be added. Finally, after years (3) the tertiary stage, manifested chiefly by cavity formation in the lungs, sets in.

The Frequency of Tuberculosis.—As a result of its irregular distribution and the variable character of the disease at successive periods of life, we get very different views of its frequency, according as these are based upon statistics derived from the death record, from the clinical history, from the autopsy findings, or from the results of tuberculin reactions.

The deaths from tuberculosis are at a maximum during the first year, as a consequence of the low resistance of the child at this age. They are infrequent during the rest of childhood until puberty approaches, when the number again increases as a result of the development of the pulmonary form. Thus, for instance, the statistics of the United States for the year 1900 show the following death record from consumption for successive age-periods of five years.

Age	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	100
Deaths	**39**	11	17	71	137	**154**	133	114	82	57	50	40	31	26	16	11	6	

Cornet properly emphasizes the fact that this statistical viewpoint is one-sided. A better comparison is secured if the number of deaths is compared with the number surviving of the age under consideration.

In Prussia, the average for sixteen years shows that the numbers of deaths per 10,000 at each age were as follows: (Cornet)—

Age	0	1	2	3	5	10	15
Boys	**23**	21	12	6.9	**4.5**	4.9	
Girls	**26**	21	14	8.0	**6.0**	8.9	

From this table it is seen that the first high figures rapidly fall during childhood. Among boys, the minimum rate is reached at about the tenth year. For girls, the figures are low during the first year and higher than those for boys during middle childhood. Later, women are at first chiefly

involved in the increase caused by pulmonary tuberculosis, but in a short time the death-rate among men surpasses it to a marked degree.

Age	15	20	25	30	40	50	60	70	80	100
Men	18	32	37	44	55	76	100	69	26	
Women	20	25	33	38	38	50	68	46	20	

According to the clinical manifestations, on the contrary, the middle years of childhood are much more actively involved, since the numerous exhibitions of the secondary stage of the disease (glandular, bone and joint tuberculosis), which are not fatal, appear at this time.

Statistics of clinical manifestations are hard to obtain, since they depend too much upon the subjective consideration of the case. The figures are extremely variable and include numerous cases of indefinite diagnosis.

The results of autopsies, on the contrary, are extremely valuable if, as Ghon and Albrecht have done, the greatest pains are taken to find even the minutest tuberculous changes in the lungs and lymph nodes.

These reports show an increasing progress of tuberculosis from the first year:

Age	0	1	2	4	6	10	14	
	15	40	60	56	63	70		Percentage of tuberculosis at autopsy, at various ages (F. Hamburger.)

This form of investigation does not, however, give an idea of the frequency of tuberculosis in general, since in some of the cases, tuberculosis has been the cause of death; in others, it has been but a secondary cause; while in still others, it is but a minor part of the findings.

The frequency of tuberculosis among apparently healthy children may be determined by the tuberculin reaction. In Vienna, Hamburger and Monti obtained the following figures from the results of the careful application of the tuberculin reaction in 509 children who showed no clinical signs of tuberculosis:

Age in years	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Percentage of positive reaction		9	20	32	52	51	61	73	71	85	93	95	94	94	

From this table the enormous frequency of tuberculosis, even in childhood, will be seen. It must not be forgotten, however, that these figures are obtained in a children's clinic frequented by the poorest of the popula-

tion of a large city. Schlossmann properly calls attention to the fact that conclusions as to the frequency of tuberculosis, among wage-workers alone, can be drawn from these figures and that the better situated classes would show very different results. Among a large number of children of well-to-do parents he found only about 5 per cent. of positive reactions.

These differences between the poor and the rich are due, doubtless, to the great fact that among the former there are to be found a larger number of adults with open phthisis and that these affected persons live in closer contact with the children. If there is an open case to spread tubercle bacilli in a family all the children become infected. Pollok found that of 285 children who lived in a tuberculous environment, only six gave negative and 279 gave positive tuberculin reactions. He then undertook to determine whether the time of infection had any effect upon the form of the disease. He learned when the phthisis began in each open case and from this he calculated the age at which the children were liable to infection. From this inquiry he discovered the important fact that of fifty-seven children who had not come in contact with an open case until after their third year, only seven exhibited clinical manifestations of the disease, while the rest showed their infection merely by the positive tuberculin reaction; that is, they had escaped with a primary lesion and the involvement of the regional lymph glands. Of sixty-one children infected during the second and third years, forty-five showed clinical symptoms which resulted in the death of seventeen of them. No spread of the process occurred save in eighteen cases.

The findings for the first year were the most important. Of 207 who were exposed to infection at this age, only 7 or 3 per cent., remained without symptoms. All the rest became ill and ninety-one of the cases proved fatal.

CLINICAL MANIFESTATIONS

Primary Stage

Even though tuberculous infection may occur at any age, as indicated by the appearance of a primary focus, the clinical symptoms of this stage are recognized only in infancy. In older children their onset has, so far, escaped recognition.

The general manifestations of this primary stage are fever, emaciation, and anemia. The fever may appear either in characteristic evening rises, or it may be very irregular in its onset. Sometimes only very slight rises above the normal point are seen. The emaciation may begin very suddenly, especially in young infants. In older children, and more rarely in infants, it may be absent for a long time. In severe emaciation the skin has a wilted appearance, a very constant manifestation of cachexia in childhood. Night-sweats are common among cases in childhood, but are rare in infancy. Anemia, like the cachexia, may be extreme in some cases and entirely lacking in others. All in all, these general initial symptoms are very indefinite and may serve only to excite a suspicion of tuberculosis, which may be confirmed by a tuberculin reaction or by the appearance of local manifestations of the disease.

The local developments of the primary stage depend entirely upon the localization of the primary lesion. In by far the larger number of cases this is in the lung and the symptoms then are due, in part, to the focus in the lung tissue, but more especially to the swelling of the bronchial and tracheal lymph nodes in its immediate region

TUBERCULOSIS OF THE BRONCHIAL LYMPH NODES

Cough is usually the first symptom that calls attention to the condition. It is of a hollow, barking quality, with a metallic ring, and occurs periodically. It may become so severely paroxysmal as to remind one of early pertussis, from which it can be distinguished only by its course. True whooping-cough goes on to severe inspiratory attacks with cyanosis and vomiting in the course of a few weeks; but a tuberculous cough remains the same or is accompanied by signs of expiratory dyspnœa. This dyspnœa is caused by the pressure of the swollen peritracheal nodes upon the trachea and bronchi and is the more frequent the younger the child, because the thinner and the softer, the respiratory tubes the more readily are they compressed.

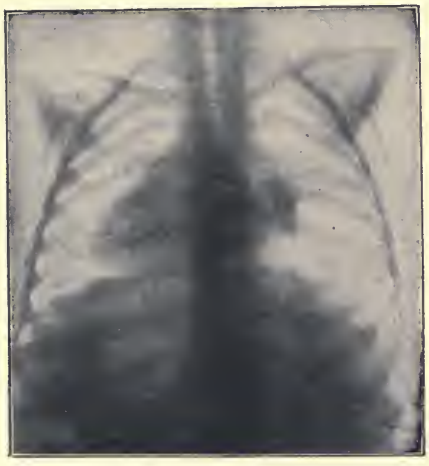


Fig. 180.—Tuberculous bronchial lymph nodes, right side. (Children's Hospital, Vienna.)

At autopsy, the larger right bronchus is usually found to be compressed by large and caseated lymph nodes. This generally occurs at a point between the bifurcation and the separation of the bronchus for the upper lobe (Schick). The expiratory dyspnœa is characterized by a loud whooping which lengthens the expiration and makes it seem forced. The inspiration is hardly audible and is often accompanied by a retraction of the thorax in this phase. The frequency of the respiratory rhythm is not notably increased. If the child has cried or coughed severely, the breathing may be very labored for a time, but with rest the expiration gradually becomes quieter and in mild cases the sound may disappear entirely (Schick).

As a result of marked pressure upon the trachea, the persistently difficult expiration may lead on to atelectasis of the lung, while the compression of the blood-vessels is shown by the distended veins and the cyanosis of the head, the face, the thorax and the drumstick fingers

The enlarged nodes cannot always be demonstrated physically. It is often possible with the Roentgen picture, but seldom by percussion.

D'Espine's sign may be of some aid in demonstrating the enlarged nodes. This sign depends upon the change of the voice sounds at the bifurcation of the trachea as heard over the spine. The change normally

occurs at the level of the seventh cervical spine or possibly at the level of the first dorsal. When the change is much lower it may be due to the transmission of the sound by the enlarged nodes in this region.

The Roentgenogram shows spots beside the spinal column. These are seen chiefly on the right; on the left they are often covered by the heart. We must remember that at this point the blood-vessels of the hilus always cause a shadow and the diagnosis of tuberculosis may be made when the shadow is larger and more intense than usual.

In those cases in which disease of the bronchial nodes has advanced far enough to give the symptom of expiratory dyspnœa, the Roentgen picture is usually very convincing (Figs. 180-181). The percussion over the spinous processes gives slight dulness in the region of the upper thoracic vertebræ (de la Camp), which cannot, however, be demonstrated beyond controversy. A dulness in the intrascapular space, especially toward the right, is more

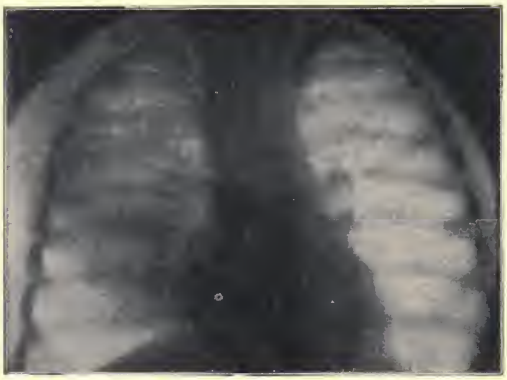


FIG. 181.—Tuberculosis of the bronchial lymph nodes and of the right upper portion of the lung in a ten-month-old child (Schick-Sluka)

significant. Anteriorly, dulness to the right of the sternum is not commonly due to disease of the bronchial nodes alone, but is also caused by tuberculous peribronchitis.

PRIMARY TUBERCULOSIS OF THE LUNGS

This suggests a further local symptom of primary tuberculosis, incident to the unavoidable spread of the bacilliary focus in the lung to the surrounding tissue.

In older children the primary stage usually ends with the infection of the lymph nodes; but in the infant the infection passes directly to the neighboring tissue. From the primary focus in the lung or by the rupture of a caseated regional node into a bronchus, the infection spreads to the adjoining lobe, resulting in either a tuberculous bronchitis or a caseating pneumonia.

The symptoms of this bronchitis can be distinguished from those of catarrhal bronchitis by the general manifestations which it accompanies and by the chronic course it pursues. It is characterized by a dry cough without

expectoration, hectic fever and a bad general condition. The history of tuberculous pneumonia is somewhat more distinctive. It is differentiated from lobar pneumonia by the absence of a high continuous fever and the ensuing crisis, and from broncho-pneumonia by its slight subjective manifestations and its long duration. It is distinguished from the chronic broncho-pneumonia, often seen in measles, pertussis and diphtheria, by the tuberculin reaction alone. The pleura shares in the process at the point where the tuberculous focus reaches it. An adhesive pleuritis occurs which is hardly ever diagnosed during life.

Pollak's compilation may be employed to give some idea of the frequency of the individual symptoms in infancy. Among ninety-two tuberculous infants, forty-six, or one-half, had clinical symptoms due to involvement of the bronchial nodes, in the way of severe cough or the expiratory whoop, or both. In seventeen cases, evidences of pulmonary infiltration or cavity formation were shown; and in two instances a serous pleural exudate was found.

In twenty-two cases, skin tuberculides were discovered; in six, phlyctenulæ were noted; in six others, tuberculosis of bone (fungus, spina ventosa, etc.); in three individuals, other forms of secondary infection were observed; and seventeen children died of miliary or meningeal tuberculosis.

We should pay more attention to the skin manifestation of tuberculosis. In doubtful cases, careful inspection of the entire body for evidences of tuberculides, lichen or erythema nodosum may be of considerable assistance on arriving at a diagnosis.

THE SECONDARY STAGES; OR THE GENERAL SPREAD OF TUBERCULOSIS

The secondary stage of tuberculosis is characterized by the spread of the tubercle bacilli in various ways. This spread is not of constant occurrence in the picture of the disease. In all probability, it is never seen in later childhood after the ordinary infections of that period have developed. On the other hand, it is almost always the cause of death in those children who have been subject to infection with the tubercle bacillus during infancy and have not succumbed to its primary manifestations.

The most serious development of the secondary stage is its miliary spread; the distribution of a large number of tubercle bacilli by way of the blood to the various organs. Usually, and particularly in children of from two to six years, the meninges are especially affected and death follows the picture of tuberculous meningitis, a form of the disease discussed in another part of this book (page 458).

If the bacilli do not pass to the brain or but in very small number, the picture presented is that of true miliary tuberculosis. In infants it is often impossible to distinguish its distinct onset, and the miliary tuberculosis is recognized only at autopsy.

In older children, however, it develops, as a rule, as an acute infectious disease.

It may be supposed that an incubation period occurs. The interval

between the first spread of the infection from its primary focus to the appearance of its early general manifestations is probably one to two weeks. This period may be entirely without symptoms, or only such indistinct prodromes as anorexia, lassitude, etc., may be noted. Later, high fever of an irregular type appears, with rapid pulse and slight cough. The spleen is at the very least enlarged enough to be palpable.

The miliary symptoms usually last two to fourteen days and rarely several weeks. The bronchitis increases and may become so severe as to simulate tracheal stenosis. Slight cyanosis is nearly always present. The sensorium is usually slightly clouded and individual signs of meningitic involvement may be observed.

The differential diagnosis must first of all exclude typhoid fever; a positive Widal reaction indicating typhoid and a positive tuberculin reaction, tuberculosis. Just as the Widal reaction may be negative at the onset of typhoid, so the tuberculin reaction may often be absent during the period of miliary dissemination. A negative result is not, therefore, significant. The gradual disappearance of the reaction when repeatedly applied or the occurrence of a cachectic reaction is of value. The appearance of tubercles in the choroid is indicative of miliary tuberculosis and for this reason an examination of the optic fundus should always be made even though it gives positive results but rarely. At times the Roentgenogram alone, showing the minute mottling of the lung, will clear up a diagnosis.

The X-ray should be employed on every case where there is a suspicion of acute miliary tuberculosis. The symptoms and signs are often so indefinite and the Roentgen findings so characteristic, that a diagnosis is only possible with the aid of the X-ray plate.

Miliary tuberculosis which can be recognized clinically, is practically always fatal. This is also true of the dissemination of tubercle in the meninges, so soon as its symptoms appear. It is impossible to affect the course of miliary tuberculosis by treatment. The twenty-four cases of recovery from meningitis collected by Barber and Gougelet, show that children may survive miliary dissemination. It is known that a limited miliary spread may often occur in the course of recent tuberculosis, without fatal result, provided few secondary foci are formed, and in not necessarily vital organs.

Most of the subcutaneous tuberculous deposits of the secondary stage of the disease, can be explained only upon the assumption, that tubercle bacilli get into the blood-stream and are arrested at some point in the systemic circulation, where they form new colonies which excite local reaction processes. The numerous manifestations of the disease in the skin, the mucous membranes, the serous membranes, the bones, the brain and the sexual organs belong in this group According to Ghon, the lymph mechanism, which is almost always coincidently affected, is not infected by way of the blood but through the lymph channels from peripheral foci.

There is still another way by which tuberculosis may spread Tubercle bacilli from pulmonary foci, reach the mouth through the upper air pas-

sages, being swallowed enter the digestive tract where they infect the intestinal mucosa and the lymph nodes of the sublingual, tonsillar, pharyngeal, cervical and mesenteric chains.

SCROFULA

In many children the secondary stage of tuberculosis leads to a symptom-complex designated as scrofula. While Laennec interpreted this group of symptoms as tuberculous, Virchow held that tuberculosis and scrofula were different diseases and it was not until the tubercle bacillus was discovered that scrofula was again regarded as a manifestation of tuberculosis.

Why tuberculous infection does not develop these phenomena in all individuals is still a matter of debate. Just at present, the theory that an hereditary predisposition, an anomaly of the tissues, or a fault of metabolism, determines this form of reaction to the tubercle toxin (Escherich,

FIG. 182.—Physiognomy in scrofula. Nose and upper lip thickened, rhinitis, conjunctivitis (phlyctenulæ and photophobia. (University Children's Hospital, Munich, Prof. Pfaundler.)

Moro), is predominant. It is supposed that children, suffering with lymphatism or an exudative diathesis, when infected with tubercle bacilli show, as a result of the peculiar quality of their tissues, the severe chronic catarrh of the mucous membranes and the bone lesions which we class as scrofula. Without such infection, they suffer only the milder indications of eczema and bronchitis and a tendency to enlargement of the lymph nodes.

It may be considered improbable that such an anomaly of metabolism favors infection with the tubercle bacilli; nor does it seem clear that a predisposition to the scrofulous type of development must always be inherited. It is a question whether the tendency may not be acquired with the tuberculous infection at a certain early age, or in the course of previous infections with other micro-organisms as a result of frequently repeated small infections.

The most important forms of scrofulous disease are found in the following organs:

1. The Lymphatic System.—The lymph nodes at the angle of the jaw are most frequently affected to a noticeable degree. A hard, painless swelling, varying in size from that of a bean to that of a cherry, is formed. Pres-

ently the swelling affects other neighboring nodes, especially those behind the sternomastoid, above the clavicle and in the sublingual space. A widespread swelling of the entire lymphatic system in the neck may result. The condition must be differentiated from leucemia by the blood findings. Pseudoleucemia is indicated by a large spleen and a negative tuberculin reaction.

If the nodes soften and suppurate the diagnosis of tuberculosis becomes clear. The skin over the enlarged nodes becomes brown and spontaneous rupture may occur. Fistulæ may form which heal slowly, leaving irregular scars.

Superficial nodes in other parts of the body may be similarly affected. This, however, usually occurs only when tuberculous disease of the skin, bone, or joints has developed in the areas drained by these nodes. We have already discussed the mediastinal and peribronchial nodes which in the scrofulous form of tuberculosis are always considerably enlarged.

Fig. 183.—Scrofula in a year and a half-old girl. Characteristic face: eczema, especially around the mouth, nose, and ears. Thickening of the upper lip. Photophobia (phlyctenular conjunctivitis). (Children's Hospital—Heidelberg, Prof. Feer.)

2. The Osseous System.—Tuberculous foci may form in various bones. Those deposits which do not affect the general well-being to any marked degree, as small foci in the bones of the fingers, the wrist and the ankle are usually classed as of the scrofulous type. Tuberculous inflammation in bone results both in destruction and necrosis and in the formation of sequestra and in periosteal proliferation. A spindle-shaped swelling of the phalanges is characteristic (Fig. 184). Such foci may be absorbed, but more frequently they rupture, forming fistulæ, large ulcers and ultimately scar tissue which is adherent to the bone. The termination of the local infection is usually good if the general condition of the child improves.

3. The Mucous Membranes.—Hypertrophy of the tonsil and chronic catarrh of the respiratory tract of children of a lymphatic habitus or with exudative diathesis cannot be classed as certainly scrofulous, since they may occur without tuberculous infection. The mucous membranes of the nose, ears and, particularly, of the eyes are involved in the tuberculous process in almost a pathognomonic relation.

Lymphatic or phlyctenular conjunctivitis begins in small nodules which are rapidly surrounded by a bundle-like arrangement of blood-vessels. These are to be considered as arising from tubercle bacilli or their toxic derivatives which enter the conjunctival sac by way of the lachyrmal duct.

These nodules usually lie at the sclerotic margin of the cornea or, more

rarely, in the middle field of the cornea. Their surface soon becomes eroded and a small ulcer forms which commonly heals within two or three weeks. This feature of the disease is in itself benign, but mechanically it often involves great danger to the eye when the lesion is in the cornea and the ulcers affect the deeper layers. When this happens the remaining thin layers may bulge and rupture in consequence of the intra-ocular pressure. If the content of the anterior chamber is evacuated the iris is dragged into

Fig. 184.—Multiple spina ventosa.

the wound, which may lead to permanent deformity and to secondary disease of the eye.

Even though the disease of the cornea is less deep, it usually leaves on healing some cloudiness which persists throughout life. Only very superficial phlyctenulæ heal without scars. The individual attack is of brief duration but the tendency to recurrence is great. Repeatedly small ulcers appear, or a single ulcer grows to a more and more central position and is marked by increased vascularity. Finally a more diffuse form of disease, the scrofulous pannus of the cornea, may appear, affecting chiefly the lower half of the cornea and persisting for a long time.

The corneal affection is always accompanied by a greatly increased secretion of tears and by a catarrhal inflammation of the connective tissue. This involves hyperplasia and in the course of time, if the condition persists,

leads to a thickening of the lids, eczema about the inner canthus, ectropion and irregular growth of the ciliary muscle.

Intense photophobia is very characteristic of scrofulous inflammation of the eyes and may at times enable one to make the diagnosis at a distance. The affected child closes his eyes, hides his head from the light and objects strenuously to examination (Fig. 185).

The disease as it appears in the nose, with the chronic coryza, the swelling of the nostrils, and the eczema of the surrounding skin gives a

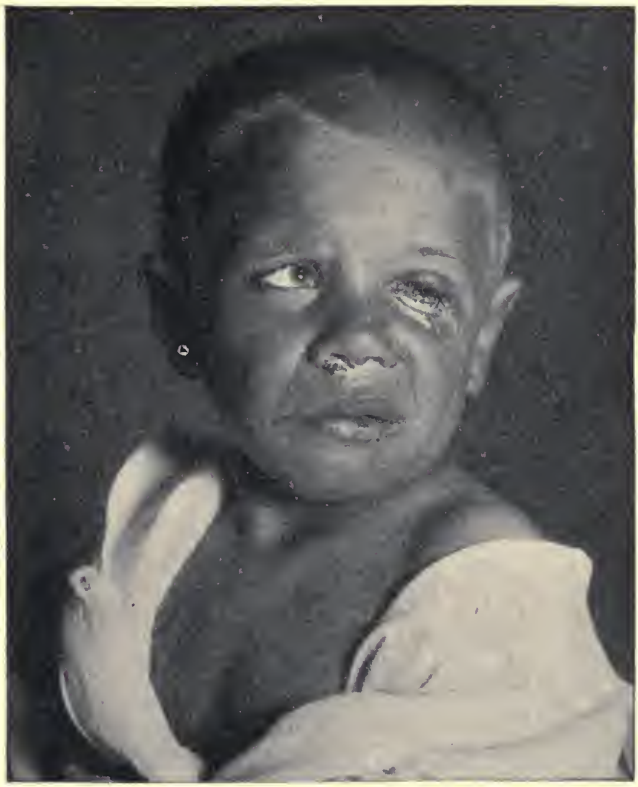


Fig. 185.—Scrofula. Chronic conjunctivitis and rhinitis, with thick upper lip. (Gisela Children's Hospital, Munich, Prof. Ibrahim.)

characteristic picture (Fig. 183). This has been described under Diseases of the Respiratory Tract; so, also, has the chronic catarrhal inflammation of the middle ear, which may lead on to destruction of the internal ear if it is combined with caries of the petrous portion of the temporal bone.

The upper lip usually shares in the swelling, affecting the nose. The mucous membrane of the lips become fissured and is covered with scabs. In other parts of the face, lichen, or a measles-like eruption, or pustules appear, which rarely extend to the skin of other portions of the body. These eruptions may, indeed, take various forms and it is hard to say how

far they may be regarded as of tuberculous origin. Such causal relation certainly obtains for the verrucous and papulosquamous tuberculides described in the chapter on Diseases of the Skin.

The manifestations of scrofula, though terrifying in appearance, are hardly ever dangerous. The tuberculous changes which terminate fatally are not those of the skin or of the mucous membranes or of the bones of the hands and feet. Of course, the rupture of any focus into the blood-stream from any of these sources may result, as it does in simple tuberculosis of the bronchial lymph nodes, and may terminate in miliary tuberculosis or meningitis. The probability of this event is hardly greater, however, than in patients in whom tuberculous formations do not appear on the surface.

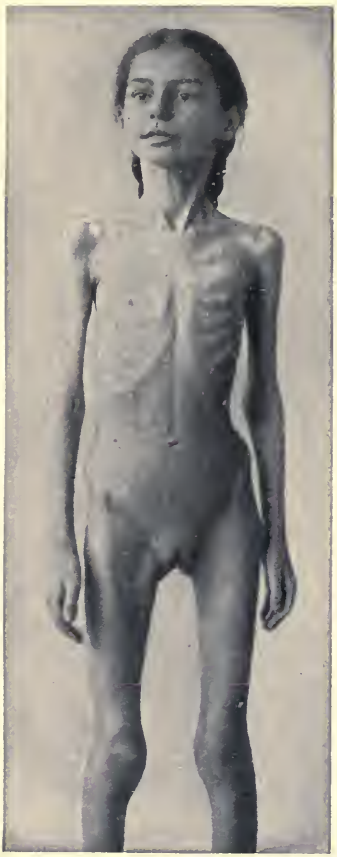


Fig. 186.—Habitus phthisicus ten-year-old girl. (University Children's Hospital, Zurich, Prof. Feer.)

With the end of childhood the symptoms of true scrofula usually disappear. They have one great disadvantage, as compared with the internal tuberculous processes, in that they very often leave such serious visible deformities, as the scars of healed lymph nodes or bone disease, or the cloudiness of the cornea.

Physicians formerly distinguished between a cachectic and an erethismic habitus in scrofula. The former included what today we term scrofula; the latter is covered under the prevailing term "habitus phthisicus" (Fig. 186). In childhood, the latter consists essentially of emaciation, since the narrow chestedness, so typical after puberty, is hardly distinct in earlier life; of a poorly nourished, dry, scaly skin, with excessive growth of hair. The face is of comparatively healthy appearance and though the cheeks are sometimes flushed or hectic, they are not markedly sunken. The cachectic habitus is very often seen in cases of visceral tuberculosis and especially, in tuberculosis of the serous membranes and of the lungs.

The Serous Membranes.—Nearly all of the surfaces may be affected by the tubercle bacillus, producing an inflammation which results in the formation of a thin serous, or fibrino-caseous exudate. Tuberculous affections of the pleura, the peritoneum, the pericardium, and the joint cavities and synovial sheaths belong in this class. The most important of these are tuberculous peritonitis (page 343), and adhesive pericarditis (page 407).

Tuberculous infection of the bones often produces serious disease-pic-

tures. This is especially true of disease of the vertebral column and of the larger joints. Infection in these parts or foci in the ribs may cause cold abscesses which descend into the pelvis.

Foci may be found in any other bones, in the testes and epididymis, and in the female generative organs. Finally, tubercles may also form in the brain and particularly in the cerebellum. They constitute the most frequent of the brain tumors of childhood (page 497).

CHRONIC PULMONARY TUBERCULOSIS; THE TERTIARY STAGE

As an uncomplicated disease, tuberculosis of the lungs becomes more and more frequent as we approach the end of childhood. This is especially true of the affection of the apices so typical in adults.

From autopsy findings and animal experiments we may conclude that chronic pulmonary tuberculosis arises from the primary focus as a tertiary stage. Clinically its relationship to the primary and secondary stages can be shown in very few cases. But the hereditary taint which, according to our present day knowledge, means nothing more than an early infection from a phthisical parent, is very frequently recognized.

The occasion for the onset of pulmonary disease may sometimes be found in the occurrence of an infectious disease and, in particular, of measles, pertussis, broncho-pneumonia. In many cases no recognizable cause is determinable.

The first symptoms of pulmonary tuberculosis are very indefinite. An arrest of gain, or a slight loss in weight, lassitude, pallor in the morning and a hectic flush at night are generally noted. Careful observation will show variations of temperature with an evening rise. Cough is not constant and expectoration occurs only after bronchial symptoms of some duration. Hemoptysis, often the first sign of phthisis in the adult, is very uncommon in children. If bloody sputum is found we must assure ourselves that it does not come from the nose, gums or throat. After several months, or even after two or three winters, marked by suggestive symptoms, but followed by remission, the fever becomes more intense, the cough more severe and especially annoying in the mornings. Often after such a period only do the physical signs become clear.

Tympany or diminished resonance is found over the apices, with sighing or bronchial breathing, usually accompanied by râles. Often the lower lobes are also affected where cavities are more easily demonstrated, especially after a fit of coughing.

At this stage the sputum is no longer swallowed and may be examined for bacilli. As a matter of self-infection from the sputum, intestinal tuberculosis, manifested by severe diarrhœa, or tuberculous laryngitis, suggested by hoarseness, may result. As in the adult, death ensues, after a high degree of emaciation, either from the tuberculosis itself or from its complications.

The tendency to recovery, however, is decidedly greater in childhood than in maturity. With proper treatment even very advanced cases may be cured.

Diagnosis.—Tuberculosis may be recognized in either one of three ways: 1. By the clinical demonstration of a typical form of the disease. 2. By the demonstration of the tubercle bacilli. 3. By the demonstration of specific antibodies (ergines), with the tuberculin reaction.

1. The forms of disease which definitely indicate tuberculous infection are, primarily, certain affections of the bones and joints, as spondylitis, fungus of the joints and spina ventosa. Lupus and the various types of skin tuberculides are rare in childhood, but are also pathognomonic.

The diagnosis is quite firmly established by the clinical symptoms of chronic exudative peritonitis, by the demonstration of cavity formation in the upper lobe of the lungs, or by the typical disease-picture of tuberculous meningitis.

The presence of serous pleuritis or the development of an expiratory whoop in the infant is less significant. In all these cases, a final diagnosis can be made only by the one or the other of these proofs, corroborative of the clinical examination.

Of the tuberculous infection of the lymph nodes very much the same thing may be said. Most of the cases of chronic hardening of the lymph nodes are undoubtedly due to tuberculosis. We cannot diagnose the disease, however, merely upon the fact that numerous small lymph nodes are palpable. Such a polyadenitis may be of non-tuberculous origin. To suggest a diagnosis of tuberculosis the gland must be of at least the size of a cherry and the swelling must have persisted for some length of time. Particularly in the case of the cervical glands one should never be premature in diagnosis, since other infections arising in the mouth may cause subacute swelling in this chain. Lymph nodes which are adherent to the skin, and especially those in which an irregularly contracted scar indicates early rupture, are usually tuberculous. Equally suggestive is the adhesion of scar tissue between skin and bone.

Pulmonary manifestations in the child require special consideration before a diagnosis is matured. Apical catarrhs are not so characteristic as in the adult and simple dulness or bronchial râles over the lower lobes may be due at any time to a chronic pneumonia of other origin.

A high degree of cachexia in a child, of from three to fourteen years, always suggests tuberculosis as a matter of priority, just as it suggests a chronic gastro-intestinal affection during infancy. Nevertheless, we should hesitate to make a diagnosis from this fact alone or from a dry, hairy condition of the skin. The hairiness may be hereditary or it may be the result of the cachexia itself, rather than a specific consequence of tuberculosis.

Roentgenography renders excellent service in the examination of the lungs. The younger the child, the more pronounced are the findings. Extensive tuberculous infiltration appears in the form of dark shadows in which cavities may often be recognized as lighter areas. Small areas of infiltration, however, especially at the apices, are not always clear. In such a case more definite information may be gained by percussion and auscultation. Small caseated or primary calcified foci often stand out very distinctly.

The X-ray of the lung in miliary tuberculosis presents a fine mottled appearance, which is more distinct as the individual tubercles become older. Pleurisy produces deep shadows, and a fibrinous pleuritic exudate may be very distinct long after the process has healed and is especially noticeable between the lobes of the lung.

The question of the recognition of enlarged bronchial nodes is the subject of much discussion. Such large masses of nodes as are seen in Fig. 180 are easily discovered, but numerous errors have been made in the diagnosis of less markedly enlarged glands. The distinction of the shadows caused by these from the normal picture of the pulmonary vessels requires much practice, which may be acquired only with the control of tuberculin reactions and autopsy findings. Moreover, it must not be forgotten that other than tuberculous processes, and particularly in pneumonia and pertussis, may lead to the swelling of the bronchial lymph nodes.

In this clinic the Roentgen examination of every child, suspected of tuberculosis, is insisted upon. In older children a fluoroscopic examination is often sufficient, but in younger ones it is better to take a picture at once. This permits, of course, only momentary exposures, since plates given more than a second are never clear.

2. The Demonstration of the Tubercle Bacillus.—While this demonstration is the most signal requirement of the diagnosis of tuberculosis in the adult we are but rarely given the opportunity of its achievement in children. This is due in part to the fact that among them open pulmonary tuberculosis is not common, and further to the fact that even in the open case the sputum is not expectorated, but is habitually swallowed. In the chronic phthisis of older children an exception to this rule is noted.

In the very young, the bacilli may sometime be obtained by passing an applicator wound with cotton into the pharynx, when the gagging induced may bring up sputum. It may be secured also by washing out the empty stomach. If these methods do not succeed the bacilli may be found in the stools.

Extirpated tonsils may be examined in stained section. Suspected material in which there may be very few tubercle bacilli, such as exudates, urinary sediments or spinal fluid, is examined by the antiformin method, when, if the findings are negative, the centrifugalized material may be injected into a guinea pig.

3. The Tuberculin Reaction.—In children the tuberculin test plays a much more important rôle than the search for bacilli. In very young subjects it should always be employed when there is the least suspicion of tuberculosis. In older children it should be employed only when some point of practical consequence depends upon its positive or negative outcome. It must not be forgotten, however, that the tuberculin reaction is merely a part of the examination. Its significance is valuable only when due consideration is given to the clinical symptoms.

Tuberculin causes a specific inflammation in the tuberculous, while it is entirely without action in the non-infected. As already noted, upon infection with tubercle bacilli, ergines, or substances in the nature of antibodies

are formed which, in the presence of bacilli or tuberculin, form apotoxins, the toxic products of digestion. These antibodies are distributed throughout the human organism and it is possible, therefore, to produce reactions in any part of the body by introducing tuberculin.

Wherever tuberculin and these antibodies come together apotoxin is formed. The reaction occurs first as a local manifestation at the point of entry; and later, as a focal reaction, at those points where tuberculous deposits and probably, therefore, a large number of antibodies are found. The reaction may develop in the original tuberculous focus, or at any place where tubercle bacilli are gathered, or may even reappear at some point where a former tuberculin injection had been given. Further, a

Cutaneous tuberculin reaction.

FIG. 187.—Applying tuberculin.

FIG. 188.—Vaccination.

general reaction is manifested by fever and malaise, which is accounted for either by the formation of apotoxins in the central viscera or by their absorption from tuberculous foci.

Focal and general reactions occur only when a large quantity of tuberculin gets into the circulation. In the application of tuberculin to the skin or the mucous membranes a general reaction is exceptional, since *so small* an amount of tuberculin is absorbed. It may be avoided in subcutaneous injections by the use of minimal doses.

The recognized methods for the introduction of tuberculin are the subcutaneous or intracutaneous injection, cutaneous vaccination, percutaneous inunction, and conjunctival instillation.

As the first test, the cutaneous vaccination is preferred. If this is refused by the patient, inunction is employed. The intradermal test serves as a further check in the case of negative results, and the subcutaneous method serves to develop focal reactions.

The technic of cutaneous vaccination is as follows: The skin of the forearm is cleansed with ether. A drop of Koch's old tuberculin is placed with a pipette or glass rod at each of two points upon the skin, about 10 cm. (4 inches), apart. A vaccinating borer, the platinum tip of which has been sterilized by heating, is twisted at a point halfway between the drops, thus giving a control. The same proceeding is had upon the skin through each drop of tuberculin.

A positive reaction appears in a few hours, at the earliest, and usually within twenty-four hours. The red areas which appear immediately after the vaccination are merely traumatic and may be seen equally at the control point. The specific reaction consists of a raised, red, indurated papule of from 5-25 mm. in diameter.

If the papule does not reach a diameter of 5 mm. it is not to be considered as definitely positive, even though the vaccination points appear larger than the control. Tuberculin may produce such a slight local reaction even in non-tuberculous persons; the test should be repeated in such cases. If the first slight reaction is positive, upon repetition it will be more definite.

Successive increase of the positive reaction is due to the fact that the inoculation of even so small an amount of tuberculin in the tuberculous individual has an influence on the formation of antibodies which stimulates the organism to the increased formation of ergines. A torpid or secondary reaction depends upon this. As previously stated, an early reaction commonly appears within twenty-four hours; more rarely it is postponed to forty-eight hours. There are occasional cases of torpid reaction, in which the papule is seen only after two, three or even eight days. These instances, however, are almost always in individuals who are clinically free from apparent tuberculosis, or in those who show healed tuberculous lesions. This applies also to the secondary reaction. In older children and in adults, it frequently happens that there is no reaction upon the first attempt, but that a secondary response appears upon the repetition of the test in eight days. Such persons are not necessarily irresponsive to tuberculin upon the first occasion, but they are less sensitive than usual. If a subcutaneous or intracutaneous injection is given with a relatively high dose of tuberculin they prove capable of reaction (F. Hamburger). Yet, they stand these high doses without general reaction, because their sensitivity is slight.

The following method of procedure is recommended for complete examination: The cutaneous test, already described is carried out first and the points of inoculation are examined after twenty-four hours. If the reaction is present its extent is noted and inspected again after a second twenty-four hours.

If the reaction is negative and, even though the tuberculin inoculation points, corresponding to the wounds made with the instrument, are distinct, we should wish to determine definitely that there is no power of reaction, one milligram ($\frac{1}{10}$ c.c.), of a 1 per cent. dilution of old tuberculin may be injected into the skin, as superficially as possible, with a fine needle (Mantaux or Stich reaction). The dilution is best prepared in a Fornier syringe, taking up 0.1 c.c. of tuberculin first and then 0.9 c.c. of sterile water. This is to be

mixed by thorough shaking, after which the excess of 0.9 c.c. is thrown away and the syringe is again refilled to 1 c.c. with sterile water. Instead, the dilution may be made by placing 5 c.c. of sterile water in a watch-glass, and adding one drop (0.05 c.c.) of old tuberculin and mixing it by drawing the

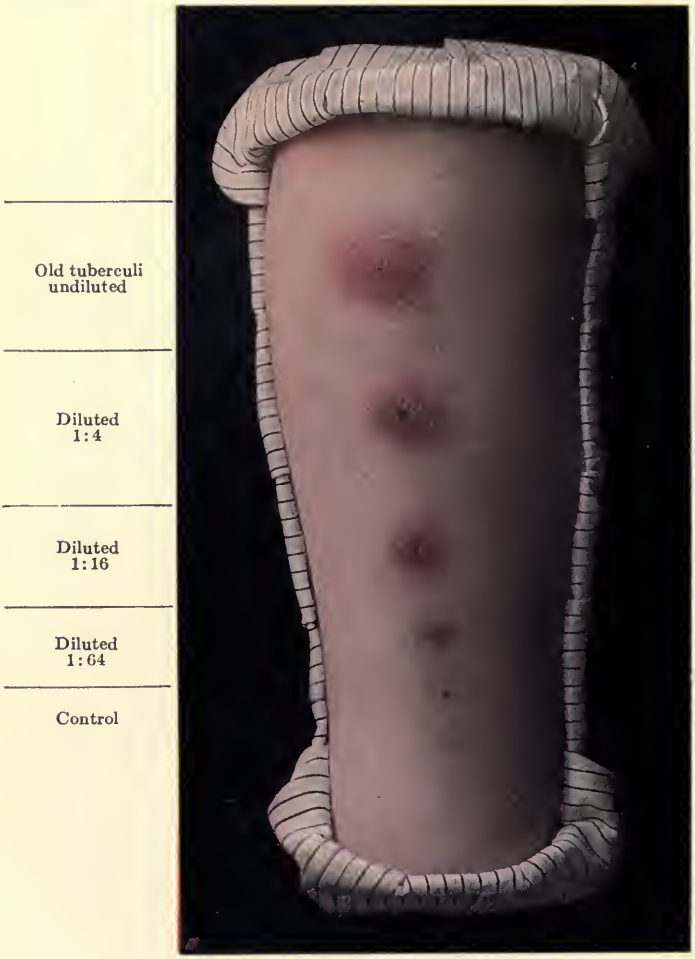


Fig. 189.—Cutaneous reaction with various dilutions of tuberculin.

fluid into the syringe several times. The doses need not be absolutely exact.

A positive reaction results in a reddened area of painful infiltration which remains sensitive several days. Intense reactions are easily interpreted, but slight ones are often doubtful. With the subcutaneous test a clear result is obtainable in most cases, if not in all.

If one does not wish to use the subcutaneous injection on account of the ever present possibility of a rise in temperature, the cutaneous test may be repeated after a week's delay. Usually an increased power of reaction,

has been established by that time, if allergy has ever been created. If the injection, or the second application of the cutaneous test, proves negative, the existence of tuberculosis may be excluded quite definitely; taking into due consideration, however, the few cases in which the power of reaction is diminished.

If the patient refuses vaccination, the Moro percutaneous test may be applied. This test depends upon the premise that sufficient tuberculin may be rubbed into the skin by thorough inunction to produce a reaction. For this purpose either undiluted old tuberculin or Moro's tuberculin ointment, prepared with equal parts of Koch's tuberculin and lanolin is used. A portion of the ointment, the size of a pea is rubbed into the skin of the back or abdomen over an area about five centimeters in diameter.

A positive reaction requires the same time for its development as in cutaneous vaccination. It consists of small lichen-like nodules. If there is

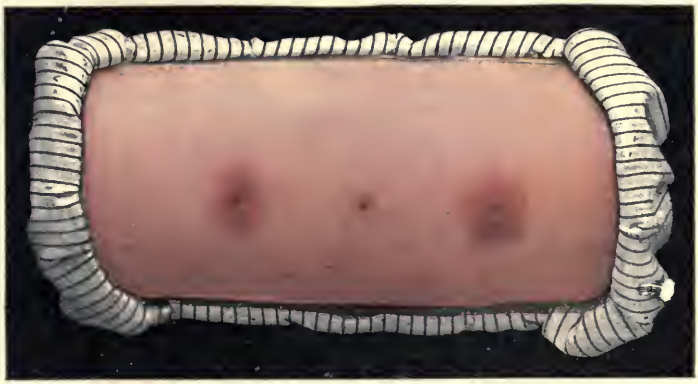


FIG. 190.—Severe cutaneous reaction with formation of areola. Forty-eight hours after application of the test. Two areas of vaccination, control in centre.

marked sensitivity, the nodules are very dense and the surrounding skin is reddened. If thoroughly and carefully applied, the test is almost as delicate as the vaccination, but is subject to many sources of error.

The instillation of a 1 per cent. dilution of tuberculin into the conjunctiva (Calmette), causes conjunctivitis in tuberculous individuals. This test cannot be recommended for children, because it may be followed by a long continued inflammation of the eye.

On account of the danger of very serious fever resulting from the original method of injection, as devised by Koch, it is no longer used in children, unless their sensitivity has been previously determined by local tests, and is then given only by gradual stages with intervals of days between doses (Loewenstein and Rappaport). The procedure is much more tedious than the methods described. A further difficulty of its use lies in the fact that this temperature test can be employed only in patients who are free from fever. The injection is indicated, nevertheless, if one wishes to obtain a focal reaction in such cases as suspected tuberculosis of the bladder. The injection should be preceded by the cutaneous test. If this is negative, one

milligram of tuberculin may be injected at the outset. If the cutaneous test is positive, the injection should begin with $\frac{1}{100}$ mg., and this should be increased to $\frac{1}{10}$ mg. and finally to 1 mg., if no focal or general reaction is shown.

What Does the Positive Tuberculin Reaction Signify?—It is still frequently interpreted as signifying tuberculous disease. This is incorrect. A positive reaction signifies merely that the individual has formed antibodies against tuberculosis, that he has at some time been infected by tubercle bacilli. In fact, the infection need not have caused actual disease, but may have been confined to a few unimportant lymph nodes.

The formation of antibodies in the organism is at its height during the years immediately following the infection, or during a recrudescence of the disease process, or after a reinfection. An intense tuberculin reaction to a first test indicates that a new factor has developed in the course of the

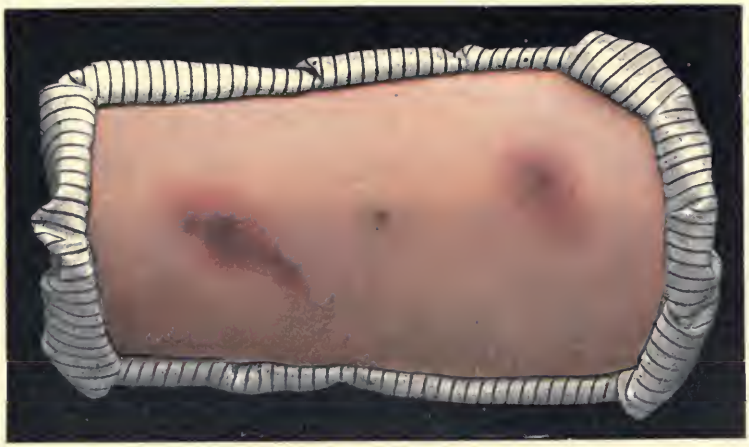


FIG. 191.—Moderate reaction. (Figs. 172-174 from wax models by Dr. Henning, Vienna.)

tuberculosis. It does not prove, however, that the disease is progressing; it may be receding.

Clinical examination, however, discovers foci suggestive of tuberculosis and if general symptoms, emaciation and the like, coexist, the reaction may be laid to tuberculosis with reasonable certainty. This probability is the greater the younger the child, since tuberculosis is rarely latent in young children.

The Significance of a Feeble Reaction.—Slight, torpid, or secondary reactions, and those which appear only after the subcutaneous injection of large doses of tuberculin have one and the same meaning. They show that the organism has been infected at one time, but is no longer at the height of its formation of antibodies. This condition is chiefly characteristic of healed processes, but may obtain in long-standing progressive tuberculosis. The pulmonary tuberculosis of adults often gives these weak reactions.

Furthermore, certain processes may cause anergy, or reduction or loss of sensitivity during an active tuberculosis. Among these are:

1. Miliary Tuberculosis.—The tuberculin reaction fails quite commonly in older children during the last week of miliary tuberculosis or tuberculous meningitis. It may be lost, also during recurrences of miliary dissemination which do not prove fatal.

2. Measles, as we have previously noted, always reduces to a minimum the sensibility to tuberculin. According to the reports of several authors lobar pneumonia has a similar influence.

3. Previous Treatment with Tuberculin.—While the introduction of minimal doses of tuberculin results, in the course of several days, in increasing the power of reaction which, on the contrary, is decreased by the injection of larger doses (Vallée, F. Hamburger). The immunity to tuberculin occurring upon rapid increase of dosage, as in the old method of Koch (Schlossmann), must be laid to the absorption of the antibodies (F. Hamburger), or to the development of anti-anaphylaxis (Bessau). Whether the loss of sensibility after very gradual increase of doses, as in Sahli's method, is to be explained in this way or to be regarded as the development of a true immunity to tuberculin is not quite clear.

What is the Significance of a Negative Reaction?.—If all three of these factors in determining a negative reaction can be ruled out, a single negative result indicates a definite poverty of antibodies and very probably the absence of any active disease process. In young children it may be accepted as evidence that there is no tuberculosis present. Two negative reactions are well-nigh conclusive.

An exception is to be noted in cases of very recent infection. It takes some time to develop the sensibility to tuberculin. Reaction to the subcutaneous injection appears earlier than to cutaneous inoculation. A quantitative reduction of the tuberculin, illustrated in Figure 189, is largely of theoretic interest. The more dilute the tuberculin which gives an initial positive reaction, the greater will be the papule which results at the point of vaccination when undiluted tuberculin is used; so that the papule serves as an approximate index to the intensity of the development of antibodies.

Prognosis.—Tuberculosis is not to be regarded as a disease, the course of which is determined by time or by degree of infection, as measles which runs its course in fourteen days, or leprosy which is slowly but surely fatal. The only constant elements in tuberculous infection are the formation of the primary lesion and the reaction of the regional lymph nodes. The development, the number and the extent of secondary lesions, and their implication of vital organs depend upon more or less accidental circumstances.

If the opportunity of making a diagnosis is given during the primary stage, when only an initial lesion of the skin, perhaps, or an expiratory dyspnœa, or a positive tuberculin reaction, without accompanying symptoms, is discoverable, the prognosis is governed by the age of the patient. During the first year all the probabilities point to a fatal termination or at least to the prospect that the child will develop distinct clinical symptoms. On the contrary, in a child of eight years or so, while the possibility of the spread of the disease cannot be gainsaid, it is altogether likely that no further symptoms will appear and that the secondary or tertiary stage will

not ensue. Any child who has a recent tuberculous focus may develop a disseminated tuberculosis at any time; but it is nevertheless true that the probability of a miliary dissemination decreases from year to year. Herbert Koch has estimated that in children during their first four years, the danger of tuberculous meningitis is one hundred and twenty times as great as it is in between the tenth and the fourteenth year. Since the tuberculin reaction has come to be of so freqent use among infants, a large number of cases has been found which have survived infection even at this dangerous age (Schick). Hahn has followed sixty-nine infants who had responded positively to the tuberculin reaction and has found that of those in their first year, 17 per cent. survived the disease; in the second year, 26 per cent.; while of those who gave a first positive reaction during the third year, 39 per cent. escaped.

Even in the second stage of tuberculosis, the prognosis depends entirely upon the extent of the disease. Thus, a widespread miliary affection which develops meningeal symptoms will be fatal. Isolated infections must be judged according to the vital importance of the organ involved. A tubercle the size of a walnut, situated in the bronchial nodes, may remain undiscovered or cause only a slight cough, while occurring in the brain it would prove fatal. A fungus of the knee-joint is annoying but is not dangerous to life, whereas the same lesion in the vertebral column leads to compression of the cord, paralysis, etc. An adhesive tuberculosis of a synovial sheath is not seriously disturbing; a similar adhesion in the pericardium gravely affects the heart action. A miliary tubercle which causes no symptoms at all in the lung, may lead to the loss of an eye if it occurs on the cornea.

The secondary stage in itself always has a rather favorable prognosis. Innumerable children, some of them marked for life with stiff joints, clouded cornea, kyphoses, or at the least with scars resulting from broken-down lymph nodes, still survive.

The tertiary stage of tuberculous infections and the pulmonary forms in general must be judged, in the main, by the extent of the process and the general condition of the patient. Small foci frequently spread if it is impossible to improve the nutritive status. The prognosis of pleurisy is good if it is not accompanied by extensive pulmonary spread. Laryngeal and intestinal tuberculosis must be considered serious for the reason that they are generally the result of severe pulmonary disease.

Prophylaxis.—The great essential factor in the prevention of tuberculosis is the separation from the child of all persons who expel tubercle bacilli. During the first year, the infant should be fairly well isolated; being protected from contact with strange children and adults as much as possible. Every chance of tuberculous infection, every casual meeting with a phthisical individual, is a menace to the infant. In certain families one child after another dies from tuberculous infection which may be traced to a grandparent, to a neighbor, or to a servant who does not suspect that the chronic cough which has troubled him from youth is infectious. The attention of parents should be invited to the fact that children should be shielded from intercourse with all persons who suffer with a cough.

Prophylaxis is far more difficult when the mother herself is a suspect. In advanced forms of tuberculosis in the mother, nursing is contraindicated for her own sake, and the infant should be removed from the house. With a slight infection the mother may nurse her babe, but she should be impressed with the fact that to caress the child or to cough in its near presence may prove fatal to it.

In well-to-do families the chances of the practical observance of such precautions are better than among the poor with whom tuberculosis is so common. The latter class of cases can be efficiently helped only by the exhaustive and systematic attention given to the phthisical in sanatoria and other similar institutions.

Treatment.—The primary stage of tuberculosis is hardly amenable to treatment. In the secondary stage there is greater opportunity to influence the general condition to the patient. In chronic cases and especially in the tertiary stage, the tendency of the organism to recovery may be markedly assisted by treatment.

Three methods may be adopted: 1. The reinforcement of metabolism by a general improvement in nutritive conditions; 2. The specific formation of bodies protective against the infection through the agency of tuberculin. 3. Failing the complete destruction of the infective organisms, the surgical removal of superficial foci within reach of the knife, or the functional resting of the lung by the development of a nitrogen pneumothorax. In the pursuit of these methods we must not forget, however, that tuberculosis is a constitutional disease and that we have at present no therapy of general sterilization.

With a view to the constitutional upbuilding of the patient, attention must be directed to an improvement of appetite and an increase of food-supply. The best stimulants to this end are light and air and a change of environment, dwelling and dietary. All varieties of climatic resort have been advised, in the course of years, for the tuberculous; the moist warmth of the Riviera and the dry heat of the desert; the moist cold of the northern seacoast and the dry cold of the Alps. The common factors of them all are the change of environment, the suggestion of recovery which directly stimulates the appetite, and the increased opportunity of life in the open air and in the sunlight. All this hardly requires any particular health resort. The same results can be obtained under efficient institutional care within the cities. In the home, however, it is hardly ever possible to so modify the life of the family as to enable the patient to live in the open as he can very easily at the seashore. The worst feature of treatment is confinement to bed in a closed room, resulting usually in a serious diminution of appetite.

It is not so much a question of keeping the patient in the open air for a certain number of hours a day, nor yet of any certain temperature of the air, nor of this or that form of therapy. The chief aim is to create an appetite and a joy in living, and the task in each individual case is to find the best means of doing this.

Does simple tuberculous enlargement of the bronchial lymph nodes require treatment if no secondary dissemination can be demonstrated? If

the child shows no appreciable symptoms, other than an occasional rise of temperature, does not lose in weight, is not anemic and has a good appetite, treatment is not considered necessary. (We believe it advisable to institute dietetic, as well as climatic and solar treatment in all cases of manifest tuberculous enlargement of the bronchial lymph nodes.) If, on the other hand, anemia and emaciation are evident, associated with a positive tuberculin reaction, rest and dietetic treatment are to be recommended, as follows: In the first week, absolute rest either in bed in a well ventilated room, or, preferably, in a lounge chair in the open air. In winter the patient should be placed upon a south veranda; during the summer a sunny garden will better serve. He may be allowed to play or read, but regular study should be discontinued.

In the second week, the patient is permitted to get up for a half-hour morning and afternoon. In the third week, the periods of activity are extended to an hour; and in the fourth week to two hours. While the child is up he is allowed to play and to run about, but short of actual tire.

After the first month, a modified form of treatment is continued for four to eight weeks. The child is kept out of school; he is given his breakfast in bed; takes an hour's rest in the afternoon; and retires at eight o'clock.

During the entire course of treatment, the appetite must be closely watched. It is well to give a bitter tonic at noon and at night. The following prescription has proved useful:

℞	Tincturæ ferri pomata	5.0 (ʒi)
	Tincturæ nucis vomicæ	1.0 (♏xii)
	Tincturæ cinchonæ composita	20.0 (ʒiv)

M. Sig.—Ten (10) drops in a teaspoonful of sweetened water, ten minutes before meals.

The child should be taught to eat slowly and to masticate his food thoroughly. Five meals a day should be given, at breakfast, forenoon, noon, afternoon and evening. Large quantities of milk, or of milk and cocoa, malt extract in amount proportioned to the action of the bowels, malt coffee, etc., are allowed. If eggs do not provoke indigestion, one or two a day may be used. If the appetite for solid food diminishes in consequence of the milk feeding, the latter must be reduced. If the child has any difficulty in going to sleep at night, a glass of milk may be given.

Care must be taken to regulate the bowel movements. If constipation ensues, especially during the first inactive weeks, the menu may be varied with malt extract, stewed fruit before breakfast, etc., or small doses of laxatives may be given to secure a bowel movement at least every forty-eight hours.

In the beginning the temperature should be taken four to eight times a day, until the form of the fever curve or the absence of fever has been satisfactorily determined. Later, it is enough to take the temperature once a day.

The patient should be weighed every week and compared with the average weight of children of the same height. For example, according to Cammerer, at ten years, the average height for boys is 130 centimeters

(52 inches), and the average weight 30 kilos (66 pounds). If the given boy is 130 centimeters tall, but weighs only 26 kilos (57 pounds), the conclusion is that he is four kilos or nine pounds, below normal and the attempt should be made to bring him up to the standard. During the first month of the rest-cure, the results are often very marked. Gains of a kilo (2.2 pounds), are frequently made, especially in restless, nervous children or in those who have been over ambitious in school. If no gain is made during the first four weeks, a mild tuberculin treatment is begun in the second month.

The treatment of manifest tuberculosis rests upon similar principles. If the nutrition demands it, the course of rest and dietetic management is is undertaken. After a certain period of treatment it is better to permit the patient to be about again, even if the results are not all that could be desired. The child does not need to be confined to bed continuously on account of a slight evening rise of temprature and he should lie down only when he feels so inclined. He should sleep late; eat a hearty breakfast, preferably in bed, and should be kept out of doors as much as possible during the day. If he moves about actively he does not need to be heavily clothed and in the summer the clothing should be as light as possible. If the child sleeps out of doors his covering should be adapted to the season and in extremely cold weather hot water bottles may be added.

If circumstances will not permit a general fresh air and dietetic therapy, as a last resort, prepared foods, remedies to stimulate the appetite, iron, a dilute solution of arsenic, or creosote (five drops in sweetened milk twice a day), may be employed. A favorite prescription follows:—

℞ Creosoti.....0.5
Oleii Morrhuæ.....100.0
M. Sig.—One teaspoonful, twice a day.

We saw the Rollier treatment carried out at the Kinderspital under Dr. Feer and believe it should constitute, if possible, part of the treatment of tuberculosis of the glands, joints and bronchial glands. The general tonic effect is so marked upon these children that recovery is more speedy and permanent.

In unilateral pulmonary infection, Forlanini's method of nitrogen insufflation is now frequently used. The thoracic wall is punctured with a trocar about 15 cm. long, at a point where there are no adhesions. A nickel tube, about 2 cm. in diameter, with lateral openings at the rounded tip, is passed through the trocar. This tube is attached to a three-way stopcock which leads through rubber tubing to a manometer and to a nitrogen reservoir. The manometer reading is first taken to show by the negative pressure that the point is actually in the pleural cavity. The stopcock is then turned to admit the nitrogen. At the first sitting no more nitrogen should be passed into the cavity than will equalize the pressure, this will require, according to the age of the patient, from 300-600 c.c. Physical and fluoroscopic examination will show that the lung begins to expand again after a few days. When this occurs, nitrogen is again given and is repeated prob-

ably every three or four weeks. This treatment requires several months. It should be discontinued when the expanding lung shows improvement.

After the use of nitrogen, the temperature usually falls. Hemoptysis ceases, the expectoration is diminished, and the appetite improves although there is no great gain in weight during the treatment.

Even young children stand the pneumothorax well. In these cases, the operation should be performed under general anesthesia in order to avoid the errors in manometer reading which may result from the struggling of the child. The operation may be complicated by the development of emphysema of the skin or serous pleuritis. There is no danger of air emboli if the manometer is carefully watched.

In severe phthisis, in brain tuberculosis, or in painful intestinal tuberculosis, hypnotics and narcotics should be given freely. Morphin may be used by mouth or hypodermically.

There remains the tuberculin method of treatment. As in the matter of general therapy, it is of chief use in chronic tuberculosis and not in the acute pulmonary affections of young children, nor in the intense manifestations of scrofulous disease.

The method of tuberculin treatment is as follows:

Three dilutions of 1:10,000; 1:100,000 and 1:1,000,000 of old tuberculin are prepared in fresh sterile physiologic salt solution. The treatment is begun with one millionth of a gram (one micromilligram). One cubic centimeter of the 1: 1,000,000 solution is drawn into a 10 c.c. syringe, and to this is added 9 c.c. of sterile physiologic salt solution. This preparation is injected subcutaneously in the back. The first injection should be given in the upper region and each succeeding injection a few centimeters lower down. The dilution of tuberculin with a large quantity of physiologic salt solution (10-20 c.c.), is to be preferred to the use of more concentrated solutions, since the former causes less local reaction (Herbert Koch).

After an interval of three days, the next stronger dilution is given and after four days more the third dilution, and so on. Two injections should be given each week. The dose is increased in geometric progression, as 10, 15, 22, 32, 47, 68, 100, unless fever ensues. In that event the identical dose should be repeated. If this dose again causes fever, the next dose should be reduced. If fever continues to appear the treatment should be stopped.

In the majority of instances, however, the gradual increase is borne without fever. In three weeks time, a dose of ten micromilligrams is reached; in six weeks a dose of one hundred; and in nine weeks of one milligram. Thereafter, six more injections of one milligram each are given, after which treatment, having continued for three months, may be considered complete.

The scrofula of the poor is most favorably influenced if the child can be removed from the home environment for several months and placed in healthy surroundings with the opportunity of proper care and a good dietary. If, with these conditions, open air treatment at the seashore or in the mountains can be combined, the results are more favorable. Benefit may be secured, however, in other sunny places which are free from dust

and smoke. Ocean baths, sun-baths or bathing in iodine-containing spring water (Halle), are especially efficacious in scrofulous cases.

In the home, improvised baths may be given once or twice a week. Two kilos (5 pounds), of rock salt are dissolved in fifty litres (12 gallons), of warm water. The child should remain in this bath for ten minutes, the water being kept at 26° C. (97° F.). The patient should be kept in bed for two hours after the bath or it may be given instead at bedtime. Inunction with soft soap (*sapo viridis*), three times a week may be employed as a substitute for the sun-bath treatment. Twenty grains (⅔ ounce) of the soap may be rubbed over the back and abdomen. It is left in place for ten to thirty minutes and is then carefully washed off.

Medically, cod-liver oil, with 1 per cent. of creosote, or large quantities of iodine- and iron-containing water may be recommended. Neither iodine, iron or arsenic have any specific action upon tuberculosis. Their favorable influence is to be attributed to their effect upon the metabolism.

The tuberculin treatment has not given any real results in scrofulous affections. The field of this method lies in chronic tuberculosis, in which there is but small power of reaction, as in infection of the lung apices or in tuberculosis of the larger bones. It is doubtful that any particular dietary can induce symptoms of scrofula in the tuberculous. That children of the poor are more commonly affected seems to indicate that it is not so much a question of wrong feeding or even of underfeeding, as it is one of poor hygienic surroundings.

In local tuberculosis, surgical interference is indicated symptomatically; but it should be undertaken only after we are assured that there is no progress toward recovery. Surgeons are becoming more conservative year by year in their treatment of tuberculosis. Special warning should be given against the total excision of indurated lymph nodes, of fungus joints, or of fingers with spina ventosa, etc. The long expectation of spontaneous recovery doubtless requires great patience, but large experience is necessary to enable one to predict that it will take place without interference.

X.

SYPHILIS

BY

ERNST MORO,

Heidelberg.

REVISED AND EDITED BY

PHILIP C. JEANS, M. D.,

Associate Professor of Pediatrics,
Washington University School of Medicine, St. Louis.

Etiology and Nature

Syphilis is caused by the spirochæta pallida, discovered by Schaudinn in 1905. Spirochætes are considered by some as allied to bacteria, by others as of protozoan nature, and by still others as differing from both bacteria and protozoa and classed in the domain of the protista. The situation is still much confused. Because of the confusion in classification, there is an uncertainty as to the proper name by which to designate the organism. Some excellent systematists prefer the name *triponema pallidum* rather than *spirochæta pallida*. As it stands at present, these two names may be used interchangeably. (*Vid.*, Noguchi, *Am. Jour. Syphilis*, 1: 261, 1917.) The spirochæta pallida is classed as a protozoan and is an extremely delicate thread-like organism, with tapering ends and with length varying from four to fourteen micromillimeters. Its corkscrew-like convolutions are rigid, short and acute. In some specimens twenty or more such convolutions are seen, but usually they are less numerous—a difference which may be due to the tearing of the threads in preparation. Fresh pallidæ are actively motile, twisting in their longitudinal axis or moving forward or backward. Frequently two or more specimens are seen lying side by side. The Y-shaped figures they present, probably indicate the process of generation by division. The various types of systematically related spirochætæ may be distinguished by their greater thickness, their smaller number of convolutions, their blunter poles and their variable readiness of reaction to the analin stains.

In the smear the spirochætæ are most readily and certainly demonstrated by Burri's method, as follows: A small quantity of the material to be examined, preferably in the form of serum taken from a lesion, is placed upon a slide and one or two loopfuls of ordinary liquid India ink, from which the larger particles have been removed by centrifuging for half an hour, are added. The black drop is then spread in a thin layer, as in the making of blood spreads, with the edge of a second slide. The preparation is dried in

the air and examined under the oil-immersion lens. The spirochæta and other corpuscular elements remain unstained and stand out prominently upon the black background. The organism has recently been cultured, (Sowadi Noguchi), but the process is not of practical use. Recently the method of dark field illumination has almost entirely superseded the older methods of demonstrating the spirochætæ.

The human being responds to infection with the spirochæta pallida by the formation of distinct antibodies. Very little is known concerning the nature of these antibodies, but it seems fairly well established that the clinical reaction of the human subject to the organism stands in very close relationship to the activity of the formed antibodies. This is the only way in which the long and fairly constant incubation period which elapses between the date of infection and the appearance of the primary lesion can be explained. It also affords a satisfactory explanation of the change which the once infected organism undergoes and which persists for the rest of life. This change is evidenced in a number of ways, but chiefly in the fact that a subsequent reinfection of the syphilitic patient with virulent material, is either altogether negative or causes a mild, abortive and more transitory reaction than it did the first time. This statement applies only to those who have had insufficient treatment. According to current opinion, syphilis may be cured in many instances and in such a case, a reinfection behaves as did the first one. In the test-tube only one substance representing these antibodies and reacting in this manner has been so far recognized. This circulates freely in the blood and is characterized by its marked affinity for lipoid substances, of human and animal origin, soluble in alcohol. As a consequence of this characteristic, it has come to be regarded as a specific reagent of great significance in the diagnosis of syphilis.

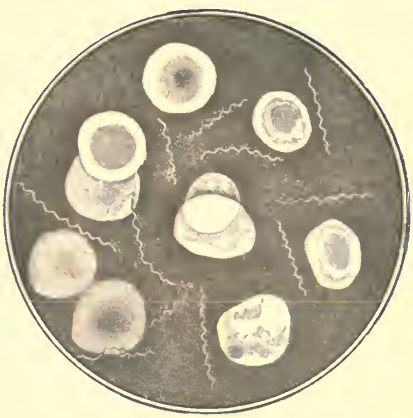


FIG. 192.—Smear of fluid from luetic papule (papulo-pustular syphilide in an infant). India ink stain by Burri method.

As in many other infections, the picture which the disease presents to us, is one of the defense of the organism against the antigens. As an actual fact, the defensive material, which the human subject can bring to bear against the attack of the spirochæta, is adequate to the complete destruction of the parasite in only very exceptional cases. Foci of infection may develop repeatedly and repeatedly stimulate the organism to the formation of new antibodies, so that an almost continuous reaction between the antigens and the antibodies is maintained and produces the picture of a chronic infectious disease.

In this, as in many other respects, the course of syphilis resembles that of tuberculosis. In both diseases, the primary lesion develops at the point

of entry of the antigen after a latent period of long duration, wholly devoid of symptoms. In both cases, again, the lymph nodes in the neighborhood of the primary focus become diseased in a specific manner. Pathogenetically, therefore, the syphilitic bubo is analogous to the diseased bronchial node, which represents the regional result of tuberculosis in the lung tissue. Almost coincidently with the appearance of the initial focus, the change of the organism or allergy, reacting to the antigen, is completed alike in both diseases. The organism has become hypersensitive toward the specific infection. This hypersensibility gives a peculiar prominence to all the succeeding phases of the reaction and is responsible, therefore, to a large extent for the clinical character of the so-called secondary and tertiary manifestations of the disease. From this point of view, the typical exanthemata and the lesions of the mucous membranes in the secondary stage of syphilis, would correspond to the manifold integumental and catarrhal reactions of tuberculosis; while the gummata, characteristic of the tertiary stage with its distinct tendency to necrotic destruction, correspond to the rapidly progressing ulcer formation of so-called phthisis.

Emphasis must be put at once upon the fact that this comparison holds good for acquired syphilis alone. In congenital syphilis, with which this discussion is chiefly concerned, entirely different conditions often obtain.

In the first place, the primary lesion is lacking in the great majority of cases, since the initial infection of the fetus occurs, as will be seen later, through the blood. This circumstance is doubtless an important factor in infantile syphilis. To this is to be added the fact, that in the fetus and the young infant the formation of antibodies is very slow and hence there is no effective bar to the increase and the spread of the spirochæta. Very often the antigen antibody reactions alluded to do not occur and the defenseless organism remains passive until it is overrun and fairly strangled by the disease parasites. Nevertheless, relatively, the simile holds good, for in fetal tuberculosis, the tubercle bacillus is planted in an organism wholly unable to defend itself.

Modes of Infection

Infection with the spirochæta pallida takes place either before, during, or after birth. If it occurs during intra-uterine life or during parturition it is termed congenital syphilis; if at any other time, acquired syphilis. Infection before birth is by far the more frequent event and is the most important source of syphilis in children. The infection of the fetus with the spirochæta always comes from the mother and from the mother alone. She may be infected before, during, or after conception. The spirochætæ circulate in her blood and reach the placenta, where they cause specific changes; the organ becoming diseased and permeable to the parasites and the fetus being thereby infected. The possibility of paternal transmission without infection of the mother is still debated. Though the weight of evidence is in favor of the statement as here made, no absolute proof has yet been advanced.

The infection of the fetus may occur at any time during pregnancy. The

earlier the infection takes place the smaller are the chances that the fetus will live. If it be possible for a spermatozoön to fertilize a previously infected ovum, there would be no probability of its further development. Such an ovum must die. On this account, the so-called paternal syphilis, a term which implies that the spirochæta reaches the ovum through the spermatozoön or through the spermatic fluid, when the mother is uninfected, cannot be given serious consideration in the pathogenesis of the congenital form of the disease.

These views regarding the transmission of congenital syphilis through the placenta are simple and easy of acceptance. Nevertheless, the long recognized fact that mothers of syphilitic children very often show not the slightest trace of syphilis and remain, to all appearances, immune to the disease all their life long, is an extremely peculiar and remarkable one. No exceptions to the persisting immunity of such cases, demonstrated by Colles and Baumes, are recorded; and accordingly it has become a justly recognized rule and is known as Colles' law. It means that a mother who gives birth to a syphilitic child and shows no symptoms in herself of the disease, is and remains immune to syphilis. Gaucher records a case of an hereditary syphilitic infant infecting its mother. Such cases are so rare that a mistake in history or observation may be suspected in this one. (Gaucher: *Ann. des. mal. ven.* 11:1 Jan., 1916.

In the past a large number of theories have been advanced to explain Colles' law. The principal one among them proposed that immune substances from the fetus, infected from the spermatozoön, pass over to the mother and confer a lasting protection upon her, so that she remains healthy. Conceding that the fetus, unable to protect itself, is yet able to form so active an immune substance; and conceding further that these substances can then pass through the placenta and become the property of the mother, it still seems highly improbable that such a passive acquired immunity can be at all permanent. This doubt is very definitely justified by numerous examples of research in artificial immunity. An immunity conferred by the mother upon the child hardly ever lasts more than a few months and the converse is probably equally true. The possibility that syphilis antigens in the form of toxins are carried from the infected fetus to the healthy mother is, of course, not wholly deniable. Under such circumstances active immunity of unlimited duration might be established in the mother. Still not a vestige of proof has been brought forward to support such a theory.

The mother of the syphilitic child is immune to syphilis because she herself has been infected; an unanswerable postulate of the theory of maternal heredity and a strong support of its conclusions. Furthermore, a large percentage of reaction bodies has been found in the blood of mothers free from any clinical evidences of syphilis, but having syphilitic children. The maternal side of the placenta frequently harbors spirochætæ.

The so-called Profeta's law, which holds that the healthy child of a recently luetic mother is protected against syphilitic infection, that is, that the child is immune to syphilis, has little foundation. Very probably

such children are actually infected, but with luetic symptoms so slight as to be overlooked. A positive Wassermann reaction is obtained in all such children and is accepted as evidence that they have been infected.

Strange and inexplicable, indeed, is the fact that the syphilis of mothers of syphilitic children is so frequently devoid of symptoms. It is not surprising that the primary lesion or the bubo should be hidden. These may be situated in some part of the uterus and in the lymph nodes of that immediate region. But that such mothers should so rarely show secondary or tertiary manifestations and that they should so frequently enjoy the best of health throughout life is extraordinary indeed. There would seem to be no doubt that the special metabolic conditions so closely related to the development and bearing of a syphilitic child play some part in these results.

Infection during birth is very uncommon and particularly hard to prove. It can be accepted as probable only when the new-born child shows a typical primary lesion several weeks after birth. Several instances have been reported in which, within the first month, a typical initial lesion appeared at the base of the nose in a child being born with a face presentation of a recently syphilized mother who showed a fresh genital chancre. Even in these cases, of course, infection after birth cannot be positively excluded.

It may be that the loosening and compression and the partial separation of the placenta during labor may be held responsible for the passage of the spirochæta from the placenta to the fetal blood. Doubtless, the protecting epithelium of the chorion may become ruptured during this process and the blood of the mother and of the fetus may then communicate directly. Rietschel lays great stress upon this intra-partum mode of infection and claims that it explains the fact that children of syphilitic mothers are so frequently born without any clinical signs of the disease, these appearing only after some weeks. This latent period would then correspond to the specific incubation period of the disease.

Infection of healthy children after birth may occur in a number of ways. This phase is discussed upon page 785 under the Acquired Syphilis of Children.

I. CONGENITAL SYPHILIS

Congenital syphilis is usually described under two heads: fetal syphilis and infantile syphilis. Actually, there is no conclusive reason for the definite distinction of these two conditions, for excepting in the event of intra-partum infection, infantile syphilis presents nothing more than a direct continuation of the disease acquired during fetal life to postnatal life. As will be seen, this is especially true of syphilis of the internal organs. For this reason, Heubner very properly speaks of the "projection of fetal visceral syphilis into infancy." Moreover, the pathology of syphilis in the two phases of its development has such identical characters that a separate discussion seems out of place on didactic grounds. Perhaps the fact that, for obvious reasons, the study of fetal syphilis lies entirely with the pathologist, while the syphilis of infancy constitutes one of the most deeply studied, serious and stimulating subjects in clinical pediatrics, may have served as a reason for the division.

FETAL SYPHILIS

The spirochæta very evidently finds the fetal organism a rich soil. As already intimated, a reason for this is to be found in the inherent lack of fetal defense—in the absence of the natural agencies of protection in the tissues of the fetus. Thus it happens that in extreme cases all the fetal organs are literally choked with the parasites. The inevitable results are seen in the frequent death of the fetus in utero and the syphilitic abortion in which the fetus is cast off dead and macerated. This maceration of the fetus is highly characteristic of syphilis. Graefenberg found the spirochæta in 80 per cent. of all fetuses that were macerated when expelled. Syphilitic abortion is most common in recent and untreated lues; it occurs chiefly in the fourth and the seventh months of pregnancy, although it may be met with at other periods. If the mother's history shows a succession of habitual abortions, syphilis must always be suspected.

The structural changes are not very typical in the first few months, or even in the first half of pregnancy. Diffuse cell proliferation; active proliferative processes; increased volume and consistency of the cells; a very active development and differentiation of organs, now infected, can be demonstrated in normal fetuses at this period. The discovery of the spirochæta at this time is rare. In all probability the death of the embryo is directly caused by the usual disease of the placenta, which shows vascular changes, scar formation, and contraction of the villi. The attack upon the placenta leads to the innutrition of the fetus incident to the insufficient supply of food material from the maternal to the fetal vessels.

In the second half of pregnancy, on the contrary, the syphilitic changes in the fetal organs are commonly distinct. Two processes, indeed, stand out most prominently: 1. Diffuse cell infiltration; 2. Retardation of growth.

1. Diffuse cell infiltration does not develop to the extent seen in the syphilis of children in any other period. Macroscopically, the condition results in a marked increase in the mass and the consistency of the affected organs alone. This is seen chiefly in the liver and the spleen. The microscope, however, shows that this process is general and that almost all the viscera—the kidneys, lungs, pancreas, thymus, and the osseous system, as well as the liver and the spleen, are affected in practically the same manner.

The cellular increase has its origin in the interstitial connective tissue lying around the smallest blood-vessels. In advanced cases, the proliferative process may become so intense as to result in veritable masses of cells, recognized even macroscopically, which have been called miliary syphilomata. Large numbers of spirochætæ are found in the proliferated perivascular tissues. In a later stage the hypertrophied connective tissues show a distinct tendency to retraction.

2. The cell proliferation delays the visceral development. The far-reaching hyperplasia of the interstitial connective tissue stands in direct contrast to the marked hypoplasia of the parenchyma.

Small remnants of parenchyma, developed at an earlier period, are often found in the midst of the interstitial cell infiltration, as foci-like agglom-

erations of cylindrical or cubical cells, or as entire epithelial tubes, etc. In the lung, an intense desquamation of alveolar epithelium, undergoing fatty degeneration, will occur into the lumen of the alveoli, accompanying the proliferation of the underlying connective tissue. As a result, the cut surface has a peculiar homogeneous, whitish-yellow appearance, the so-called white pneumonia. Wholly similar processes may occur in the thymus, making this organ look as though it were filled with numerous cysts filled with a pus-like secretion. This general retardation of growth and development is the coincident cause of the almost invariably low body-weight and the small size of syphilitic premature infants as compared with normal children of the same period. This abnormally low weight is all the more remarkable since these children show a considerable increase in the mass of the internal organs.

Another notable fact is that the skin, the organ which later, with syphilitic infants, plays so important a rôle in the clinical history, usually remains unimpaired in fetal life. Now and then an infected child is born with a syphilitic eruption, the ominous papular pemphigus. Other skin manifestations always appear later. Hochsinger explains this peculiar difference of the intact skin and the notable visceral involvement during fetal life, as due to the relatively late glandular development of the skin and the early differentiation and rapid growth of the internal organs in utero. The syphilitic poison is supposed to possess a special affinity for highly vascularized tissues. It might be expected that the cutaneous surface, subject to suddenly increased irritation by the stimuli of extra-utrine life, would be the more disposed thereby to exthanthematous eruptions. Nevertheless, it is a fact that those tissues in which the most active growth obtains, as at the boundary lines of bone and cartilage, succumb in most characteristic manner, even during fetal life, to the disease. This seems to indicate the probable relationship of a formative stimulus, as suggested by Hochsinger, between the syphilitic process and the process of tissue differentiation.

The inflammation at the margin between bone and cartilage, a syphilitic osteochondritis, first accurately described by Wegner, is, with the exception of splenic enlargement, the most constant and the most easily demonstrable sign of congenital lues. It occurs chiefly at the ends of all the long bones and at the anterior extremities of the ribs.

Macroscopically it may be seen, in longitudinal section, that the osteochondral boundary, normally marked by a whitish line hardly a half millimeter in thickness, is widened. It may measure two millimeters or more. This line, moreover, has a yellowish tinge and is no longer of regular width, but presents a jagged outline, the toothpoints extending into the cartilage. In advanced stages, a peculiar grating is noticed as the knife passes through the tissue, feeling as though it were in contact with fine particles of lime or mortar. A small section of this hard substance may become loosened and fall out.

These impressions in the gross are fully supported and explained by the microscope. The entire process is confined to the zone of temporary calcification. The area is abnormally wide. The calcification, though irregular,

proceeds rapidly, but resorption is delayed. The transition to bone does not take place, since the osteoblasts lack the bone-forming elements and actually constitute a useless granulation tissue. The irregular border is due to the persistence of calcified remnants of cells, and the yellow color to a more or less characteristic granulation tissue, through which typical syphilitic changes in the form of small gummatous masses may be scattered. In consequence of these departures, the ordinary formation of anastomoses between the irregular newly-formed bone-platelets is lacking. This granulation tissue is entirely cut off from its blood supply and this occurs all the earlier if the syphilitic inflammation is very active. As a further result, the continuity of the scaffold of the bone-platelets is not only weakened, but may be completely destroyed, and the separation of the diaphysis from the epiphysis in the zone of calcification finally ensues.

Such extremes of visceral change as this cannot, of course, be found in every case of fetal syphilis. In case of difficulty in the matter of diagnosis, the problem may be solved oftentimes most readily by the histologic examination of the kidneys. Hecker found, in 90 per cent. of his cases, specific renal changes, especially in the form of small cell infiltration around the cortical vessels. Others claim, on the contrary, that this organ is the best preserved in macerated fetuses. In doubtful cases it is often possible to demonstrate spirochætæ. For their discovery the umbilical cord and the adrenals, seats of election, must be studied. Spirochætæ are said to be especially numerous near the fetal insertion of the former.

INFANTILE SYPHILIS

In the infant, congenital syphilis manifests itself either immediately after birth or within the first weeks of postnatal life. Not infrequently the disease becomes clinically apparent only within the second month. Of the clinical signs with which syphilitic infants are born, three are especially prominent and characteristic: 1. Coryza. 2. Pustular eruption on the hands and feet. 3. Enlargement of the spleen.

These three symptoms form a, by no means, constant triad. At times they occur coincidently. In a child with pemphigus, the splenic tumor is hardly ever absent, but each symptom is so significant in itself, that its presence at least demands a further inquiry. With congenital pemphigus on the hands and feet every diagnostic doubt is laid.

1. Coryza or syphilitic rhinitis is indicated, at first, like every other coryza, by embarrassed nasal respiration. The nostrils are narrowed and plugged. A peculiar snuffling, with the passage of air, results. This is often discernible at a distance. Naturally great difficulty results in getting such children to nurse.

Examination of the nose often reveals nothing more than marked swelling of the nasal mucous membrane, especially in the postnasal areas. At first there is little or no secretion and the whitish foamy mucus of ordinary coryza is never present. Later, as the process develops, a purulent, and, at times, a bloody secretion appears. Sometimes the coryza subsides after a

few weeks, but generally it is very obstinate and persists unvaryingly for a long period.

Of course this symptom is not present in all syphilitic infants. Many of them are entirely free from coryza. If it does occur, it is usually congenital or appears very soon after birth; at the very latest from the fourth to the eighth week. This congenital coryza is usually so noticeable that the mother never forgets it. If in the history appears a record of "snuffles during the first few weeks," it is a cardinal point in the recognition of lues.

The well-known nasal deformities, in the form of pug nose or saddle nose seen in syphilitic infants may be charged to this process. They are consequent upon a severe rhinitis, which may be completed in early fetal life, but leads to a retardation of intrinsic growth and of the development of the cartilaginous and bony framework of the nose (see Fig. 213).

2. Syphilitic pemphigus consists of vesicles, varying in size from that of a pea to that of a cherry, rising from an inflammatory base. Their content

Fig. 193.—Syphilitic pemphigus of the new-born on the soles of the feet.

is at first serous and slightly cloudy, but it soon becomes purulent. Often numerous spirochætæ may be demonstrated in the fluid. The localization of the eruption on the palms of the hands and the soles of the feet and on the plantar surfaces of the fingers and toes is highly characteristic. The syphilitic pemphigus is usually present at birth, but it may appear during the first week and more rarely during the second to the fourth week. At times it is found on other parts of the body. The eruption never retains its vesicular character for long. The vessels rapidly dry up; they crumble or burst, and the bleeding corium lies exposed, the site surrounded at its circumference by the shreds of the torn covering of the vesicle. Experience shows that it is a very bad sign if infants are born with the developed eruption; they invariably die, sooner or later.

3. Congenital enlargement of the spleen is always extremely suggestive of lues. It is an easily established sign of visceral syphilis which the newborn carries over from the fetal to the postnatal life. Of course, the

enlargement is not always clinically apparent; very often the organ is not palpable below the margin of the ribs.

Splenic tumor, however, is a very constant occurrence in infantile syphilis. Even though it be absent during the first few days it will surely develop, if but for a short period. The careful observer will hardly ever overlook it. True, the enlargement of the spleen is given third place in this recital because it is not as prominent a symptom as the other two, which as indicators par excellence immediately lead the physician in the right direction. The cases are very rare in which a hard, easily palpated spleen, discovered during the first three months, and that means before the period of florid rickets, signifies anything but syphilis or tuberculosis.

Syphilitic disease of the liver hardly ever causes any distinct manifestations in infancy. The liver of the new-born is frequently and normally large and therefore it is not always easy to draw any absolute line between its physiologic and pathologic conditions. In studying the status of the liver it is necessary to note the factor of consistency as well as that of size. If, on the other hand, the liver has been the seat of extended changes during fetal life, the infant may be born with a true cirrhosis. In this event, the liver is very hard, friable, and usually markedly enlarged; the abdomen is distended and tense and a distinctly outlined venous net-work is seen on the surface. The firm splenic tumor is then always present. Icterus and ascites may develop, but are usually absent. If, however, progressive retraction of the interstitial liver tissue causes obstruction to the flow of bile, a very severe icterus will, of course, ensue. This event may determine the entire disease-picture. If, following the physiologic jaundice of the new-born, a high grade of icterus persists, the child retaining the lemon-yellow color for weeks or months, with white acholic stools and dark brown urine, syphilis must always be suspected, provided that congenital malformations involving the liver can be ruled out. In such a case, strangely enough, other manifestations of lues may be lacking.

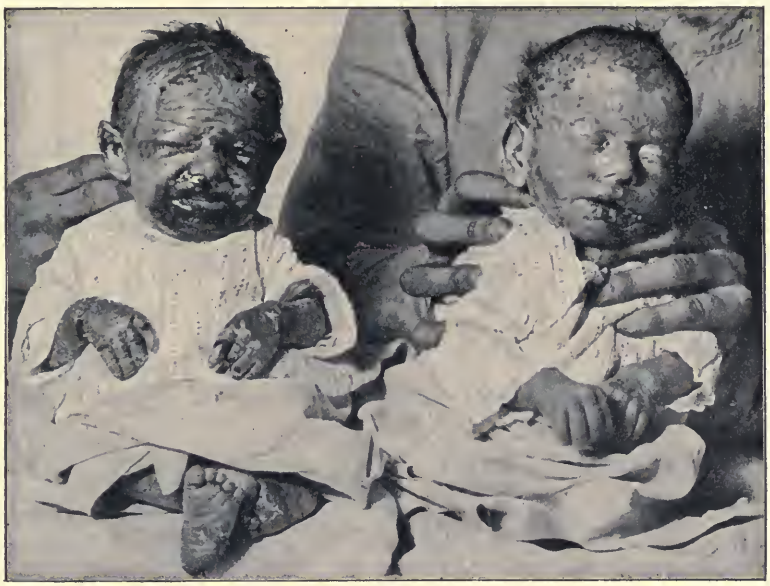
Protein and casts are frequently found in the urine of syphilitic children. These findings are to be regarded as evidences of specific injury to the kidneys, since pathologic renal changes have been clearly demonstrated microscopically, not alone in the fetus, but in the infant. Nevertheless, it is always to be remembered that albuminuria is a symptom of all possible sorts of disease among infants and is especially common in the course of disturbances of nutrition.

Osteochondritis, so extremely characteristic of fetal lues, has been discussed and will be considered even more fully in a later page. It should be said, only, that certain children are born with bone diseases in advanced stages. Sometimes the joints are so severely affected that they cannot be moved. The limb lies entirely motionless. If an arm is involved one may be led at first to suspect a birth paralysis, thus putting the obstetrician under suspicion and often causing great injury by the improper treatment of the child. A careful analysis of the local condition in relation to the general disease will readily put one on the right track.

Syphilitic disease of the nervous system and of the sense organs usually

does not appear until a later period of life. Though the disease does not appear clinically at this time, the infection of the nervous system which causes later symptoms is already present. Among the congenital affections of the eye, however, primary plastic iritis, with extensive posterior synechia, should be mentioned, since it represents a condition very pathognomonic of lues.

It is not surprising that syphilitic children are often, and indeed commonly, below normal weight even though born at term. Primarily, specific retardations of growth doubtless contribute to this result. In the second place, and probably of even greater importance, is the interference with the



R. *L.*

FIG. 194.—Congenitally syphilitic twins.

R. The diffuse infiltration of the skin is very distinct, especially on the hands, arms and feet. Crusty syphilide surrounding the mouth and on the chin. *L.* Circumscribed pustular syphilide in face, especially marked on forehead. Rhagades of the lips (Children's Hospital, Munich, Prof. Von Pfaundler).

normal nutritive interchange of the fetus with the placenta in consequence of the intra-uterine infection. With suitable care and systematic treatment such children recover more rapidly than might be expected, despite of their congenital debility.

In lues of severe grade, moreover, the conditions of intra-uterine existence are so poor that birth commonly occurs too early. What has been said of habitual abortion applies also to successive and repeated premature births. They are typical, in a degree, of syphilis and are almost always to be found in the history of lues.

All the symptoms described are not necessarily present at birth. The infant may be born apparently healthy and one or another sign may appear distinctly during postnatal life. Snuffles, pemphigus, the enlarge-

ment of the spleen or liver, albuminuria, or osteochondritis may make their appearance at any time, although seldom later than the close of the second month and usually during the first few weeks.

This is particularly true of the great group of exanthemata and other skin manifestations of hereditary and infantile syphilis. Of this group we have mentioned only pemphigus of the new-born because this is so frequently present at birth or makes its appearance during the first few days of life. The eruptions of the other exanthemata generally appear at a later period. Sometimes these eruptions develop gradually in the course of

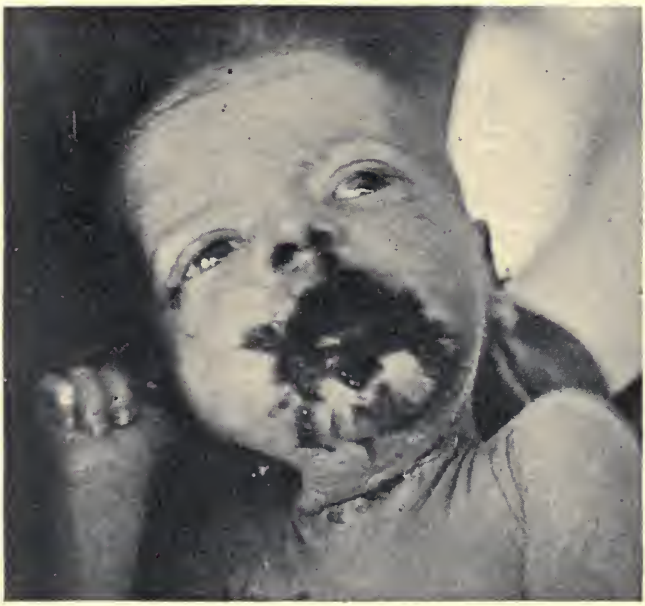


FIG. 195.—Hereditary syphilis, rhagades, bloody rhinitis and excoriations around mouth before treatment.

other symptoms, or again they break out suddenly and disclose at once the whole dark disease-picture.

In infancy two main groups of syphilitic dermatoses, broadly separated in their clinical features, are recognized:

1. The diffuse flat syphilide, or the diffuse infiltration of the skin.

2. The circumscribed eruptions, or the syphilitic exanthemata, in the narrower sense of the term.

1. The diffuse cell infiltration has already been described as the most important histologic sign of syphilitic disease of the congenital type in the internal organs. The skin may become diseased, at a later period, in a manner entirely analogous to the visceral process. Deep-seated, widespread and continually progressive cell proliferation occurs, which eventually leads to a diffuse infiltration of the skin, recognizable at the first glance. Clinically this infiltration appears as a thickening and toughening

of the skin. In extreme cases the normal elasticity of the cutaneous tissues is entirely lost. Wrinkles and fissures form, especially in parts where the unyielding dense tissue is subjected to mechanical force. Very often the affected parts of this skin are reddened and inflamed. This diffuse infiltration of the skin is never present at birth, but always develops at a later

Fig. 196.—Hereditary syphilis, rhagades after treatment. Same case as Fig. 195.

period. Nevertheless, it is a special peculiarity of congenital syphilis and never occurs in the acquired form.

At times the entire skin from head to foot takes on the characters described. Usually, however, certain parts only of the skin are affected and more particularly on the face, the hands and the feet. The trunk commonly remains entirely free.

Many of the external signs of lues are associated with this pathologic condition of the skin. The peculiar pallor of the face of the congenitally syphilitic infant is one of these and while it is not constant, it is still seen very frequently. There has been no lack of effort to find a term to fitly describe this typical color of the skin, but no better designation than that of earthy sallowness or waxy pallor has been discovered. Trousseau compares the color to that of weak coffee diluted with a large quantity of milk. After

the pallor has persisted for some time and large deposits of pigment have occurred he likens it to the stains on the fingers of the cigarette smoker. Often the pale red of the lips of such children offers so little contrast to the color of the face that the line of the lips is not sharp and seems to blend with the surrounding skin. In other words, this pseudoanemia of young syphilitic infants is not due to a lack of blood-supply, but rather to the thickening and increased tension of the skin of the face.

The infiltration is especially marked around the mouth, nose, and eyelids. With the frequent movement of these parts of the skin, superficial and sometimes deeper fissures are readily formed. This is seen particularly in the radiating rhagades of the lips, an extremely important stigma of the luetic facies. If the infiltration is distributed over the entire skin, the face naturally takes on a rigid, mask-like expression.

Very frequently a scab-like, dessicating eruption develops on the bases of this diffuse infiltration. This eruption shows a very close resemblance to the ordinary impetiginous eczema. The parts in which fissures readily occur are especially predisposed. Accordingly, the dessicative eruption is found chiefly around the mouth, nose and eyebrows, as well as on the forehead and scalp which it sometimes covers like a helmet. The scabs are removed very readily and do not leave a bleeding surface. The bases thus exposed are but slightly reddened and present a peculiar satiny sheen. These are all points of importance in distinguishing these eruptions from the impetiginous form of constitutional eczema of the face, which usually makes its appearance at a later period.

Fig. 197.—Typical alopecia in a four and one-half-week-old syphilitic infant.

It is clear that this process of infiltration, if it persists long enough, will lead to serious disturbances of the nutrition of the skin. As one result, we frequently see that the hair of the affected parts disappears. The eyebrows and eyelashes fall out and even at this early date a widespread alopecia of the scalp may appear. Often the hair falls out over certain areas only; thus one-half of the head, from the forehead to the occiput, looks as though it had been shaved, while the hair at the back of the head remains intact. The area of the bald spot is just the opposite of that found in rickitic children.

Other points of predilection for this diffuse infiltration of the skin are those particular parts exposed to special external irritation. Thus it is frequently found in large areas over the nates, on the flexor surfaces of both legs, and especially when these parts are affected with intertrigo. The bright red, wet surfaces gradually become dry and rigid, have a brownish color and present a glistening appearance.

Another favorite localization of the diffuse infiltration upon the soles of

the feet and the palms of the hand is very significant. A similar tendency is seen in syphilitic pemphigus of the new-born and the papular forms of exanthem appearing later, seem to have a predilection for these areas. This is also true of the diffuse flat syphilide. The cause of these localizations is hard to fix. It may be that the early and numerous presence of sweat glands in this region plays an essential rôle (Hochsinger). In any event, the condition is very clearly characteristic of syphilis. Sometimes the soles of the feet are the only parts on which the diffuse cutaneous infiltration is found. The experienced physician will never neglect the careful examination of the soles of the feet when lues is suspected.

The infiltrated skin of the soles is usually reddened or more often livid. The peculiar sheen is especially typical. Often times the entire sole looks as though it had been varnished or covered with shellac. This is sometimes called the shiny or varnished heel. The sheen is caused mainly by the extreme tenseness of the infiltrated skin. It is usually impossible to

FIG. 198.—Syphilitic paronychia of hands and feet. Papulo-pustular syphilide around mouth. Rhagades of lips, loss of hair, of eyebrows, and shaved forehead.

wrinkle the skin, or if one does succeed in this, the folds include only the superficial epidermis which is but loosely connected with the underlying tissue. For this reason large pieces of the horny layer may at times peel off. Not infrequently the first efflorescence of a later exanthem appears upon the basis of the infiltration on the soles of the feet.

The diffuse infiltration of the skin causes very typical manifestations on the finger and toe-nails. A cushion-like inflammatory swelling of the bed of the nail occurs, resulting in deep, trophic disturbances of the nail, the syphilitic paronychia. The region of the nail-fold is of a brownish-red color, is markedly thickened, swollen, shiny and scaly, and is at times covered with scabs and crusts. The nails themselves become soft, thin, and striped, or ridged. They are brittle and break off, or they become soft and maybe completely shed. In milder cases a fraying of the free ends, as a result of which they become of a pure white color, is characteristic.

2. The circumscribed skin eruptions of congenital lues resemble those of acquired syphilis. Unlike the diffuse infiltration of the skin they are not peculiar to infantile syphilis alone.

Syphilitic pemphigus has already been discussed. Attention has also

been called to the fact that this exanthem is not always present at birth and that an essentially similar pustular eruption may appear later, and usually during the first weeks of life. This eruption also occurs chiefly on the soles of the feet and the palms of the hands, but may appear in other parts of the body. The vesicles are generally few in number. In children affected with this late pemphigus (Hochsinger), the prognosis is not so unfavorable as it is with those in whom the vesicles are present at birth or appear during the first or second day.

Frequently the eruption has a more papular character from the beginning, resembling the pustules of small-pox. It is then termed a papulopustular syphilide. The content of the pustule dries up, forming a thick scab which frequently presents a concave, oyster-shell-like form, the rupial syphilide. More rarely, the pustular exanthem appears by preference on the dorsal surfaces of the hands and feet, where it consists of small papules,

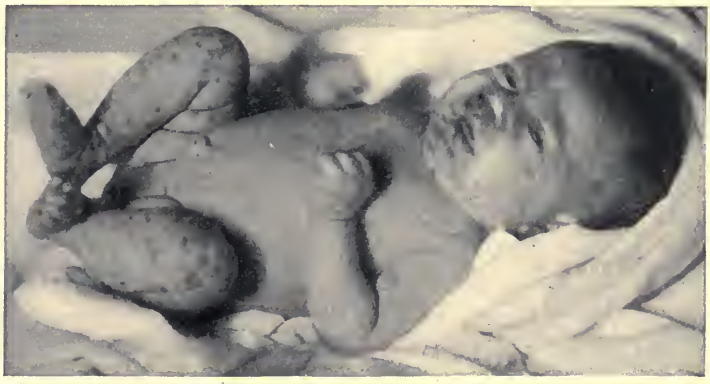


Fig. 199.—Maculopapular syphilide, especially marked on the extremities and face. (Children's Hospital, Heidelberg, Prof. Moro.)

set closely together in circles. The eruption then has a definite circinate form which persists until it becomes confluent.

Generally speaking, however, in infancy this syphilitic exanthem forms the so-called maculopapular syphilide. It is so described because the efflorescence gives, at first sight, the impression of simple spots, but on closer examination shows a slightly raised flat surface formed in layers. These are actually found to be small infiltrated areas. Dermatologically, therefore, this exanthem is not analogous to the roseola of acquired lues. The latter does not occur in congenital lues at all (Hochsinger)

The maculopapular exanthem never appears immediately after birth, but requires an incubation period of several weeks. It consists of small flat sheets, varying in size from one-eighth to one-half inch, which are at first of bright red and later change to a yellowish-brown or salmon color. It is distributed chiefly on the extremities and particularly on the extensor surfaces and the sides of the legs, on the soles of the feet and the palms of the hands, and on the neck and face, while the trunk is nearly always remarkably free from the eruption. Sometimes the crop is so scanty that

much care must be taken to discover occasional spots on the soles of the feet, the forehead or the chin. In other cases, the entire body is thickly covered, so that the exanthem, at first sight, reminds one of measles. In those portions of the skin which are irritated, either mechanically or otherwise, especially around the anus, the exanthem spreads in all directions and not infrequently takes on the character of the so-called broad condylomata.

Subsequently, the surface of the efflorescence is either exfoliated, leaving a smooth, glistening, circular surface; or the exanthem is resorbed from the centre, leaving a very characteristic light brown pigmentation which gives a mottled appearance.

Fig. 200.—Parrot's pseudoparalysis. Typical position of right arm and hand. Spindle-form swelling in the region of the elbow.

The sudden eruption of a widespread maculopapular syphilide in infancy is by no means a bad sign. On the contrary, this event rather indicates that the organism is in active response or reaction to the syphilis antigens. In delicate, weak infants, in fact, the exanthem is very mild; and this coincides fully with the clinical experience, that infants with especially severe cutaneous symptoms show but slight visceral changes. Enlargement of the spleen and the liver, on the other hand, are most frequently found in children whose skin is spared the exanthemata throughout the disease. Such cases are not uncommon. Hochsinger has called particular attention to this fact and has described such cases under the term *syphilis congentia sine exanthemata.*

Specific disease of the mucous membranes, in direct contrast to the frequency of syphilitic skin affections, of which only the more important have been described, are uncommon in early infancy. Occasionally, we find large discrete placques on the tongue, lips, or soft palate. Hoarseness and aphonia, probably due to an affection of the mucous membrane of the larynx, are quite common.

Syphilitic diseases of the bones are frequent in early childhood. The characteristic osteochondritis has been mentioned already and its anatomy described under fetal lues. Milder degrees of osteochondral inflammation hardly cause any clinical manifestations. If the process continues and reaches a severe grade it will sooner or later produce a very remarkable picture. As might be anticipated from the nature of the disease, it is always localized first in the epiphysis. The lower epiphysis of the humerus and in the knee, the epiphyses of the femur or the tibia are chiefly affected. In some instances all four of the extremities are affected. If, as is very frequently the case, the lower epiphysis of the humerus is affected, the elbow

joint often appears spindle-shaped and the entire region is slightly reddened and heated. The serious disturbance of motion which the entire arm suffers is, however, the most noticeable symptom. It lies beside the body in a flaccid paralysis and is rotated inwards with the back of the hand turned toward the trunk. If the arm is lifted by the fingers, which remain movable

FIG. 201.—Hereditary syphilis. Epiphysitis and periostitis of lower ends of ulna and radius.

throughout, it falls back upon the bed as though lifeless. This is not true paralysis, however, as is the plexus paralysis due to birth traumata, to which it is closely related clinically. The nerve supply is perfectly intact; the paralysis is only apparent. It may be due to various causes.

(a) It may be that as a final result of osteochondral inflammation, the epiphysis may have separated from the diaphysis, producing signs essentially the same as those of true fracture.

(b) The process does not necessarily go on to actual separation, but the inflammation passes to the periosteum and to the neighboring musculature, resulting in intense pain, as may be shown by every attempt at motion or at times by even the slightest touch. The pain sufficiently explains the lack of motion in the affected limb. Periostitis and myositis also cause the characteristic spindle-form swelling around the joint.

(c) If there is neither separation of the epiphysis nor severe pain, not uncommonly true in this form of pseudoparalysis, there may be an inflammatory affection of the muscles alone, causing diffuse swelling, a polymyo-

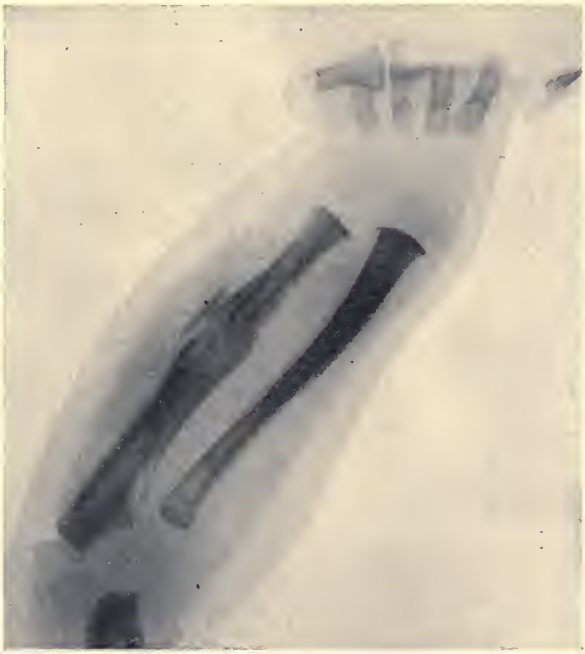


FIG. 202.—Hereditary syphilis. Osteoperiostitis of ulna resembling acute osteomyelitis. An uncommon lesion.

sitis over the elbow-joint which completely prevents every active movement.

The Roentgenogram of osteochondritis is often very instructive. A widened irregular, jagged, dark epiphysial line giving, at times, a broken epiphysial outline is seen, with a considerably lighter zone toward the diaphysis, representing the granulation tissue found in this area. The ossifying periostitis is marked by a dark shadow which covers the shaft like a cloak. The inflammation of the fingers in syphilitic infants—phalangitis syphilitica (Hochsinger), is less clearly marked, since it always begins insidiously, is painless, and never causes serious disturbance of function. In extreme degrees it is easily recognized. The affected phalanx is enlarged, giving an olive-shaped swelling, and the skin over it is tense, shiny and red. The process usually develops in the proximal phalanx, so that the finger becomes

bottle-shaped. At times, the distal phalanges may also be affected. The joints and the soft tissues are always intact. The condition develops very early, usually during the first month and is generally multiple. Hardly ever does it lead to the formation of pus or to a fistula. These points are important in its differentiation from the tuberculous spina ventosa which it may closely resemble in many other respects.

Fig. 203.—Hereditary syphilis. Multiple bone and joint involvements; skull, left humerus, right tibia and both elbow joints. The following four illustrations of bone and joint lesions are from this patient.

The central nervous system is more frequently the seat of syphilitic disease in infancy than was formerly supposed. The brain and its membranes are naturally considered first. Aside from the formation of gummata and the development of inflammatory infiltration and sclerosis which, beginning during fetal life, may lay the foundation of later idiocy, we must first consider internal hydrocephalus. The gradual appearance of this condition must be classed among the common manifestations of congenital syphilis. It is not an early symptom and commonly does not occur until the third or fourth month, or even later. It may, however, be of congenital origin and it may then reach an extraordinary size. The acquired form, in contrast to the so-called rickitic hydrocephalus, is comparatively small. The fontanelle is tense and bulging. The peculiar hydrocephalic stare, with inverted downcast eyes, shown in Figure 203, is also present. The head is but slightly enlarged. This may be due, in part, to the fact that the cranium has become rigid and unyielding as a result of preceding or coincident inflammatory process, giving the type of caput quadratum. In such cases, however, the pressure symptoms are more marked and the frequent appearance of convulsions in these infants is easily explained. The convulsions may also be explained by the presence of an active syphilitic meningitis. The cerebrospinal fluid obtained by puncture is clear and its protein content is but slightly increased. The Wassermann reaction on the cerebrospinal fluid is positive and the cells are increased, often to four or five hundred per cubic millimeter, if the meninges are actively involved. Inflammatory diseases of the choroid plexus and of the ependyma, the

primary disease of the ventricles, is probably the most frequent cause of internal hydrocephalus.

In the causation of the rarer external hydrocephalus, the anatomic basis of which—a pachymeningitis, causes a collection of fluid between the dura and the pia, lues must be primarily considered. Clinically, the

Fig. 204.—Hereditary syphilis osteitis of skull. See Fig. 203.

condition is actually shown only by the slightly sanguineous spinal fluid, suggestive of pachymeningitis interna. To give proper valuation to this discoloration of the cerebrospinal fluid one must be able, of course, to exclude hemorrhage due to the puncture itself.

Of the sense organs the eye is most frequently affected. The plastic iritis of the new-born has been mentioned. At a later period inflammation of the retina, a syphilitic retinitis, is a symptom of specific disease which the

ophthalmologists consider valuable. Choroiditis also often develops. The rare optic neuritis of infantile syphilis rapidly leads to total blindness. Parenchymatous keratitis, so common in congenital syphilis of tardy development, is remarkably infrequent in infancy.

The vascular system often shows marked venous ectasia. This is especially distinct in the scalp and over the temporal region where the course of the veins is sometimes marked by deep winding grooves in the bone.

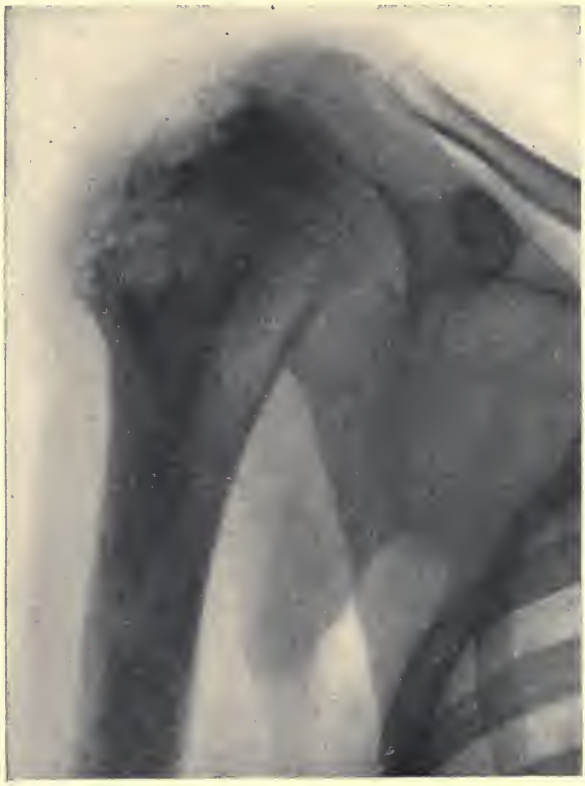


Fig. 205.—Hereditary syphilis, osteitis of humerus. See Fig. 203.

Fournier considers this to be in the nature of a dystrophy. Hochsinger, on the other hand, attempts to find the cause of the enlargement of the temporal veins in a coexisting hydrocephalus. It must be remembered, however, that markedly ectatic veins are seen where there is no hydrocephalus or but in very slight degree. They may be found sometimes in other parts of the body and even in the extremities.

Syphilitic endarteritis, a very common pathologic finding, even in infancy (Heubner), is very seldom determined clinically at this early age. This phase of the disease, which affects chiefly the blood-vessels of the

brain, is readily recognized after the first years of life. As a frequent cause of encephalitic processes it plays an important part.

In this disease, as in tuberculosis, a general swelling of the lymph nodes, in the form of small, hard tumors, appears. The diagnostic value of this micropolyadenitis must, of course, be very carefully weighed, since it may be found, also, in non-syphilitic infants suffering with disturbances of nutrition. The frequent affection of the cubital nodes, while not pathognomonic by any means of lues, is important, for the nodes of this region are but rarely affected in other conditions.

FIG. 206.—Hereditary syphilis, elbow-joint, the "Whorled" appearance is characteristic. Same patient as Fig. 203.

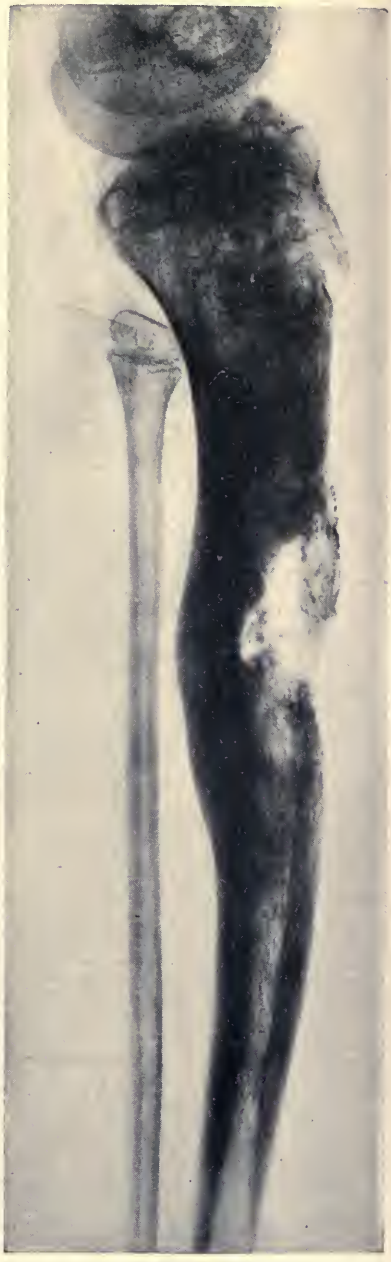


FIG. 207.—Hereditary syphilis osteoperiostitis of right tibia. See Fig. 203.

Since syphilis is a chronic infectious disease, it is not surprising to find occasional rises of temperature without apparent cause. This finding does not necessarily imply a mixed or secondary infection or any other complication. An active syphilis is sufficient cause in itself for such a reaction. Of course, the temperature rise is never very great and is by no means characteristic.

Sooner or later a high grade of anemia is a feature of the disease-picture. This is often associated with considerable reduction of the number of red blood-cells and a marked diminution of their hemoglobin content. Pathologic blood elements also appear in the stained preparation from time to time. The lymphocytes, as a rule, are notably increased. There are periods in the course of the disease during which the uniform delicate pallor is very pronounced, when indeed, it presents the only symptom. Children so anemic are very frail and may die suddenly as the result of some slight indisposition. Furthermore, nearly all these little syphilitics show an increased predisposition to disease and to disturbances of nutrition. La grippe, pneumonia, etc., usually affect them with much more than ordinary severity. This will be readily understood when it is remembered that these children have had to fight a hard fight, that they have constantly fought under the deteriorating influence of a powerful poison, which has reduced almost the entire organism to a condition of functional debility. To this status the terms parasyphilis, syphilitic deuteropathy, or syphilism have been applied and we speak of parasyphilitic anemia, parasyphilitic debility, etc. It might be better, however, to reserve this con-

FIG. 208.—Multiple thickening of the bones in congenital lues. (University Children's Hospital, Breslau, Prof. Tobler.)

FIG. 209.—Moderate syphilitic hydrocephalus, and natiform head. One and one-half-year-old girl. (University Children's Hospital, Zurich, Prof. Feer.)

ception for those cases in which these anomalies appear as signs of constitutional degeneration in the non-syphilitic descendants of syphilitic parents.

Recurrences In Early Childhood

From time to time, manifestations of the disease appear suddenly in congenitally syphilitic children, after long intervals of apparent recovery, which must be looked upon as recurrences. The manifestations already described are in the main merely individual symptoms in a continuous reaction process, the persistent and uninterrupted course of which is not appreciated because of the occasional absence of definite clinical signs to mark it with unmistakable clearness. Naturally many of these reappearing phenomena suggest relapse, since they announce the reawakening of a process which often runs its complete course during fetal life. Nevertheless, their recurrent quality is not so clear as when, after many months or even years of repair, fresh outbreaks occur suddenly and show the renewed activity of lingering foci of spirochætes. In the meantime, partly as the result of a measure of immunity attained during the struggle of these early months and partly because of its more advanced development, the organism has acquired a different sort of reaction to the syphilitic poison, distinctly shown in the altered character of the clinical symptoms. The evidences of reaction are no longer of so general a nature. They are more definitely localized and confined to special parts, where they assume a more intensive type. Very often the course of these reactions is extremely rapid. Eruptions appear suddenly and disappear as quickly. Accordingly, they are very amenable to local treatment. This is particularly true of the broad condylomata of the skin and of the syphilitic placques of the mucous membranes, both of which are so typical of this recurrent period as to have suggested the term, the condylomatous stage (Heubner).

Fig. 210.—Recent papules of the labia. Wide condylomata around the anus. Recurrence of congential syphilis in two and one-half-year-old child.

Externally, these condylomata are exactly like those of acquired lues. They are red, exudative, proliferating papules, varying from one-eighth to one-half inch in diameter. They spread rapidly in all directions, often showing irregular indentations and fissures upon their surfaces. They are found most frequently around the anus and the genital organs, while the structurally similar placques are found on the lips, on the upper surface of the tongue, or on the tonsils. Children of two to four years of age are commonly affected.

Gummatous neoplasms are of rarer but occasional occurrence at this period. They appear in the skin and subcutaneous tissues and especially

on the fingers, feet and scalp. They develop as multiple nodular infiltrates. If these are not recognized and treated early they break down rapidly and leave sharply chiselled, white ulcers, with indurated bases, which prove extremely obstinate. In the larynx they occur in the form of knotty, papular proliferations which sometimes cause croupy symptoms. In the viscera, and especially in the liver, they form the so-called solitary syphilide.

The nodular swellings of the testes which are occasionally observed in the course of these relapses are not, as a rule, true gummata but represent a diffuse interstitial cell proliferation.

General exanthemata, resembling those of the earlier eruptive period, also occur. They are comparatively rare and run a characteristically milder course.

Infantile pseudoleukemic anemia, with its enormous, hard spleen, is not uncommon in syphilitic children and usually develops during the second year. Severe rickets is commonly associated with it, but the essential part which syphilis plays in its causation cannot be determined. The possibility, however, must be admitted. The frequency with which pseudoleukemic anemia occurs with rickets alone, when no syphilis can be demonstrated, casts considerable doubt on syphilis as a factor.

Late Congenital Syphilis

Congenital syphilis may break out anew, and in special guise, at the period of the second dentition, and frequently even later during the development of puberty. While in early childhood the condylomata give a characteristic feature to the relapse, the entire clinical picture is now governed by the gummata. Gummatous proliferations are found in the bones, the periosteum, and the bone-marrow; gummatous nodules in the skin and mucous membranes; gummata in the brain, the liver, the spleen and the lymph nodes. None of these processes are distinguishable, either clinically or anatomically from the tertiary phenomena of acquired lues.

Gummata of the bones are found most commonly in the tibia, the cranium and the sternum. The nodules, primarily soft, soon become hard, and subsequently show a great tendency to ulceration. They may go on to deep, irregular, and obstinate sores. If the nodules are resorbed, permanent scars, the so-called tophi, firmly adherent to the skin, are left in the bone. These gummata do not arise from the bone substance itself, but from the periosteum. In the hard palate and in the nasal septum the gumma is often situated in the marrow and proliferating thence may finally lead to deep erosions and complete perforation, leaving cavities of variable size. The saddle nose of syphilitic children, however, usually dates back to the period of the first eruption.

Similar perforations arise also from the mucous membranes. Not infrequently depressed radiation scars of the soft palate, the faucial pillars and the uvula are seen. In the tonsils, at first swollen, indurated and deeply infected, the gummatous process readily leads to ulcerative breaking down of tissue. The remnants present a yellowish-white slimy mass. The inexperienced observer immediately thinks of diphtheria and may even resort

to antitoxin. Of course, the serum fails of result. Severe and readily recurring affections of the mucous membranes are observed, especially in those syphilitics who in early childhood have shown the distinct manifestations of exudative diathesis, giving the so-called scrofulo-syphilis.

The gummata of the skin are sometimes very large when they arise from the subcutaneous cellular tissue. They exhibit a course similar to the gummata of the earlier period of recurrence already described. Again they may be small, varying from the size of a pinhead to that of a pea. These small nodules always lie very closely together, as in lupus; they ulcerate readily and very frequently have a serpiginous or a circular arrangement.

Fig. 211.—Periostitic thickening of both tibia, especially marked on right, case of late congenital syphilis. Six-year-old girl.

Of the viscera, the liver is most frequently affected. The large gumma of this organ and the hypertrophic cirrhosis frequently associated with it, develop enormous tumors. In such cases the spleen, also, is always markedly enlarged. If ascites is added, the condition may simulate tuberculous peritonitis, but the confusion is cleared if jaundice appears. Moreover, in these cases, other stigmata of syphilis can always be found.

The rare circumscribed gummatous formations in the brain result in intense headache, occurring chiefly at night, and lead on to epileptic attacks and paralyses. True gummata of the lymph nodes appear in the very slow but noticeable intumescence of isolated groups, especially in the neck. The common enlargement of the cubital nodes, usually presents, pathologically, only a simple hypertrophy.

The hyperplastic periostitis of the diaphysis of the tibia is a frequent, a very typical, and a quite pathognomonic symptom of this period. The overlying skin is thickened, tense, shiny and slightly reddened. Usually the condition is attributed to a trauma which has occurred at some previous date and which may enter into the consideration as a causative factor. Upon palpation, which may or may not be painful, one gets the impression of a solid spindle-shaped tumor. Often times an irregular outline due to small excavations makes the sharp edge of the tibia feel like a blunt saw. At times the edge cannot be felt at all, being completely rounded off.

The disease affects mainly the periosteum which gradually ossifies and repeatedly forms a new shell around the bone. In advanced cases the tibia may become curved, the curve characteristically taking a forward direction and resulting in the well-known saber tibia—a beautiful subject for radiographic demonstration.

At this period, a very important, although not an absolutely pathognomonic sign of late syphilis appears in the form of a parenchymatous keratitis. This lesion sometimes leaves a permanent clouding of the cornea, which serves as an important stigma. Manifestations of late syphilis in the internal ear almost always cause complete deafness.

Keratitis and deafness not infrequently occur together. To these may be added a third external sign affecting the incisors. This completes the Hutchinson's triad, so-named from its original demonstrator.

This physician also described the anomalies of the teeth very accurately and, as a result, they are known as "Hutchinson's teeth." The cutting edges of the upper central incisors are concave and their lateral edges are rounded

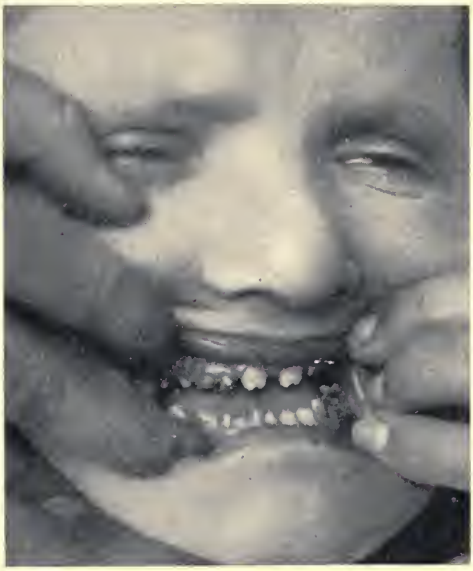


FIG. 212.—Hutchinson's teeth and microdontia in late congenital lues. Ten-year-old girl.

or convex. Frequently, but not invariably, the dentine is exposed at the base of the concave edges as a result of enamel defect.

According to Heubner, another symptom may form the fourth member of this group in the form of a chronic, bilateral, ankylosing inflammation of the knee-joint, as a result of a syphilitic gonitis. This begins as a simple effusion into the joint cavity and gradually extends to the neighboring bone causing a permanent deformity.

Regarding progressive infantile paralysis and infantile tabes, both of which may be quite apparent even before puberty, the reader is referred to the chapters on Nervous Diseases.

THE DIAGNOSIS OF CONGENITAL SYPHILIS

Not much can be added upon this question, since the chief diagnostic points may be deduced from what has been said. For the rest, there is no

field in clinical medicine in which we must depend so much upon the teachings of practical experience and personal observation as in this particular domain. Descriptions, although repeatedly studied, are of little value. Unless the physician learns to observe and to study the conditions for himself he may never become an adept in the diagnosis of syphilis and he may inflict much injury, as he does, for instance, in placing a syphilitic child with a healthy wet-nurse. Three points may be especially emphasized.

1. The Very Rare Occurrence of Single and Very Discrete Clinical Manifestations.—It must not be imagined that the entire list of signs and symptoms described appears in any one case. In the infant, during the first few weeks, the snuffles, or a palpable spleen, or a waxy pallor of the face, with a few small fissures around the mouth, or a small suspicious-looking spot on

FIG. 213.—Saddle nose in congenital syphilis. (Children's Hospital, Munich, Prof. Pfaundler.)

the forehead, or a peculiarly shiny heel are not infrequently the only signs discoverable upon examination. By careful observation of the course of the disease it is usually possible to discover other signs from time to time.

In very rare cases, however, early syphilis takes its course even more uneventfully. If this were not the case it would hardly be possible to find experienced men who still believe in "syphilis hereditaria tarda sensu strictiori," in which congenital lues is supposed to become manifest for the very first time in advanced childhood, or even later.

2. Syphilitic Stigmata.—These consist chiefly in scars left by passing specific processes and in permanent changes in the skeleton. It may readily be imagined that the discovery of such stigmata often decides at once the nature of a cerebral disturbance or a chronic pelvic condition. Besides the less common formation of scars in the mouth and the throat and around the anus, often indistinct radial scars at the edges of the lips are an undeniable

sign of congenital lues (Hochsinger). In the osseous system, the saddle nose must be considered a most important stigma, especially when it is combined with a hydrocephalic cranium, and a protruding forehead and occiput—the caput quadratum.

We must be warned, however, against laying too great stress upon the diagnostic value of a "pug nose." In such a case, the form of the noses of the parents must be taken into consideration in order to determine whether it is not a question of an ordinary familial peculiarity. In early infancy, in fact, a "pug nose" is a very common thing. The presence of Hutchinson's teeth alone is not a very definite sign.

While children with congenital syphilis are often small and of poor sexual development (infantilism), this is not necessarily true.

3. Suspicious Signs in Non-syphilitic Children or Symptoms of Pseudo-lues.—The beginner often makes the diagnosis of syphilis upon the strength

FIG. 214.—Plaques erosives (syphilitic erythema of the nates). (Dresden Infants Institute, Prof. Schlossmann.)

of changes which have no real relation to lues. Especially misleading in this respect are the so-called placques erosives, which very often occur in areas of improperly treated intertrigo or in the course of a papulo-vesicular dermatitis of the nates. Decubitus of the heel in atrophic infants also causes mistake. More pardonable is the confusion of certain cases of dermatitis exfoliativa with syphilis, especially when the region of the mouth is infiltrated and covered with excoriations and fissures (see Fig. 195). On the contrary, erythrodermia, with its general seborrhœa, is easily distinguished. The snoring respiration of infants with adenoids may possibly resemble, in a degree, the snuffles of the luetic. Bednar's aphthæ and thrush have no more relation to lues than has the geographical tongue. It is not impossible, however, that a very distinct geographical tongue may develop as a late result of a syphilo-toxic dyscrasia.

The Wassermann reaction is a great aid in the determination of a dif-

ferential diagnosis. This reaction depends upon the presence of a thermostabile body in the blood-serum of the syphilitic individual, which possesses great affinity for lipoid organic substances soluble in alcohol. The original method of Wassermann has so far proved most reliable. By this method the inactivated serum to be examined is mixed with a watery extract of the liver of syphilitic fetuses. If the serum of guinea pigs, containing complement, is added to this mixture, the complement is combined if the serum to be tested is luetic. This combination of the complement does not permit any further action. If now a so-called hemolytic system (*e. g.*, blood-cells of a sheep inactivated anti-sheep's blood immune serum from the rabbit), is added, no hemolysis or very incomplete hemolysis of the sheep corpuscles

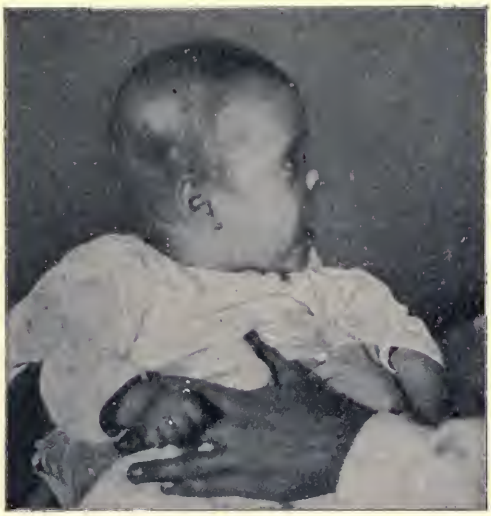


FIG. 215.—Luetic facies of a two and one-half-year-old child. Typical saddle nose, moderate hydrocephalus.

takes place; whereas, if the complement had remained entirely free the erythrocytes would have been entirely dissolved. The resulting limitation of the hemolysis, or its absence, serves as the indicator.

For the principles, the technic, and the details of the reaction the reader is referred to special works on serology. If it is carried out by an experienced technician this method is simple and reliable.

The original method of Wassermann has been modified by most laboratory workers by the use of extracts of beef or guinea pig heart instead of syphilitic liver as antigen and by using smaller amounts of reagents. The reaction is non-specific and is just as reliable with non-syphilitic as with syphilitic antigen. The chief advantage of the modification is the availability of non-syphilitic antigen.

A distinctly positive reaction is conclusive, but a negative reaction does not absolutely exclude lues. Nevertheless, the reaction is extremely useful

in practice and particularly in the recognition of occult cases of congenital late syphilis.

The demonstration of the spirochætes, which are readily found in the eruption of infantile lues, is hardly necessary for a clinical diagnosis.

Prognosis

The prognosis is dependent upon four main factors:

1. The Care and Feeding.—With proper care and natural feeding the prospects are generally good. With inefficient care and artificial feeding, on the contrary, they are bad. In the mere matter of artificial food, however, it is not safe to say that non-syphilitic children have much advantage over the syphilitic. Nevertheless the syphilitic infant is usually weakened from the start, and, therefore, shows less resistance to the innumerable abuses of feeding and care. To these dangers must be added the greater one of secondary infection through numerous wounds, rhagades and fissures. But even under the most exemplary care the syphilitic child fares better with natural than with artificial feeding, be the latter ever so exactly and so scientifically conducted.

2. The General Condition.—Weak syphilitic prematures generally have a poorer prognosis than stronger full-term luetics. This is quite self-evident. In both cases the prognosis becomes more grave when some severe disturbance of nutrition, or an attack of la grippe or pneumonia is added.

3. The Character of the Syphilis.—Actually, the prognosis depends to a great extent upon the character of the syphilis of the parents. The prospects are much worse with recent syphilis than with old infections and also more serious in untreated than in treated cases. The well established fact that the number of still-births and premature births is in a general way proportionate to the age of the parental syphilis (Kassowitz), bears out these conclusions. With regard to the nature of the infantile syphilis, there can be no doubt that the prognosis is more favorable if the first signs of eruption appear late. Children who are born with distinct symptoms almost always succumb. The seriousness of pemphigus in the new-born has already been emphasized. Similarly, children with marked visceral syphilis, in whom no distinct eruption ever occurs, hardly ever reach advanced childhood.

4. Finally the prognosis depends upon the period at which treatment is instituted. The earlier the specific treatment is begun the better is the prognosis with regard, not only to life, but also to recovery. Cases in which the treatment is begun at the proper time and is continued for a sufficiently long period may never have any recurrences. Exceptions to this rule are to be noted however.

The condylomatous stage of recurrence in early childhood is generally benign if it is properly treated. This is not true, however, of late syphilis, which is extremely obstinate. Of course the prognosis depends largely upon the organs affected. Disease of the skin or the bones will cause less anxiety than that of the brain, liver or kidney.

The Treatment of Congenital Syphilis

Before entering upon the medicinal treatment of syphilis two points of great importance must be discussed:

1. The prophylactic treatment of the parents; 2. The question of feeding.

1. The tragedy of repeated still births and the occurrence of very severe cases of syphilis of the new-born are associated with untreated or but slightly treated parental disease. After a recent and thorough course of mercury the conditions are markedly improved. Undoubtedly it is possible that a father with recent and untreated syphilis can beget non-syphilitic children, but this is so rare an exception that it has small significance. A physician cannot give his consent to the marriage of the syphilitic unless the infection of the man, probably the most frequent question under consideration, is at least four years old, unless the patient has undergone several systematic courses of treatment, and has shown no relapses during the entire period. Shortly before marriage another course of treatment with mercury or salvarsan should be instituted. Even then the advisor cannot promise full immunity. If the offspring of such a marriage is infected an energetic course of specific treatment should be advised for both parents in order to avoid infection in future offspring. The mother should be treated even though she be pregnant and without considering whether she has or has not shown symptoms.

2. We have already recommended natural feeding for syphilitic infants. This presents no difficulties if the mother can nurse the child. She cannot be infected by her own child, since she is already infected. If, however, the mother cannot nurse the child for one reason or another, the question of a suitable wet-nurse arises and only a syphilitic wet-nurse can serve a manifestly syphilitic child. Aside from the obvious unpleasantness of employing such a person, it is not always possible to find her. The employment of a healthy wet-nurse for a syphilitic child cannot be considered and must be absolutely prohibited. It should be forbidden even when a nurse, with full knowledge of the circumstances, is to be hired, with the intention of keeping her own child at the breast to prevent the loss of her milk and of feeding the syphilitic child the expressed milk only. Aside from the danger of infection to the nurse, her child, also, would be exposed. Such an arrangement can be made without objection only in institutions. The use of mother's milk obtained away from the home, with complementary feeding later, is another possible solution. If this is impossible, there is nothing for it but to fall back upon artificial feeding.

If the child is entirely without symptoms and it remains so for the first four to six weeks it may be given to a wet-nurse. But at the slightest sign of infection in the infant, under the most careful observation, it must be taken away.

Mercury and iodides play the leading rôle in the therapy of syphilis in children. Numerous observations of the treatment with salvarsan are reported, but the experiences with it in young children and especially in infants have led to no definite conclusions. In older children the results

seem to be about the same as those obtained in adults. The views of the effects of salvarsan in infants are widely divergent. Intramuscular or subcutaneous injection is out of the question on account of the danger of local irritation. Intravenous injection, on the other hand, always meets with serious technical difficulties, whether the injection be made into the prepared vein of the arm or into a vein of the scalp. It is the editor's opinion that arsphenamin products have the same usefulness in infancy as in later childhood or in adults with acquired syphilis, and that for one with ordinary skill serious technical difficulties in the intravenous administration are seldom encountered. In the majority of instances the external jugular vein is readily available without preparation. Though the longitudinal sinus is easily accessible it is recommended that it be not used for administration of arsphenamin because of the serious result attendant upon extravenous administration of even minute amounts.

With the small doses employed in infants, there is no essential difference between salvarsan and neosalvarsan. It is the editor's opinion that there is the same difference as with the large doses employed in adults. As a matter of fact, the dose is relatively as large as in adults, *viz.*, 0.01 gram arsphenamin or 0.015 gram neoarsphenamin per kilogram of body-weight. When treatment is first started it is advisable to use somewhat smaller doses and to proceed cautiously, but later the full dose stated above may be used with safety. A safe and effective method is administration every five to seven days for three doses, and a repetition of the three doses every two to three months. For practical purposes, however, neosalvarsan is probably to be preferred on account of its simpler technic and readier solubility. The salvarsan treatment, even in infants, should be combined with mercurial treatment. Mercury is employed in two ways in infancy:

(1) By mouth, in powder form; and (2), by inunction in the form of ointment. With young infants the first method will be found sufficient. Among the preparations which may be given in this way the yellow iodide of mercury (hydrargyrum iodatum flavum), the so-called protoiodide, deserves first mention. It is given in doses of 0.01 gm. (gr. $\frac{1}{6}$), each day and, if possible, is continued until all symptoms of syphilis have disappeared. The treatment may be continued for a subsequent fourteen days if desired. It is unfortunate that so many otherwise excellent syphilographers practice and teach the treatment of infantile syphilis only until the disappearance of symptoms. The same reasons for continued treatment apply here as in the case of the adult with early acquired syphilis. The goal should be the complete eradication of the infection resulting in freedom from the later manifestations of the disease. In some instances diarrhœa appears during the administration of the protoiodide of mercury and probably as its result. But these intestinal disturbances are usually more pleasant than the annoying eczema which may appear as the result of the more difficult mercurial inunction in the delicate skin.

The inunction treatment is usually employed with older children. It does not make much difference what form of mercurial ointment is employed.

The important thing is that one gram of the ointment be gently but thoroughly rubbed for five minutes into the skin, previously washed well with soap and water.

In order to protect the skin, the inunction is made over a different part each time; for instance, the first day, on the chest; the second day, over the abdomen; the third day, on the upper part of the back; the fourth day, to the lower part of the back; the fifth day, to the thigh; and the sixth day, on the upper arm. This rotation is interrupted on the seventh day for a cleansing bath and the whole process is repeated several times.

If there are deep-seated local processes, such as a tibial periosteitis, local inunctions may be made over it.

Condylomata disappear most rapidly with these inunctions, although the same results may be obtained by powdering the growths with calomel daily.

For the local treatment of an obstinate rhinitis an ointment of yellow mercuric oxide or of sozoiodolate of mercury (½-1 per cent.), may be introduced into the nose on small cotton tampons.

Rhagades and other excoriations may be touched with silver nitrate stick or in any other appropriate manner. They should never be ignored.

In the manifestations of late syphilis, the iodides are used to great advantage in combination with the mercury. The editor knows no valid reason for not administering the two drugs during the same period if it is desired. Either potassium or sodium iodide in aqueous solution may be used, giving one teaspoonful to one dessertspoonful of a 5 per cent. solution three times a day according to the age of the child. In order to get efficient results, the iodide must be continued for several months. Springs, the output of which contains iodides, such as are found at Tälz, in Bavaria, and at Halle, in Austria, are appropriate substitutes. When, either by clinical observation or by examination of the cerebrospinal fluid, the central nervous system is known to be involved, it is found that in most early cases and in many late cases the general systemic treatment as outlined above is effectual in accomplishing at least a serologic cure and frequently a clinical cure. There can be no replacement of destroyed nervous tissue. If, after six months to a year of such treatment, no improvement is noted either in the clinical manifestations or the pathology of the cerebrospinal fluid, it then becomes desirable to administer treatment intraspinally. Though the value of intraspinal therapy is in dispute the weight of evidence is in favor of the usefulness of this measure. Our personal experience would also bear this out. The injection of either neoarsphenamin or arsphenamin in aqueous solution directly into the subarachnoid space has been shown to be dangerous because of the irritant effects. Serum arsphenaminized either *in vitro* or *in vivo* is much less irritant and quite safe in proper dosage. Serum obtained from blood withdrawn one hour after intravenous arsphenamin contains in the neighborhood of 0.015 mg. of arsphenamin per cubic centimeter. The amount of serum injected varies from 5 to 15 c.c. according to the size of the child. Inactivation of the serum at 56° C. for one-half hour increases its efficacy. Because of the variability of the amount of arsphenamin to be found in serum arsphenaminized *in vivo* a more constant dosage

is obtained by adding the arsphenamin *in vitro* before inactivation. The dose should be no greater than that noted above and care must be taken not to over-alkalinize. The intraspinous treatments are more effective when combined with intravenous. They may be repeated every five to ten days depending upon the amount of reaction from the treatment. Three to five such injections may be made to a series.

Before declaring a patient as possibly cured Fournier required a minimum of three to four years of treatment and a period of absence of symptoms for eighteen months to two years. Modern arsenical treatment has permitted the shortening of the treatment period. It is also recognized that an infection treated early is more quickly eradicated than one treated late, so that much depends upon the stage of the disease. Even with our more modern methods it is scarcely safe to treat an infant less than one year or an older child less than two years regardless of the effect treatment has had upon the Wassermann reaction. If either the blood or cerebrospinal fluid Wassermann is positive after this, time treatment must be continued until both are negative. Observations should then be made at intervals for several years both in regard to the Wassermann reaction and recurrence of clinical manifestations. Should there be a recurrence the treatment should be undertaken as if none had ever been given. To the present our experience has been that once a cerebrospinal fluid is negative it always remains negative. When a spinal fluid is once known to be negative lumbar puncture need be made again only for final discharge. Amounts of spinal fluid up to 2 c.c. should be used for the Wassermann reaction and in addition there should exist no other pathology such as positive colloidal gold, globulin or increased cells. When determining the status of the infection by means of the Wassermann reaction all treatment should be discontinued for at least two weeks prior to taking the blood specimen.

It is the common impression that hereditary syphilis is cured with difficulty or not at all. There may be some question as to the absolute cure of the disease, but with the modern methods of treatment a persistently negative Wassermann reaction and freedom from symptoms has been the rule in our hands. In some cases as much as three years of treatment may be required. Sight is not restored to a child with optic atrophy nor does power always return to paralyzed muscles but except for any such irreparable damage it is our belief that every child with hereditary syphilis may be cured as judged by our present standards of cure.

II. ACQUIRED SYPHILIS IN CHILDREN

Syphilis may be acquired in numerous ways in childhood. The infection may be transmitted during the act of suckling of the syphilitic mother or wet-nurse; or by means of utensils; by caresses, by venereal attendants, etc. Consequently, the most common location of the primary lesion in children is the mouth and especially the lower lip.

Otherwise than in the mode of infection, acquired syphilis in the child presents the same course as in the adult. Primary lesion and bubo; second-

ary stage with exanthem and condylomata; and tertiary stage with the symptoms of gumma formation are alike.

The question may arise frequently, whether in a given case we have to deal with congenital or acquired syphilis, especially since very mild initial manifestations may be entirely overlooked in the infant. In a three-year-old child, for example, with several condylomata around the anus, the question is not always readily answered; for in such a case we may have to deal with a recurrence of congenital syphilis or with the secondary stage of an acquired lues. Similar difficulties are presented in the determination of the manifestations of the tertiary stage. Such questions must not be regarded as mere clinical subtilties, for they are certainly of practical importance. In the first place, it can hardly be a matter of indifference with the father, whose conscience is not quite clear upon questions of the past, to know whether he is to blame for the unfortunate condition of his child or whether the infection has come to the child from an attendant. In the second place, if acquired syphilis is proved, every effort must be made to discover the source, in order to avoid further accidents. In such cases, the stigmata, described on page 778, are of great value and the most important among them are the radial linear scars of the lips which indicate beyond doubt congenital syphilis (Hochsinger). If, however, no stigmata whatever are to be found in a strong healthy-looking, but infected child, acquired lues must be considered.

It must be noted that the general exanthemata of acquired syphilis are always of a more spotted character and are not maculopapular as those of congenital syphilis. True roseola, completely lacking in the picture of congenital syphilis, is occasionally met with in this group.

Acquired syphilis usually takes a milder course than the congenital form, and consequently its recurrent manifestations are especially amenable to treatment.

XI.

DISEASES OF THE SKIN

BY

ERNST MORO,
Heidelberg.

EDITED AND REVISED, WITH SUPPLEMENT, BY

HARRY G. IRVINE, M.D.,
Associate Professor of Dermatology and Syphilis, Medical School, University of Minnesota; Director of the Division of Venereal Diseases, Minnesota State Board of Health.

INTRODUCTION

In looking over the polyclinic records of former years, we find the diagnoses of eczema, lichen urticatus, impetigo, etc., fully as often as we do those of dyspepsia, bronchitis, angina, and the like. We may conclude then that diseases of the skin are very characteristic to childhood; and, not only that, we even find, among these diseases at this early age, varieties and types which are seldom or never met with in later life. For this reason alone, a clinical knowledge of the various dermatoses of childhood is of great importance to every physician.

To this consideration must be added another and probably even more important argument. Internal disorders are often revealed very distinctly upon the skin. Thus, in the infant, exanthemata of extremely discrete character may easily clear up the doubtful diagnosis of syphilis and tuberculosis. It is not possible, of course, to make a positive diagnosis from a single lentil-sized papule on the forehead, or from two or three minute reddish-brown papules on the abdomen. But further examination is directed by these important discoveries along certain definite lines which might not have been followed or, at least, so quickly followed, had not the uncovered skin shown these changes, in themselves so apparently insignificant.

A large number of such examples might be cited and each show, with equal distinctness, that the cutaneous surfaces offer, indeed, a veritable mine of suggestive symptoms and hence that their changes deserve the closest attention.

Of course, the relations suggested are not always as clear as in syphilis or tuberculosis. It would be extremely difficult, in fact to place the proper value upon the most ordinary dermatoses of childhood, such as eczema and urticaria, had not clinical experience and observation taught us that, at least in early childhood, even these exanthemata must be regarded as expressions of an internal disease condition hardly second to rickets in frequency.

This definite disease condition has been studied and can be differentiated clinically, even though we have as yet no definite knowledge of its true

nature, which is surely often congenital and sometimes hereditary. It appears, therefore, that in it we have to deal with an abnormal condition which amounts to a constitutional anomaly or diathesis. Since its chief characteristic is the marked tendency to reactive inflammation, the condition is suggestively termed the inflammatory, lymphatic or exudative diathesis.

In the conception of the writer, an important element in this predisposition of infancy is the excessive and often severe reaction to stimuli of the vasomotor mechanism. The external evidences of this reaction are many and it is a largely responsible factor in the development of skin eruption.

At times this predisposition becomes manifest even during the first few weeks of infancy. It is seen in the excess of the physiologic seborrhœa of the scalp. The abnormal formation of these scales is the result of a marked increase in the secretion of sebum. The term exudative diathesis, proposed by Czerny, seems suggestive, in the very meaning of the word exudation, of the common characteristic of the condition—an abnormal output or excretion from the skin.

Whether every form of eczema and every exhibition of urticaria in childhood is to be regarded as a cutaneous manifestation of this diathesis remains a matter of opinion. For certain infantile eczematous and urticarial outbreaks, without any apparent exciting cause, this view has indeed a clinical justification. However, this is a matter of very secondary importance. The essential point is that the frequency of this causative relationship be recognized and that the cases be treated accordingly. For the constitutional anomaly in question is not absolutely irreparable, but yields in large measure to suitable methods of treatment. If the physician is successful in this, he not only frees the child from an unpleasant and annoying skin affection but probably saves it, also, from many other serious expressions of the disease.

The writer makes this brief diversion into a subject, already fully discussed in another part of this work, in order to emphasize the point that even the most trivial affection of the skin should not be lightly regarded; that we must not content ourselves merely, in each and every case, with purely symptomatic diagnosis. He wishes, moreover, to suggest the especial importance of this conception of a predisposition in the domain of dermatology.

The predisposition to skin affections, however, is not always congenital. It may be acquired. By way of illustration may be mentioned, the acquired predisposition to various skin conditions of a non-specific nature, which result from infection with the tubercle bacillus. If this acquired predisposition accidentally falls upon the soil of a congenital diathesis, manifestations arise, which indicate a mixture of the two conditions and, as a result, we have the characteristic picture of scrofula, with its extremely peculiar skin appearance often discernible even from a distance.

Another example of an acquired predisposition to certain skin affections is seen in the frequent appearance of furunculosis in infants who, for a long time, have been subject to disturbances of nutrition. The causal agents of these multiple skin abscesses are pyogenic cocci, but these common in-

habitants of the skin would not have caused inflammation and suppuration had they not found a favorable soil. It is immaterial whether gross chemical alterations of the cutaneous tissue have produced this change in the soil, as suggested by Blochs, or whether the invasion of the infective agent is to be considered solely as an expression of the loss of the natural resistive power of the body as a whole. However this may be, the pyogenic cocci serve, as in the group of impetigos, as causative factors of the cutaneous inflammation.

This suggests, again, a very essential point in the pathogenesis of skin disorders, the causative factor. In case bacteria enter into the equation, the problem is a comparatively simple one. These micro-organisms penetrate the skin and cause an inflammatory reaction. Of course this is not invariably true, since, if it were, the same opportunity of infection or an identical inoculative experiment with virulent cocci would always cause impetigo or, at least, an inflammatory reaction in all skins. Since, however, we find that only a certain group of individuals develop an impetigo under given conditions, we must look to discover in them a special predisposition of the organism, to attack.

In a large number of skin affections, moreover, bacteria do not enter into the consideration at all; or at least are not primarily responsible. The causative factors in the production of skin diseases must be of other origin. In general it may be said that there are, doubtless, stimuli, either of endogenous or ectogenous character, that is of internal or cutaneous origin, which potentialize these reactions.

Internal Stimuli.—A fitting example of a dermatosis of endogenous character is found in the exanthem, which is frequently seen upon the skin after the injection of a serum foreign to the species. The mode of action of this result is now fairly clear. By the action of antibodies formed within the organism, a toxic principle is liberated from the injected substance (antigen) which may provoke, among other things, an inflammatory reaction of the skin (von Pirquet, Schick). The explanation, for want of a better, will probably serve for the appearance of a large number of acute exanthemata, and also, within certain limits, will account for a number of so-called toxic eruptions which appear as individual idiosyncrasies.

Very often the toxins of intestinal bacteria and the enterogenous products of putrefraction are cited as the internal causes of the dermatoses of childhood. While it is possible that these factors play some part, nevertheless, too much stress is probably laid upon their importance. While it is often true that children with eczema or urticaria are habitually constipated, it may by no means be considered proved that fecal retention is the causative agent of the skin condition. The clinical observation merely goes to show that obstipation is frequently found accompanying an existing constitutional anomaly. If, as the result of an increase of vegetable food-stuffs in the dietary and a decrease of milk, the obstipation disappears, together with the dermatosis, the coincidence may be properly attributed to the favorable influence of the improved method of feeding upon the digestion in general. Indeed, the occasional beneficial result obtained from catharsis

and from the use of the so-called intestinal antiseptics, the value of which will not be discussed here, may be nothing more than the effect of a temporary reduction of the dietary.

Moreover, there are certain dermatoses, of familiar and widespread occurrence, which not uncommonly appear in children with functional disturbances of the organs of internal secretion, or at puberty when the genital organs are maturing. In these cases, toxic substances of endogenous origin probably serve as common factors. At present, however, there is so little positive knowledge of this relationship that we must content ourselves with the mere mention of the matter.

Practically the same thing is true of infantile eczema, the skin disease of greatest clinical importance in childhood. We have already emphasized the fact that the basic cause of its appearance is to be found in the constitutional anomaly. Nevertheless, in spite of the most persistent research, no definite knowledge as to the nature of this relationship has been achieved. Clinical experience would indicate that overfeeding of milk has an important bearing. The endogenous toxin causative of eczema has been ascribed to one or another inappropriate component of the food, *e. g.*, fat or salt. At the present time, however, it is impossible to make any definite statement of results obtained by the removal of any such supposedly injurious component of the dietary. Under a marked reduction of the milk-supply, the conditions have not always permitted us to form precise conclusions of benefit or the reverse.

External causes, doubtless, play a large part in the causation of the dermatoses. That these external injuries suffice to induce inflammatory skin affections is exemplified by the wheals of nettlerash and the urticaria following insect bites. Indeed, the most important factor often seems to lie in the sensory irritation. This is not surprising if we remember that the sensory nerve termini represent the receiving organs of the reflex neurones of the peripheral vasomotor mechanism.

A good example of an eczematous dermatosis dependent upon essentially external causes is presented in the inflammation of the skin, generally recognized under the term intertrigo. The external irritation of the nates by the diaper, wet with the urinary and fecal discharges and of the neck kept moist by the acid vomitus, staining the collar of the infant's dress which constantly rubs the skin, are etiologic factors of the first degree. With the removal of these causes, the condition itself disappears, but, of course, not with equal rapidity in all children. If an intertrigo obstinately persists, in spite of proper external treatment, or if it reappears over extensive areas upon the slightest provocation, it should always arouse suspicion, for which experience affords ample ground that it is an early indication of the presence of exudative diathesis and, as such, is apparently as important as the development of eczema or the unexpected appearance of strophulus.

An important and significant external influence which, in the presence of an existing predisposition, very often causes the development of dermatoses, is scratching. The dermatosis in this case is not always and necessarily produced by the transmission and implantation of pus cocci, it

results rather from the sensory irritation caused by the scratching. This is very clearly noted in infants affected by constitutional eczema of the face; when, as a result of persistent scratching, new eruptions appear in various parts of the body, until the entire skin is finally covered by constantly enlarging and multiplying patches of eczema. In urticaria, this is even more clear than in eczema. How apparent then is the importance of the prevention of scratching in these skin diseases, even though such prevention requires the exercise of force.

Although scratching of the eczematous skin be prevented as far as possible, other external sources of irritation arise which may affect the skin in a similar manner and serve to spread the dermatosis. Thus, the tense vesicle itself itches, probably because it causes pressure upon the nerves in the deeper tissues. So also the dry seborrhœic scales, lying upon the exposed corium, excite itching. Bacterial products, always present in large quantities, cause irritation; while in weeping eczema the exudate flowing over the papillæ is intensely irritating. It is incumbent upon the clinician, therefore, to guard against these external injuries as far as possible and the local treatment, mainly directed to this end, is theoretically inspired and practically justified by the object in view.

The itch-mite and the head louse are certainly not regarded as the causative factors of an extensive dermatosis on account of any toxin they excrete or even because of their bites, but chiefly on account of the unbearable itching to which their host is subjected. A general reflex eczema of the scalp, which may be diagnosed almost at sight, and virtually represents a distinct disease in itself, appears only after the finger-nails have thoroughly lacerated the skin.

Doubtlesse exposure to light, temperature, wind and other climatic influences, also plays an important part in the causation of skin disorders. It may be that the predilection of infantile eczema for exposed parts of the body is relational to these influences. The peculiar sensitivity of the cutaneous vasomotor mechanism again comes into consideration at this point.

With the superficial and gross picture of these dermatoses of childhood, contrasted in our mind with the still imperfect knowledge we possess of their underlying conditions, it should be apparent that the three cardinal guiding points, predisposition, internal causation, and external injury, must always be borne in mind. It is especially necessary that we remember and be governed by these considerations in the treatment of these diseases.

ECZEMA

Eczema is a mild catarrh of the skin, a superficial cutaneous inflammation with very active serous and cellular excretion. The inflammation arises in the corium, and in distinction from other forms of dermatitis, begins in small punctate discrete foci. The process rapidly extends to the epithelium and the small primary lesions appear on the surface in the form of individual papules or vesicles. These initial forms are essentially characteristic of eczema, but the picture changes easily and rapidly. The

individual foci become confluent and soon present an extensive inflamed surface. Still at some point in the periphery, where the process is less advanced, it is usually possible to find one or more of the primary eczema papules. Discrete papules or vesicles, superficial extension and itching, are the pathognomonic signs of the varied group of skin affections which we term eczema.

In the course of an eczema, persisting unrestrained for a long time, it is possible to distinguish various stages. Theoretically, their definite systematic arrangements has no great value. The local treatment, however, is so dependent upon the stage of progress at which the eczematous inflammation is found, that, for purely practical purposes, it appears necessary to recognize and discuss these several divisions of the process, in the order of their development:

1. The erythematous stage, *eczema erythematosum;* the skin being slightly reddened, edematous and tense.

2. The papular stage, *eczema papulosum;* marked by the appearance of small reddish papules upon the skin, which they become confluent and form slightly raised plaques.

3. The vesicular stage, *eczema vesiculosum*; showing the development of a serous exudate in the papules.

4. The impetiginous stage, *eczema pustulosum*; with secondary infection and suppuration of the vesicles.

5. The weeping stage, *eczema madidans;* when the pustules rupture or are scratched open, forming many small openings in the horny layer of the skin, or the entire horny layer is loosened so that the rete lies exposed. In consequence, there is marked weeping on the surface, a fact which suggests the name.

6. The encrusted stage, *eczema crustosum;* the exudate drying rapidly, in so far as it is not removed, and resulting in the formation of crusts and scabs.

7. The squamous stage, *eczema squamosum;* when, in consequence of excessive cell proliferation, an increased desquamation of the epithelial cells occurs.

As a rule, eczema squamosum is the terminal stage of the disease and in wholly uncomplicated cases tends to healing without scar formation.

Eczema may go through all these stages in the order recited. In other instances the disease may pass directly from the first or second stage to the last.

The classification of the eczemas of childhood into acute and chronic forms meets with certain difficulties. Such a classification gives a wide range to the judgment of the individual observer. Usually an acute course marks all those forms of eczematous inflammation of the skin in the etiology of which external irritation is, by far, the most pronounced factor and which, being removed, the eczema heals quickly and completely. On the other hand, a more chronic course is to be expected of those forms of eczema which are chiefly dependent upon internal causes. Unless these are completely removed, and this is very frequently beyond the power of the physician—new irritations and reactions repeatedly complicate the eczema in an unlimited series of recrudescences.

As examples of eczematous dermatites running an acute course, we may note the various forms of intertrigo, eczema solare, and the so-called sudaminous eczemata. However it is better, perhaps, to classify these forms as artificial eczemata and to reserve the term acute eczema for those relatively rare cases of more or less widespread dermatitis, which appear very suddenly, often with initial fever, moderate itching, and marked redness; and in their further course soon disclose their eczematous nature. The points of predilection for these acute eczemata are the face, the nose, the ears and the region of the genitals. The turgescent redness and the fairly sharp margins which are peculiar to these circumscribed inflammations cause a very close resemblance to erysipelas. This likeness is further emphasized by the very frequent appearance, in some part of the inflamed area, of a large tense bleb filled with serous fluid. Nevertheless, the redness is not so intense nor the surface so glistening as in erysipelas, while the swelling is much more marked and the vesico-papular form of eruption is predominant. Its course is briefer, entirely favorable and it is hardly ever influenced to any extent by local treatment; *e. g.*, cold applications, lead water solutions or bland ointments.

INTERTRIGINOUS ECZEMA

This is frequently called intertrigo and is, especially in infants, a very prevalent skin affection.

The clinical picture permits division into two principle types:

1. The intertrigo of fat, overfed and frequently constipated infants.
2. The intertrigo of dyspeptics.

In the first group, it is not the extent of the inflamed areas which is marked, but rather the locations between the genitals and the thighs, in the groin, the axillæ, at the elbow and on the neck where there are heavy folds of skin produced by its excessive panniculus. The close contact of these fatty folds prevents the evaporation of the skin excretions and these parts remain constantly moist. If in addition the clothing is too warm and close-fitting, intertrigo can hardly be avoided in these "prize babies." This form of intertrigo has more of the character of a simple dermatitis. Papules and vesicles are not usually seen. Dyspeptic disturbances are not present in these cases but they commonly show an obstinate constipation.

The second form differs essentially, in that, although it may occasionally be seen in obese children, it is always accompanied by symptoms of dyspepsia or is, at least, ushered in with them. He who understands the character of thrush, will not be surprised to find that patches in the mouth frequently accompany this form of the skin disorder.

Diarrhœal, acid stools, an irritating urine frequently of ammoniacal odor, and clothing soaked by quantities of vomited sour milk are to be considered chief among the etiologic factors. Accordingly, the nates, the skin about the external genitalia, the neck (front and sides) and the chin are the sites of predilection. At the height of the inflammatory process, these affected areas are of a fiery red color, hot, glistening and swollen. They are tense and very sensitive to the touch. Usually papules and vesicles are

discoverable only at the beginning of the attack, and then soon disappear in the diffuse erythema.

The severest grade of this dermatitis is seen in pale, feeble and badly neglected infants. In these cases, an eyrthema is often seen continued over the back, abdomen, legs and heels; and if, in addition, there is an intertrigo of the neck, it is difficult to discover a spot of unaffected skin. If the papules and vesicles around the nates are scratched, numerous erosions and excoriations remain often in puzzle-shaped areas after the diffuse inflammation has subsided.

It should be noted that neither this condition, nor the round, light red, scaly, somewhat indurated plaques, which sometimes persist over the area of a previous intertrigo (Finkelstein), the so-called plaques erosive, or *erytheme syphiloide posterosiv*, of the French authors, have any connection with lues.

In the treatment of intertrigo, far-reaching prophylactic measures are, of course, the most important thing and consequently the condition is very uncommonly seen in properly conducted institutions for infant care. Proper methods of feeding play a very essential part in its prevention. This is also true of already existing intertrigo, especially of the second form which is found in combination with dyspepsia. If we are successful in removing the digestive disturbance, the intertrigo also quickly disappears. In weeping intertrigo, the influence of the dietetic treatment may be assisted by lightly painting the surface with a 5 per cent. solution of silver nitrate, once each day, and by the free use of dusting powder. The application to the affected areas of a zinc paste is also to be recommended in spite of numerous theoretical objections urged against it. It should be employed, at least, during the night in order to protect the inflamed parts from further irritation. Inasmuch as intertrigo corresponds markedly to the seborrhœic eczemas of adults, antiseborrhœics such as sulphur, resorcin and salicylic acid are of value. After application of silver nitrate, the following salves will be found of value; 1 per cent. acid salicylic, 3 per cent. lac sulphur in either petrolatum or modified zinc paste, as

Acidi Salicylic		0.3
Sulphuris-Lac		0.9
Zinc Oxidi		
Amyli	āā	5.0
Petrolati Flavum		20.0

If large areas of the corium are exposed, treatment similar to that used in burns of the second degree is indicated. Lead water fomentations and linimentum calcis are very useful for this purpose. Wet packs of 2 per cent. resorcin are frequently very valuable.

Sometimes even these measures do not give the desired results. The intertrigo proves very obstinate. On the neck, over the nape of the neck and on the nates it may involve extensive areas which are often very sharply marginated. In some regions, especially over the flexor surfaces of the elbows, the eruption is likely to become chronic. Almost always there is an extremely marked tendency to desquamation and seborrhœa. The in-

flamed areas of the skin are covered with numerous and extensive lamellar scales and with seborrhœic exudates which on the scalp often form a thick cap-like crust. Children, so affected, are delicate and pale, of deficient turgor, of flabby musculature and show insufficient gains in weight. They are almost always to be classed among the subjects of exudative diatheses; and in their treatment much more may usually be accomplished by dietetic measures than by external treatment alone. The beginning of such intertrigos is almost always to be traced to an acid dyspepsia.

ERYTHRODERMIA

Several years ago, Leiner described a peculiar general dermatosis under the name of desquamative erythrodermia. This condition has many points in common with the form of intertrigo just described; and should probably be regarded merely as its extreme type. It consists in a general

Fig. 216.—Desquamative erythrodermia (from intertrigo) moderate degree, complete recovery during second week. Breast-fed infant.

inflammation of the skin, with intensive desquamation of the epidermis and a very marked seborrhœa of the scalp. Leiner leaves the question of the etiology of this condition an open one, but suggests the hypothesis that it is an auto-toxemic erythema doubtless closely related to the intestinal disturbances always found in these children. The preponderance of breast-fed infants among them and the high mortality (one-third of the cases, Leiner), is remarkable.

If careful inquiry into the history of these children is made, it will be found that in almost every case, the condition is preceded by diarrhœa and intertrigo, and usually accompanied by thrush. The only peculiar feature is the extremely rapid spread of the dermatitis over the entire body and the remarkably intense general seborrhœa which seems, indeed, to give its clinical picture something of an individuality.

The disease is to be considered a general dermatitis following intertrigo which may reach this extreme degree in children with a distinct status seborrhœicus. The lack of discrete papules is not sufficient reason for

excluding entirely this dermatosis from the group of eczemata, since we so often miss the primary element of eczema even in the course of the usual intertrigo.

If the dermatosis occurs in the breast-fed infant, the first indication is for mixed feeding. Good results are usually obtained with milk and gruel mixtures or with buttermilk. The main object of the treatment, in fact, should be the relief of the dyspepsia. Beyond this, we should employ measures to soften the dry scaly skin. This may be readily accomplished by means of oil packs. Later on, the parts should be bandaged with zinc oxide in oil, cod-liver oil, or zinc ointment; and, still later, dusted with talcum powder. The dermatosis heals with relative rapidity and without leaving any traces whatever upon the skin. So far, the writer has not seen any fatal cases, although this may be a purely accidental matter.

CONSTITUTIONAL ECZEMA OF INFANTS

The severe seborrhœa, which at the height of the disease covers the bright red skin of erythrodermic children with innumerable scales, and the thick seborrhœic crust on the scalp, suggest an abnormal constitutional quality as the basis also of this type of dermatitis. This constitutional anomaly is particularly apparent in the very common and clinically important group of infantile eczemata which, as a matter of fact, have long been designated as of constitutional type.

According to Feer, two clinical varieties of this condition may be differentiated: (1) The weeping, crusted eczema of the head; and (2), the disseminated dry eczema.

The first form, by far the most common, affects chiefly fat, overfed children, while the second affects chiefly emaciated and sickly children. All infantile eczèmas cannot, of course, be classified definitely under these two primary types which, strictly speaking, represent the terminal conditions in an uninterrupted course. Transitions from the first into the second form appear quite commonly. Nevertheless, this classification has a certain distinctive merit since the two groups have a different prognosis and the dietetic treatment with the first form seems to give better results than with the second.

In the weeping crusted eczema of the head the eruption is, for a time at least, confined strictly to the face and scalp. The skin of the rest of the body is exceptionally free from any efflorescence, pink and velvety, giving a distinct contrast to the face where the closely crowded eczematous crusts leave hardly any unaffected area.

The most frequent starting points of this eczema are two local seborrhœic processes, a seborrhœa of the scalp and the dry scaly lesions of the cheeks. From here the eczema spreads more or less rapidly to the back of the neck, the temples and the forehead. The initial papules and vesicles are scratched open or burst, and through an inevitable secondary infection are changed to pustules. These dry rapidly, scab, and present the picture of an impetiginous or crusted eczema. The secretion from the deeper layers continues, the crusts are sloughed or torn off by scratching, and here and

there the bright red, weeping and bleeding corium lies exposed. In mild cases, the nose, mouth and chin are not involved; but in others the crusts are especially thick about the margins of the mucous membranes, and then the lids are often so markedly affected that the eyes can hardly be opened.

As the disease progresses, the lymph nodes of the submaxillary region and of the anterior and posterior cervical chains regularly become enlarged. Suppuration is, however, quite uncommon. In children who are given to much scratching, separate placques of eczema may appear later upon the extremities and the trunk; but even in these cases the eczema of the head remains the most conspicuous feature.

This head eczema occurs in breast-fed, as well as artificially-fed infants and usually during the first half-year.

From the prognostic viewpoint it is instructive to note that in some countries this exanthem is known as "the forty weeks eruption," because, as a rule, it lasts for that length of time. It is well, therefore, to prepare the parents for a long period of laborious care and patient waiting.

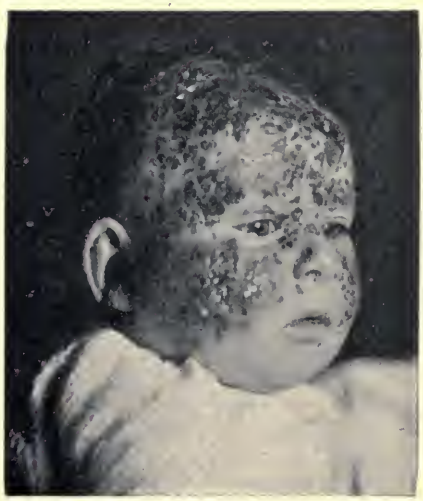


Fig. 217.—Crusty, moist eczema of the head. Conjunctiva and nose not affected in distinction from scrofula. (Gisela Children's Hospital, Munich, Prof. Ibrahim.)

Disseminated dry eczema is found almost exclusively in bottle-fed infants who do not show any special tendency to put on any great amount of fat. They are often of a pale, emaciated and flabby type. The condition is one, which appears at a later period than eczema of the head, and usually in the latter half or toward the end of the first year. The head is not entirely free from the disease even in this form, but the most characteristic feature is seen in the presence of numerous disseminated, dry, indurated and fairly well marginated plaques on the trunk and on the extremities. This form of eczema is often exceedingly persistent and from the standpoint of therapy more difficult to influence than the eczema of the head. This depends probably not so much upon the local process as upon the constitutional condition which seems to be worse than in those children subject to the head eczema.

The metabolism of eczematous children shows no characteristic deviations from the normal. Very recently, however, certain positive facts have been recorded, which are of importance. It has been shown, in the first place, that the limit of assimilation of sugars, and especially of maltose is too low, and that a diet rich in carbohydrates readily leads to alimentary glycosuria (Aschenheim). Further, an increased and abnormal tendency to water and chlorine retention, followed by an excessively rapid excretion of the retained chlorine upon the reduction of the chlorine in the food has been

demonstrated (Freund and Menschikoff). This last observation agrees with that of Czerny that in exudative diathesis there is a congenital defect in the chemism of those tissues which permits a great variation in the water content.

Clinically, eczema is often found to disappear either entirely or in part, during rapid losses of weight, especially during the course of acute febrile diseases. This phenomenon may be attributed, in part, to a drying of the skin as a result of the large loss of water; and still further to the influence

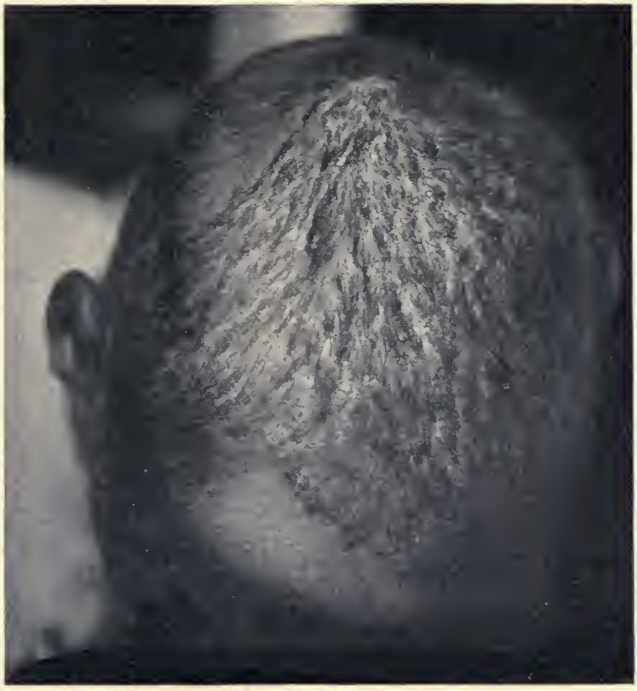


FIG. 218.—Seborrhœic dermatitis of scalp (courtesy of Richard L. Sutton).

of the fever itself in diminishing, in large measure, the disposition of the skin to inflammatory reaction.

The supposition that overfeeding plays an important rôle in the pathogenesis of infantile eczema is not a new one. This is suggested indeed, in the application to its cardinal features of the terms "crusta lactea" and "milk rash," by which physicians and laity alike have designated infantile eczema for centuries; terms which doubtless arose from the conception that the eruption stood in close relationship to an excessive milk diet. Czerny, in recent times, was the first to recall attention, with some emphasis, to this fact. It is possible that quality as well as quantity is to be considered in certain individuals. Nothing definite of this is known however even to-day. Perhaps one must also consider the correlation of the various components of the food especially the fat and salts.

Dietetic Treatment.—In the study of the dietary, two principal points should be borne in mind: (1) The avoidance of excessive increases in weight; and (2) the eliminations, so far as possible, of milk from the food.

It is self-evident that these precepts are more easily obeyed with fat overfed children who have passed the first year, than with young and emaciated nurslings. In every instance the aim should be to reduce the quantity of milk to the minimum compatible with the individual welfare, replacing the calories thus sacrificed by the addition of gruels and flour. The temporary use of skim-milk is to be recommended. In children who are in the second half-year, milk may be left out of the diet for several weeks without injury, substituting a varied menu of flour soups and gruels, fruits and fruit juices and fresh vegetables.

Fig. 219.—Disseminate eczema (en plaques).

Eggs are to be prohibited (Czerny), and unfavorable results are reported with broths (Feer).

Breast-fed infants should be permitted to continue nursing. For fat babies the number of feedings should be reduced to four. After the third month, the breast should be given only three times a day, adding gruel to the diet, under careful control by daily weighings.

All these dietetic expedients must be undertaken with extreme care in the weak, emaciated, bottle-fed infant; for the treatment can hardly be considered successful, though the eczema disappear, should the child eventually succumb.

The local treatment as suggested in the introductory chapter, is an essential part of the management of eczema. Its detail depends entirely upon the stage at which the disease is presented. Before taking up its several features, however, it may be well to cite the principles of especial practical importance upon which the local treatment of the disease is based.

a. Scratching must be prevented as rigidly as possible. It may not be possible to prevent scratching entirely. The infant will inevitably find new ways of gratifying the irresistible impulse. Nevertheless, all possible precautions should be taken. The finger-nails should be trimmed as short as may be; the hands may be bandaged or covered with mittens. Splints, over the elbow-joints; the fastening of the hands to the bed; the pinning

of the sleeves to the bedclothes, may prevent the child from scratching its face. Care must be taken to prevent scratching even when changing the bandages.

b. The eczematous areas of the skin should be cleansed either with an alcoholic solution with 2 per cent. of salicylic or boric acid, or with petrolatum. (Acid salicylic 2.0, spiritus vini rectificatissimus 60 per cent., 100.0.) Water and soap are often irritating. The chief objection to their use is that the skin cannot be thoroughly dried after their use. The healthy skin can be washed and dried, as usual.

c. The application of ointments and pastes is not in itself sufficient. They must be covered with a firm bandage or, in mild cases, may be thoroughly covered with powder.

FIG. 220.—Face mask and arm cuffs for treatment of facial eczema.

The ointment or paste should be thickly applied upon a very soft sterile cloth, which may be held in place by the ordinary gauze bandage. Sterile absorbent lint is best used with the ointment applied to the fuzzy side.

d. So long as the desired results are attained with any remedy it should be continued. In fact, it is better not to change a chosen course of treatment any oftener than is absolutely necessary.

e. In a general way, the direction to treat weeping eczema with drying methods (pastes, silver nitrate, etc.), and the dry eczema by moist methods (fatty ointments, etc.), serves usefully. It is a mistake to apply the latter to exuding surfaces; the exudate collects beneath the covering ointment and is the more irritating because of the retention.

f. Tar should be applied with great caution.

It is best to test the preparation first upon small areas. The remedy is contraindicated as long as the eczema is in the early papulo-vesicular or encrusted stages. This is especially true of eczema of the face. Recently crude tar as obtained directly at the gas house has come into use and it is rather an exception to this rule, inasmuch as it can frequently be very successfully applied to acute eczemas. It may be used as an ointment:

Crude Tar	2.0	Tarrolin	4.0
Zinc Oxide	2.0	Yellow Petrolatum	16.0
Starch	12.0		

or painted on daily in the following mixture.

Crude Tar Acetone Collodion āā

In young children with generalized cases, it is well to go cautiously.

g. If the eczema has, clinically speaking, disappeared a rational after-treatment of the formerly affected skin areas should be undertaken.

The avoidance of mechanical irritation; the careful use of soap and water; the brief employment of bran baths, followed by careful applications of cold cream, promote the gradual healing and hardening of the skin.

In the initial stage of eczema, cleansing with alcohol and thorough powdering with the stearate of zinc is sufficient. Where powdering is indicated, a practical application is zinc oxide, amyli āā 20.0, glycerini, aquæ dest. āā 40.0.

If the patient is first presented with an impetiginous encrusted eczema, as is usually the case, it is necessary, first, to remove the crusts and scabs. This is readily accomplished by the use of an oil-cap on the scalp and of oiled bandages on the face. Olive oil is usually recommended, but the ordinary cotton-seed oil is satisfactory. The most important thing is that it be correctly applied. A piece of absorbent cotton or a strip of flannel or, preferably, sterile gauze is saturated with oil and applied over the crusts to be softened. This is in turn covered with a thin layer of dry cotton and with rubber tissue or oil silk. The head is covered with a close-fitting hood and the face is protected with a gauze mask. The oil dressings are changed each morning and evening. Softening of the crusts proceeds rapidly and then they may be removed readily by means of a cotton-mounted probe or a pair of blunt tweezers.

Some authors recommend the use of hydrogen dioxide (3 per cent.), for the removal and deodorization of the crusts, while others prefer the following prescription: ℞. Acidi salicylici 2.0 (℥ss); olei ricini, 40.0 (℥x.); olei oliv. ad 100.0 (℥iiss).

Unguentum diachylon may also be applied thick, it will not only soften and remove crusts but frequently has a beneficial effect upon the eczema itself. It should be changed twice daily.

When the eczema has been cleaned in this manner, the actual treatment, after the following described methods, is to be inaugurated. If not only the cheeks and forehead, but also the mouth and chin are affected, the use of a so-called eczema mask made of a piece of sterile absorbent lint or fine sterilized muslin is to be recommended. The lint or muslin may be laid over the child's face and the eyes, nose and mouth marked in with a pencil. Holes are then cut in the marked points so as to leave eyes, nose and mouth uncovered. Tie-strings may be sewn to the mask to hold it in place.

If we have to deal with a weeping eczema, an eczema madidans, very mild measures for allaying the inflammation must first be employed with the intention of gradually drying up the secreting surfaces.

For this purpose, dressings moistened with a solution of aluminum acetate (1:10), or with the usual boric acid solution (4 per cent.), are very helpful. Later, when the inflammation has subsided and the secretion has diminished the application of a paste, consisting of zinc oxide, talcum, lanolin, and petrolatum, in equal parts, will serve to dry up the secretion still more. Fox paste may be used here with benefit. A moderate amount

(5-10 per cent.), of the triacetate of pyrogallol (lenigallol), added to this ointment may act favorably. The itching may be allayed by adding 1 to 5 per cent. of tumenol or naphthalene (naphthalene, adeps lanæ), āā, 50.0 (℥xii), acidi borici, 10.0 (℥iiss); zinci oxidi, 20.0 (℥v). If the secretion persists, the surface should be painted with silver nitrate (1 per cent.), once or twice a day; or an application of gauze moistened with a silver solution (1-10,000), may be left in place for an hour or so with good effect. It should be remembered this may stain. In the intervals the surfaces may be covered with powder or paste.

When the secretion has ceased and the eczematous areas appear perfectly dry, fatty applications are indicated. This is very satisfactorily carried out by equal parts of zinc oxide and olive oil. A five per cent. boric acid ointment may be used instead.

Emphasis must be put upon the fact, however, that this therapy, simple and rational as it appears, is completely successful only in occasional cases. In hospitals, with skilled attendants and unremitting care, the results are more satisfactory than in the home. Usually a point is reached at which improvement ceases and even where exacerbations may occur. In the latter event, the treatment must be patiently repeated from the beginning. If, however, a mere arrest of improvement occurs, but without any acute relapse, the eczema may be said to have taken on a more chronic character and treatment with some tar preparation should be inaugurated, without delay. It would be a great mistake at this juncture to stop external treatment. The entire course of treatment would have been undertaken in vain. The disease process still persists in the deeper tissues; the skin is usually still hyperemic; is densely infiltrated; and the itching continues.

The following will serve as an example of a mild tar ointment, suitable for this phase of the disease: ℞: Zinci oxidi, talci āā 10.0 (℥iiss); petrolati 20.0 (℥v); oleum picis liquidæ 0.5 (minims viii), or less. The anthrasol zinc ointment, anthrasol 1.0 (minims xv): unguenti zinci Wilsonii, 30.0 (℥i), may be substituted.

Tar is also the most useful of applications in the disseminated form of chronic eczema, in eczematous placques and, therefore, in the secondary form of the disease. Usually, it gives results rapidly, while other remedies afford no improvement. The author prefers the old Wilkinson's sulphur ointment. This may be accompanied by the use of sulphur baths and arsenic internally. The diachylon ointment prepared according to the old formula of Hebra, without lavandula, is a remedy with which very rapid improvement takes place, excepting with very sluggish, old eczematous placques.

Experience has shown that children affected with eczema sometimes die suddenly without any definite or apparent cause. We speak, in fact, of death from acute eczema. Pale and pasty infants are especially endangered, and they show an alarming rate of mortality. Should such a misfortune occur during a particularly energetic course of treatment, the popular tendency is to lay the blame to the effect produced in "driving in the eruption." The writer is not at all inclined to regard such forced conclusions as entirely senseless or foolish, since we know nothing of the real

significance of eczema as a natural process of reaction. The fact that in these cases a status lymphaticus is often, although by no means always, found at autopsy, is very significant indeed, although it affords no satisfactory indication of any relationship between the eczema death and the sudden cessation of the superficial efflorescence. It must be remembered, too that sudden death may happen in eczematous children from extrinsic causes altogether unrelated to the treatment of the eczema. It has been known to occur, for instance, during the application of a sweat pack.

URTICARIA

Under this caption are grouped a number of diseases which are rather variable in their external manifestations. It will include ordinary urticaria, acute circumscribed edema of the skin (Quincke), strophulus and prurigo, because, from the first, a proper emphasis should be put upon their etiologic relationship. The sufferer from any of these diseases always exhibits a marked and excessive variability of vascular tone, due to an increased irritability of the vasomotor mechanism, accompanied, probably, by abnormally increased permeability of the vessel walls. As a result, the patient reacts to slight stimuli with the appearance of true urticarial wheals —which represent clinically the fundamental type of lesion of this large group of dermatoses.

Fig. 221.—Eczema of mouth and chin. (Courtesy of Richard L. Sutton.)

The structural feature of the urticarial wheal is found in a marked injection of the papillary layer and the corium, which is rapidly followed by serous infiltration. The elevation of the skin resulting from this appears red (*urticaria rubra*), when the flush of the injected vessels of the papillary layer is visible through the epidermis; it appears white (*urticaria porcellanea*), when an extensive serous exudate in the rete forces the blood out of the underlying papillary layer. If the extravasated serum is equally diffused over an extensive area and if it penetrates also to the deeper subcutaneous tissue, the entire area becomes edematous, the feature of circumscribed edema. If the capillary injection is more intense and the exudation is very slight, a flat, very slightly raised redness appears (*erythema urticatum*). If the centres of the wheals are changed by induration, to an inflammatory papule, strophulus results (*urticaria papulosa*, or *lichen urticatus*) which, if the irritatitive agent persists, may go on to the development of a typical and, at times, a very obstinate dermatosis with its indurated papules, characteristically localized on the extensor surfaces of the extremities (*prurigo*).

A very characteristic clinical sign of all the diseases of the urticarial

group is the itching. Peculiar as it is in a degree to all these superficial eruptions it reaches its greatest intensity in prurigo. The several forms of urticaria recur readily. While, however, urticaria, circumscribed edema of the skin, and strophulus are, generally speaking, of an extremely transitory nature, true prurigo is distinguished by its eminently chronic course.

From the case-history we very frequently learn that the urticarial affection is familial; that brothers and sisters, parents, grandparents or other blood relatives have suffered or still suffer from similar affections. Not infrequently the condition has been preceded, in early life, by obstinate eczema of the face and head. Such experiences and many other established facts lead us to suspect the existence of a congenital factor, an hereditary constitutional anomaly which enters into the etiology of the urticarial dermatoses and expresses itself in a marked predisposition to angio-neurotic inflammation.

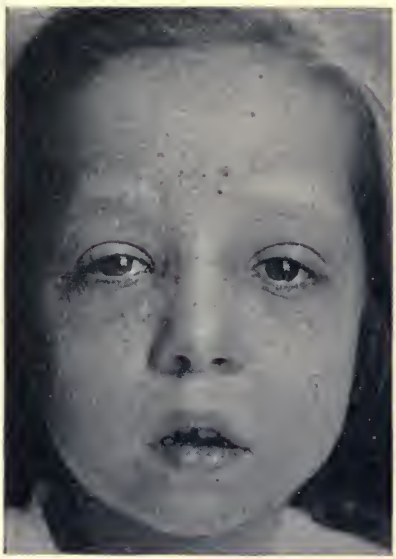


Fig. 222.—Anaphylactic eczema. (Courtesy of Richard L. Sutton.)

Simple Urticaria

Urticaria, with its multiple transitory eruption of wheals is a very common dermatosis of childhood. Sometimes the entire body is thickly covered by the eruption. The face may be greatly distorted, swollen, mottled with red blotches, cyanosed and edematous.

If neighboring wheals coalesce, large raised plaques, often three or four inches in diameter, are formed, which present either a bright red or a more whitish color.

The eruption may or may not be accompanied by fever. Usually the urticaria appears unexpectedly overnight in an apparently healthy child and disappears with equal rapidity, possibly during the very same day. In other instances, however, the rash is preceded by an indefinite prodromal period of several days, marked by vague symptoms of malaise, gastric fever, etc., which continue until a general urticaria, with intense itching, suddenly appears and clears up the indefinite disease-picture at once.

Doubtless there is an urticaria of the mucous membranes essentially similar in nature to the external cutaneous disease. This runs its course with severe but rapidly passing symptoms of irritation and swelling in the intestinal tract, the pharynx, the larynx and bronchi. A typical example of this peculiarity is seen in the acute asthmatic attacks suffered by certain predisposed persons, attacks which occur suddenly after the ingestion of eggs, lobster, strawberries or some other particular food, and are followed immediately by the customary urticarial eruption on the skin.

We have no definite knowledge of the actual cause of urticaria. Doubtless stimuli of alimentary toxic origin, and from colds, exciting the urticarial skin reaction by way of the sympathetic nervous system, play an important part. Urticarial serum rashes and cow-pox exanthemata frequently appear respectively after the injection of a foreign serum (antitoxin serum), and after vaccination, and most probably may be attributed to the action of some toxic substance liberated by the combination of antigen with antibody (the so-called anaphylatotoxin). To what extent toxins of gastro-intestinal origin are connected with the causation of urticaria is an open question. Many authors consider them of great etiologic importance, especially since children who suffer with urticaria are very often obstinately constipated.

For acute general urticaria, rest in bed and a dose of castor oil should be prescribed. The itching may be allayed by washing the skin with a weak alcoholic solution of menthol, or with vinegar and water (equal parts) and by the subsequent use of cooling ointments. The inauguration of a dietary consisting largely of vegetables, as great a reduction of milk as possible and the avoidance of eggs may prevent recurrences. Baths with sodium bicarbonate, salt, borax, ammonium chloride one-half ounce to one ounce to bath.

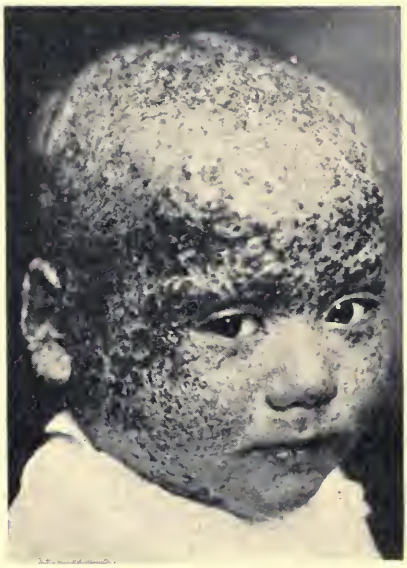


Fig. 223.—Infantile eczema. (Courtesy of Richard L. Sutton.)

Acute Circumscribed Edema of the Skin (Giant Urticaria)

This condition is very closely related to simple urticaria. In rare cases it occurs as a distinct disease unaccompanied by urticaria. In such an instance it is of a typical familiar character. The favorite site of this acute edema is on the face and particularly in the loose connective tissue of the eyelids. Usually the symptoms completely disappear in a few hours. Coincident edema of the pharynx or larynx may cause serious difficulty in swallowing and breathing.

Some authors claim to have obtained rapid relief with cold sitz-baths or ice-packs.

STROPHULUS

In infants and young children urticaria very frequently takes a papular form (strophulus, lichen urticatus, urticaria papulosa). If opportunity is given to observe an early case, one sees clearly how, at the centre of the wheal or in the smaller urticarial macules, hard globular papules of dark red

color, begin to arise. After a day or two the redness and the urticarial base disappears, the papules become paler and clearer and persist, then unchanged for a time, as small waxy hard hemispheral papules. At times a small vesicle appears at the apex, which may under certain circumstances become purulent (*strophulus vesiculosus* or *impetiginosus*). At this stage,

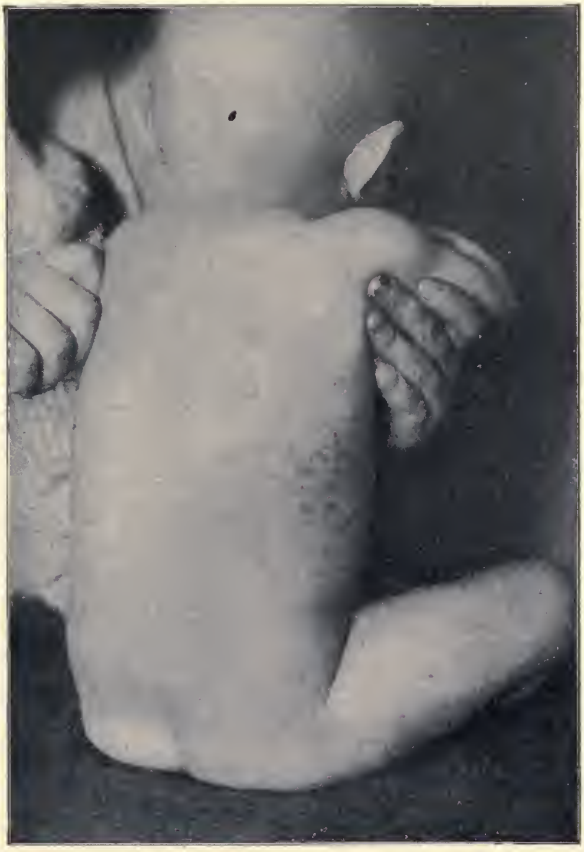


Fig. 224.—Lichen urticatus or strophulus. Arranged in groups. (Dresden Infant's Home, Prof. Schlossmann.)

the urticarial nature of the malady is hardly determinable unless, as frequently happens, new urticarial wheals appear on other areas of the skin.

The outbreak of strophulus is rarely general. Usually, the exanthem is confined to certain areas of the body. Its fairly dense grouping on the arms and legs and especially in the region of the joints is quite characteristic. Sometimes the strophular eruption follows quite closely the course of an intercostal nerve. The intense itching which attends the eruption is typical. As a result the strophular papules are often severely scratched and covered with small blood crusts.

What has been said of the etiology and treatment of urticaria is also

true, in a general way, of this condition. The frequency of occurrence of the disease in this form in very young children may be dependent upon structural peculiarities of the skin. This may readily explain the noticeable fact that a post-vaccination exanthem, occurring during the first year of life, very frequently takes on the form of strophulus.

The treatment of strophulus must be chiefly dietetic. In infants, one should radically reduce the quantity of milk as recommended in eczema. In children past infancy all milk and eggs should be absolutely prohibited. Good results have been gained, in many cases, by following this principle of treatment.

PRURIGO

The term prurigo is applied to an intensely itching, chronic, papular eruption, which localizes itself in typical cases, chiefly on the extensor surfaces of the extremities. The primary lesion of the exanthem occurs as the so-called prurigo papules. These are hard elevations in the upper layers of the skin, varying from a pinhead to a hemp-seed in size, of either a pale red or a whitish color. Usually as the result of scratching, they show small blood crusts at their apices. If the hand is passed over the surface of the affected skin, a feeling similar to that of contact with a fine grater is noted.

The clinical relationship of prurigo to urticaria is shown by the fact that the former almost always arises from the previously described lichen urticatus. Strophulus, or lichen urticatus, indeed, may be looked upon as an earlier stage of an ensuing prurigo. Furthermore, one frequently sees upon the site of an advanced prurigo repetitional crops of urticarial wheals and lichen papules appearing spontaneously or as a result of scratching.

The onset of a prurigo may always be traced back to early childhood. The transitory strophulus shows a tendency to persist, and the temporary angioneurosis passes into an essential dermatosis. While the earlier eruption is diffused over the entire body, in this chronic stage with the appearance of the small inflamed prurigo papules, it is peculiarly confined to the extensor surfaces, particularly of the legs, where it persists unchanged for a long time.

The nodules, frequently scratched and secondarily infected, involve the regional lymph nodes, which often become greatly enlarged and form the so-called prurigo bubos. These may be easily palpable and even visible in the groin. The coincident appearance of acute hemorrhagic nephritis is not at all uncommon.

In childhood we usually meet with a mild form of prurigo, the so-called prurigo simplex or mitis, which is fairly amenable to external treatment. The unconquerable, troublesome and much dreaded prurigo ferox of Hebra, appears, probably, only at a later period.

The causes which underlie prurigo, are, of course, identical with those which produce urticaria. An important causative influence is undoubtedly to be found in inadequate care; since, otherwise it would be hard to under-

stand why so many chronic cases recover completely with no other treatment than that of complete rest in a clean hospital bed.

External treatment is directed chiefly to the maceration and softening of the indurated epidermis. Sweat-producing packs are extremely useful and applied locally to the legs and arms may be continued for several hours at a time during a period of four days. Cloths, moistened in a warm

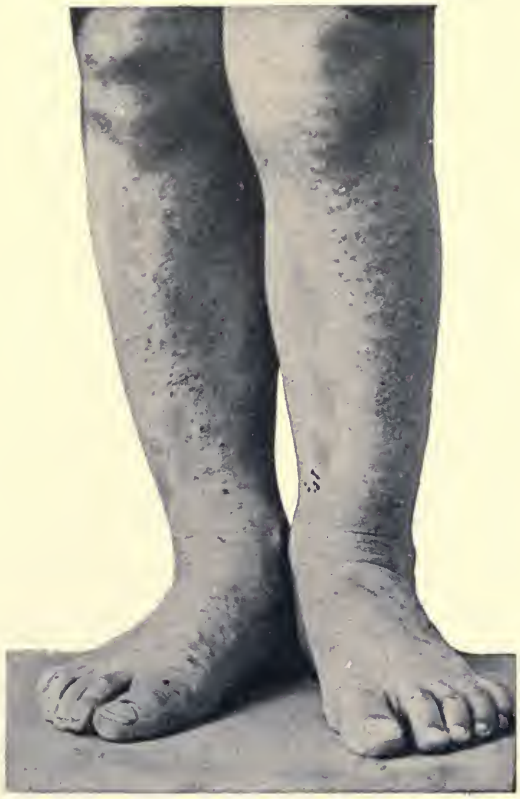


FIG. 225.—Prurigo. Area of predilection of the numerous scratched nodules on the extensor surfaces of the extremities.

solution of salicylic acid (ʒi to O ii), and covered with flannel may be satisfactorily used for this purpose. During the intervals between the warm fomentations, the affected skin areas should be protected with a bland ointment.

The highly praised treatment by intestinal disinfection can hardly be considered efficacious. If it is considered possible to improve the general condition of the patient by the use of iron preparations or the so-called roborants, and if one believes that he may "correct metabolic errors" by the use of these remedies, they may be tried.

MULTIPLE ERYTHEMA

(Erythema Multiforme)

A large variety of cutaneous reactions are classed in the group of erythemata. According to their morphologic characteristics and the stage of development in which they appear, they are termed erythema papulatum, tuberculatum, hemorrhagicum, contusiforma, marginatum, annulare, vesiculosum and iris. Erythema nodosum, which is often thought to require a special classification is also very closely related to this group of dermatoses.

Pathologically, an inflammatory process is found in all these cases. This constitutes a true dermatitis, occurring in foci in the skin and the subcutaneous tissues. In their later course, these foci may form fairly circumscribed papular or large nodular exudations. The old general name erythema is, therefore, not very appropriate, since the disease is really more than a diffuse reddening of the skin depending upon a pure hyperemia. For this reason, these erythemata, since the time of Hebra, have been differentiated from simple erythema by emphasizing the exudative process they involve and by distinguishing the group as *erythema exudativum multiforme*. From the purely clinical standpoint, many subdivisions of the erythemata may be made upon the basis of the numerous gross variations. In the first place, one may distinguish between the more diffuse general erythemata and the circumscribed localized forms. The former may resemble measles or rubella, or may even simulate the picture of a scarlet fever eruption. The careful observer, however, will usually note the more nodular or finely papular character of the individual lesions of the erythemata.

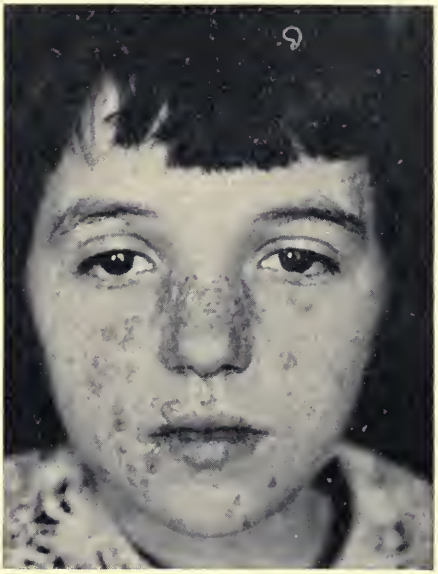


Fig. 226.—Erythema multiforme. (Courtesy of Richard L. Sutton.)

In the case of the localized erythemata, we again meet at least two principal types: (1) A form with an unmistakable tendency to superficial spread of the erythema; and (2) a form essentially circumscribed, in which discrete, nodular infiltration foci, varying from a hazel-nut to a pigeon's egg in size, occur in small distinctly limited areas (erythema nodosum). Especially remarkable is the typical localization of both of these erythemas on the extensor surfaces of the extremities; in a general way, the superficial eruption on the upper extremities particularly around the wrist and elbow joints, and the nodular eruption on the extensors of the lower extremities,

especially over the edge of the tibia. Both forms show a certain tendency to small hemorrhages, but this is more characteristic of the nodular than of the superficial type. If the nodules over the tibia are discolored by the extravasated hemoglobin, obviously they may be designated also as contusiform erythema, according to Hebra.

Presumably all these erythemata have a common pathologic basis in some noxious agent serving as a stimulus within the organism provocative of toxic reaction. Accordingly it is not surprising that the eruption is often accompanied by a more or less severe malaise and may at times be ushered in or accompanied by high febrile disturbance.

The noxious agent itself may be found:

a. In the toxic action of bacteria and bacterial products, *e. g.*, in septic erythema, erythema in the course of such diseases as cerebrospinal meningitis, rheumatism, and influenza.

b. In the toxic action incident to the binding of specific antigens and antibodies; *e. g.*, the erythema of serum disease, of post-vaccination, of active tuberculosis, or as a feature of the tuberculin reaction; erythema during measles and following scarlet fever.

c. In the action of a medicinal agent against which the patient has an idiosyncrasy, a so-called drug eruption.

It should be noted that the type of cutaneous reaction is by no means specific or constant. One and the same noxious agent may now produce one form of erythema and again a different type. Then, for instance, one may see, as a result of the percutaneous application of tuberculin, at different times, a general exanthem resembling scarlet fever or measles, a localized erythema of the flat superficial type or the nodular form, which, in one case recorded, was a typical erythema nodosum. Close relation apparently exists between erythema nodosum and tuberculosis. Children suffering with the former disease usually show an intense positive reaction to tuberculin; a behavior, however, which is not constant.

The relationship of erythema multiforme, especially in its localized forms, to rheumatic conditions is very distinct, but is by no means understood. Joint affections and muscular pains are not infrequently combined with erythema. Cases are common in the experience of every clinician which present, on first acquaintance, some form of erythema and reappear after a few years with peliosis rheumatica, with chorea, or with a systolic murmur audible at the apex. If the signs of erythema multiforme appear in the skin over an affected joint during the course of acute articular rheumatism the pain often disappears very suddenly.

In their differential diagnosis the infectious erythemata, the so-called fourth and fifth diseases present special difficulties, which at times, appear insurmountable if one cannot locate the source of infection or if they do not occur in the course of an epidemic.

Cool fomentations of a solution of aluminum acetate have a favorable affect upon the local inflammation. Acetyl-salicylic acid should be given in event of a rheumatic relation in the localized erythema.

IMPETIGO

This group includes impetigo contagiosa, pemphigus neonatorum, and dermatitis exfoliativa, all of which are contagious dermatoses caused by the pyogenous, staphylo- and streptococci. Their grouping is dependent upon their etiology.

Especially instructive are the comparisons to be drawn between the results of pemphigus infection in infants producing a typical impetigo in older children or adults. The evident factor of difference by which one and the same infection will, in the young infant, produce large pemphigus blebs or even extensive exfoliation of the epidermis (*dermatitis exfoliativa*), while in later childhood it causes only small impetiginous pustules, lies in the anatomic structure of the skin. In the delicate skin of the new-born an extensive separation of the thin stratum corneum occurs very readily; in the older and firmer cutaneous structures, on the contrary, the same exudative process loosens only a small area. Furthermore one sometimes sees true, small impetiginous pustules in the new-born, which show no tendency to superficial spread and, strangely enough, are without tendency to incrustation (*impetigo bullosa*).

IMPETIGO CONTAGIOSA

The primary lesion in impetigo contagiosa is the characteristic pustule. This is, at first, a small but rapidly growing purulent vesicle surrounded by a small areola of inflammation. The pustules soon rupture, dry, and are then covered with light yellow or brownish crusts. On account of the contagiousness of the process, the lesions of impetigo usually soon become confluent. On the face and around the mouth and nose they form wreath-like figures, or on the scalp, a dense encrusted mass. As a result of scratching, the pyogenic organisms are often carried to distant parts of the body and new crops of impetiginous pustules appear on the hands, arms, legs or trunk. The intra-scapular space alone is hardly ever infected since the child cannot reach it with its finger-nails.

It is a remarkable fact, that in spite of the demonstrated contagiousness of impetigo, and with exactly the same chance of infection, not all, but only a certain group of children are affected with the disease. It is probable that the germs of impetigo can invade only those skins which react to the irritation of the infective organism by a local inflammation, to which reaction certain individuals are especially and naturally predisposed. The pyogenic cocci thrive in the products of this inflammatory process and pustules result.

If the impetigo appears as an independent primary affection, the skin around the pustule shows little or no change. This gives the round crusts the appearance of being stuck on the skin. In secondary impetigo an entirely different condition is found, which often results from scratching or uncleanliness on a skin soil previously affected by eczema, urticaria, or strophulus.

A peculiar form of contagious impetigo is the ordinary ecthyma, which, contrasted with the conditions just described, seems to arise from the

deeper layers of the skin. A hard, tensely infiltrated inflammatory nodule of a bright red color appears on the skin. From this the pustule develops and usually passes through the same stages as an ordinary impetigo. The pustules of ecthyma show no tendency to group, but are always discrete. They are most frequently seen on the extensor surfaces of the lower extremities and on the nates. This form of eruption is most frequently seen following scabies. If scratched, it leaves distinct scars which persist much longer than the pale red spots following impetigo.

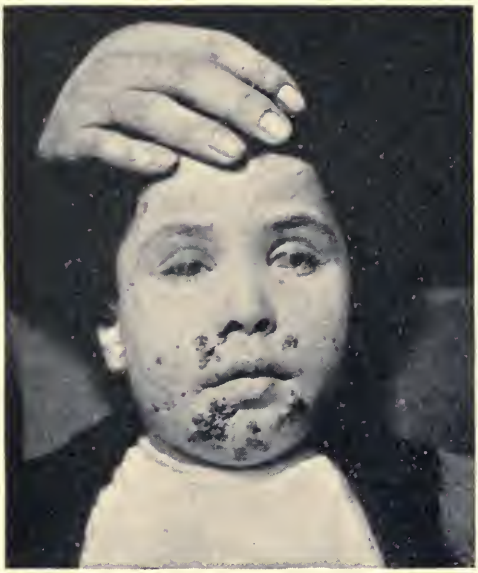


FIG. 227.—Impetigo contagiosa. (Courtesy of H. H. Hazen.)

In the treatment of impetigo excellent results are obtained by the use of oil-packs to soften the crusts followed by applications of the unguentum hydrargyrum ammoniatum (2 per cent.). If the pustules of impetigo are scattered over the body, the individual lesions may be covered with adhesive plaster containing ammoniated mercury. Under proper treatment impetigo disappears in a few days. Ecthyma is much more obstinate and the application to it of ammoniated mercury ointment should be preceded by the use of dressings moistened with a solution of aluminum acetate.

PEMPHIGUS NEONATORUM

This is a very characteristic skin disease of the new-born, accurately described as early as the beginning of the seventeenth century. It consists

FIG. 228.—Pemphigus neonatorum. Intact and ruptured vesicles. Favorable termination.

in the appearance of discrete vesicles, varying in diameter from one to three centimeters, filled with a slightly cloudy fluid and resting upon a normal mound or very slightly inflamed base. The epidermis over the smaller vesicles is tense, while that over the large ones is loose and rather redundant. Frequently the vesicles have ruptured and are empty when the patient is first seen. There remain red, circular or oval areas which are still moist or have dried up and are surrounded with the delicate whitish shreds of the ruptured horny layer.

This eruption of superficial vesicles will occur in children who are in perfectly good health. Any constitutional symptoms are of secondary nature or are unrelated to the skin condition.

Therapy.—Daily baths of a weak potassium permanganate solution should be followed by the free use of such an antiseptic drying powder as stearate of zinc.

DERMATITIS EXFOLIATIVA

Under this name von Ritter (1870) described a very peculiar exudative dermatosis which occurred in young infants and lead to extensive exfoliation of the epidermis. The disease appeared at that time in epidemic form in the Foundling's Institute at Prague.

This comparatively rare dermatosis may attack children in the very first week of life. It begins with a general reddening of the skin, first appearing on the face and especially around the mouth and often spreading rapidly over the entire body. It is always accompanied by the eruption of numerous small discrete vesicles. These areas of the epidermis then become undermined by a widespread edema which separates the superficial tissues from the corium, so that the former may be easily moved back and forth with the finger. The delicate outer covering is very easily broken, the horny layer is peeled off in large shreds and the inflamed rete lies bare.

Fig. 229.—Exfoliative dermatitis (Ritter's disease). Fourteen-day-old infant.

On the face, the edematous infiltration of the skin is primary. Radial fissures and deep rhagades are formed, especially around the mouth, presenting a picture which closely resembles that of lues, saving that it is much more pronounced and grotesque. A similar process may develop, at the same time, on the mucous membranes, the conjunctiva, in the mouth and in the anterior nares.

It is really remarkable that delicate babes are not more seriously affected from the beginning by this terrible dermatosis. In uncomplicated cases the course is entirely without fever and the nutritional functions are undisturbed for some time. Nevertheless, the prognosis, especially in artificially-fed children, is grave, since septicemia may very readily develop upon this specially favorable soil and may change the outlook very suddenly. According to von Ritter, the mortality is fifty per cent.

Therapy.—Fomentations of dilute aluminum acetate solution are lightly applied to the highly inflamed parts; or these may be painted with solution of silver nitrate (3 per cent.). One or two baths in a solution of tannin may be given daily. After thorough drying, the entire body is to be covered with dusting powder.

FURUNCULOSIS

Furunculosis of the cellular tissue occurring in older children does not differ, either in nature or treatment, from the same process in the adult. In infancy, however, we often see a very characteristic type of furuncle in the skin, presenting, in fact, a skin lesion peculiar to this age. These skin furuncles of infancy are superficial to the cellular tissue and are always multiple. Multiple furunculosis of the skin and multiple cutaneous abscesses, in infancy, are interchangeable terms.

Furuncles do not occur in healthy infants. Their presence is always a sign of illness and conclusive evidence that the natural resistance is markedly below par. The condition is frequently resultant from severe disturbances of nutrition.

An enormous number of abscesses may develop. In some instances a hundred or more have been seen. At first they may be no larger than a pea; persisting, there may be some as large as, or even larger than, a walnut. In this event they are apt to become soft and flabby. If they are punctured, pus and blood are often forcibly evacuated. They are commonly known as boils.

Clinically, two types may readily be distinguished: (1) The most common seat of the furuncles is over the occiput, the neck and the back; that is on those parts of the body which are exposed to friction and perspire more freely than others. At first, numerous small pustules appear, corresponding in site to the orifices of the sweat glands (Lewandowski's periporitis). From these pustules, abscesses later develop. Obviously an ectogenous mode of infection plays a most important part in such a process. At the same time the child so affected often suffers from disturbances of nutrition, although this is by no means invariably true. (2) In the other form, the furuncles are quite as numerous, but on the breast, the nates and the extremities. No special area of predilection is determinable however. Oftentimes the entire body is literally covered with boils. The resulting abscesses enlarge with great rapidity and are of a livid color. The sufferers from this dermatosis are, without exception, pale emaciated run-down infants who show very distinctly the clinical evidences of atrophy. The appearance of successive crops of furuncles can hardly be avoided in chil-

dren of this type even though the skin be most carefully protected against infection. Such a furunculosis probably springs from an endo-hematogenous source. This does not imply the existence of a septicemia, since a few pyogenic organisms in the circulating blood can easily be demonstrated in many cachectic infants with poor resistance, who are by no means in a condition, clinically, of sepsis.

The treatment requires great patience. Each furuncle is to be opened with a sharp scalpel and the exuding pus removed immediately with a pledget of cotton, moistened in mercuric chloride solution. Large furuncles should be incised while the patient is in a warm sublimate bath. Following this the skin is freely treated with a bland antiseptic powder. It is best to lay the child on a bed of zinc powder or bran. Moist packs and

Fig. 230.—Multiple skin abscesses in an infant.

ointments are not indicated and are not desirable even when combined with an antiseptic. Recently, cauterization of the centre of the furuncle with a sharp cautery point, has given good results in several cases of the first group. Excellent results are obtained with autogenous vaccines and even stock vaccines may be useful.

PARASITIC SKIN DISEASES

Pediculosis

With inadequate care, the head louse (*pediculus capitus*), a very common inhabitant of the hair of children of the less educated classes, may lead to a dermatosis of an impetiginous character, which, on account of its etiology, is known as pediculosis. The scalp itches intensely, its epidermis desquamates freely, and it is covered, here and there, with large thick and rather firmly adherent crusts, with which the hair is more or less matted. Eczematous papules forming, when scratched, fresh pustules are often found

at or near the hair-line. Especially characteristic is a secondary eczema at the nape of the neck which, incident to scratching, extends down between the scapulæ in a narrow streak. If this sign shows itself we may be very certain that the head louse is or has been present. Close inspection will reveal numerous nits if not the living lice.

The treatment should be directed, first, against the living parasites; second, against the resulting skin conditions; and, lastly, against the nits. The first objective may be reached by washing and then soaking the hair with equal parts of kerosene and olive oil, after which the entire head is firmly covered with a towel left in place overnight. The hair is washed again in the morning.

The second step in the treatment consists in removing the softened crusts and treating their bases with an ointment of ammoniated mercury. The eczema will disappear spontaneously after the parasites have been destroyed and the itching has ceased. Later, if necessary, pastes and powders may be used.

The third requirement, the removal of the nits may be accomplished by the application of vinegar or a 1 per cent. solution of acetic acid to dissolve the chitin, after which the hair is repeatedly combed with a fine-tooth comb.

Shaving or clipping the hair makes the treatment much easier, of course, but can hardly be done with girls, excepting in extreme cases. It may be necessary to remove the hair from the larger pustular areas.

SCABIES

The etiologic factor in scabies is the itch-mite (Sarcoptes hominis or Acarus scabiei), the burrowing of which into the skin causes an extremely annoying affliction commonly known as the itch. Here, again, the most important features of the clinical picture are the secondary manifestations which often appear over the entire skin in consequence of the intense itching and the severe scratching. The itching becomes almost unbearable, especially at night when the body is thoroughly warmed. The numerous bloody crusts and streaks which appear prominently on the skin of the back and chest, are valuable diagnostic signs of the itch. They show plainly how recklessly the patient scratches the skin in order to alleviate the unbearable itching. This practice fully accounts for the manifold secondary skin symptoms which appear. Among these symptoms, we note a reflex eczema (see page 791), urticaria and prurigo, numerous papules and suppurating vesicles, impetigo and ecthyma, all of which, following in the wake of scabies, are essential parts of the clinical picture. The fact that these manifestations are more numerous and more distinct than in the scabies of later years, is due to the tenderness and high fluid content of the child's skin.

In the examination of a case of scabies a definite conclusion depends upon the discovery of the parasite itself; but even without this positive evidence or even without the demonstration of the typical burrows made by the organism we cannot err in making the diagnosis of scabies if the con-

ditions described are found. This is best shown by the favorable results of proper treatment.

The burrows are most frequently formed on the hands and feet and especially upon the inner surface of the wrist, the inner edges of the fingers and on the dorsum of the foot. They may occur, also in other parts of the body and are often seen in the bend of the elbow, the popliteal area and even in the tough epidermis of the palms of the hands and the soles of the feet (see Figures 231-232). The burrows are either straight or slightly curved and are from one-half to one centimeter long and from one-half to one millimeter wide. The termination of a burrow is distinguished by the fact that a small whitish raised point is usually found there. This is the parasite. In order to demonstrate the mite, this spot is opened up with the point of a

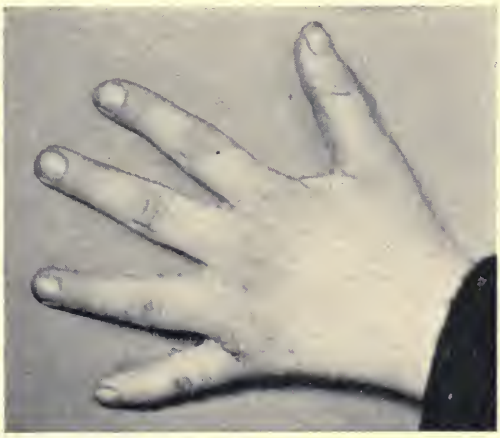


Fig. 231.—Scabies. (Courtesy of Richard L. Sutton.)

sharp knife. It is not necessary to open the entire duct, since this usually causes irritation.

Various antiparasitics have been recommended for the treatment of scabies. A combination of sulphur, tar, and soap has gained great popularity.

We have used, for years, Wilkinson's ointment as modified by Hebra, as follows:

℞	Calcii carbonatis precipitati		10.0	(℥iiss)
	Sulphuris sublimati			
	Oleii cadini	āā	15.0	(℥ss)
	Saponis viridis			
	Adepis tanæ	āā	30.0	(℥i)
M. et fiat in unguentum.				

The method of use is as follows: In the evening, the entire body is to be covered with green soap, thoroughly rubbed in, after which the patient is given a warm bath and rubbed dry. Then the ointment prescribed is applied and rubbed in thoroughly. The hands and feet should receive special attention.

On the following day, the ointment is repeated in the morning. The child is kept in bed and a further inunction is given at night.

On the second day, in the morning, the remains of the ointment are removed with absorbent cotton and oil, after which the entire body is powdered with zinc oxide talcum. In the evening a warm bath is given and a complete change of clothing and bedclothes is provided.

It is really remarkable how well the acute and maltreated eczema of scabies responds to this severe treatment as compared to eczemata of other causation. Irritations are comparatively uncommon. If they do occur the inflamed skin must be furthur treated with zinc ointment and powder.

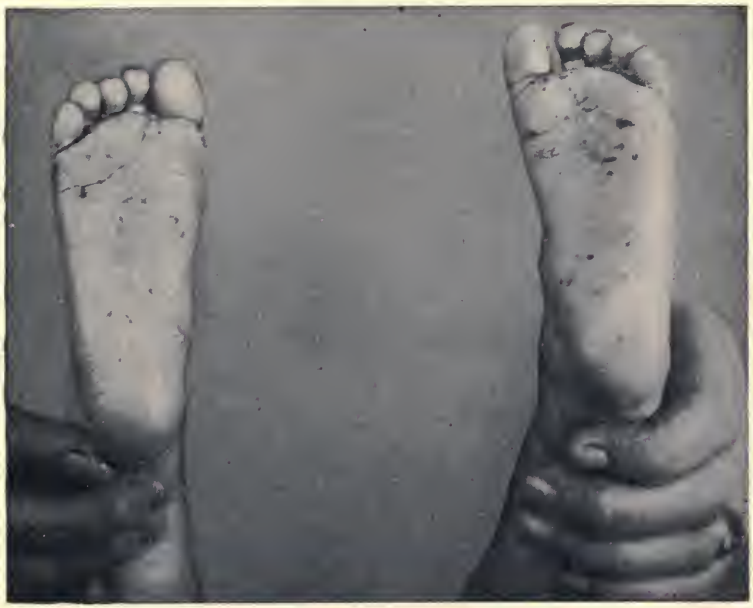


Fig. 232.—Scabies, pustular exanthem.

In infancy, milder ointments, *e. g.*, a mixture of equal parts of balsam peru and olive oil or styrax, are usually employed. The treatment must be continued, however, for several days.

TUBERCULOSIS

The child's skin is probably very rarely the primary seat of tuberculosis. This might be true in lupus more than in any other form of the disease, but even then it cannot be proved that the skin has served as the primary port of entry for the tubercle bacillus. The fact is that at the autopsies of children with lupus, which are quite uncommon, organic tuberculosis is always found although perhaps only in the form of small caseated lymph nodes or as old fibrous foci. Accordingly, the cutaneous disease must be considered a secondary infection, suggesting an acquired hypersensibility, which leads to the usually very characteristic skin changes, the

specific nature of which is now generally recognized. This secondary infection of the skin is either ectogenous or endogenous. An iso ated upus in the vicinity of the nose, probably arises ectogenously, while multiple disseminated cutaneous lesions can be traced only to an endogenous source.

If one who shows tuberculous skin changes be injected with tuberculin, the areas of cutaneous reaction show an acute inflammatory process, a phenomenon which since the time of Koch has been known as a reaction. Skin lesions of a non-tuberculous nature are entirely unaffected by the injection. The specificity of this phenomenon is absolutely determined. It is possible, in doubtful cases, to determine the tuberculous or non-tuberculous character of the skin disease in question in this simple manner.

Another question of secondary importance is whether the skin lesion under consideration is caused by living or dead bacilli, or whether it is caused only by the poisonous products or endotoxins of the bacteria. This question has aroused much interest and has been the subject of much discussion. The bacillary origin of lupus and scrofuloderma, has been established, however for a considerable period of time. As a result of very careful examinations by Leiner, Spieler and Zieler, we know that bacilli or bacillary debris can be demonstrated in numerous other skin conditions of a tuberculous nature. Nevertheless, we should hardly be justified in drawing the conclusion from this fact that all the cutaneous conditions under consideration are always of a purely bacillary nature. For, aside from numerous negative findings, we often see essentially similar changes of the integument, especially in lichen and acne, and when tuberculin has been rubbed into the skin, even though finely filtered tuberculin is used in which there can be no question surely of bacillary debris. For this reason, a differentiated classification of the tuberculous skin disease which shows an attempt to divide them into true or actual tuberculosis and tuberculoid forms is, narrowly speaking, impossible, since one and the same process as, for example, in lichen scrofulosum, may be at one time of a bacillary nature, and at another non-bacillary.

In using the term tuberculide in the ensuing pages, it is not meant to recognize a condition differing in principle from lupus or scrofuloderma. The most important and characteristic point in all these processes is the specific reaction of an oversensitive skin to an invading antigen. The term tuberculide is retained merely for clinical reasons, as are other subdivisions of the several forms of reaction to be regarded as for clinical and diagnostic purposes alone.

LUPUS

The characteristic structural element of lupus is the typical nodules, a small, remarkably soft, brownish-red infiltrate which at first lies embedded in the skin. Its color does not disappear when pressure is put upon it with a microscope slide, although it becomes slightly paler and peculiarly transparent. These nodules are always multiple. If they become confluent in areas where they are crowded together, flat subepithelial infiltrates result, above which the epidermis, deprived of its nutrition, desquamates in large

scales. At the edge of these infiltrated areas new nodules appear, which by their vertical growth gradually rise from their original bed above the level of the normal skin and often completely surround the squamous area of lupus. This is the usual picture of lupus vulgaris, as it is most frequently seen on the face and especially on or about the nose (Figs. 231-4). In cases of favorable termination, this lightest form quickly shows a tendency to involution and goes on to the formation of a slight superficial scar of peculiar silky sheen, in which the original lupus infiltrates are hardly discoverable. More frequently, however, there is a tendency to ulcerative degeneration in the broken down papules and nodules. Ulcers of variable size result which are rapidly encrusted. If these

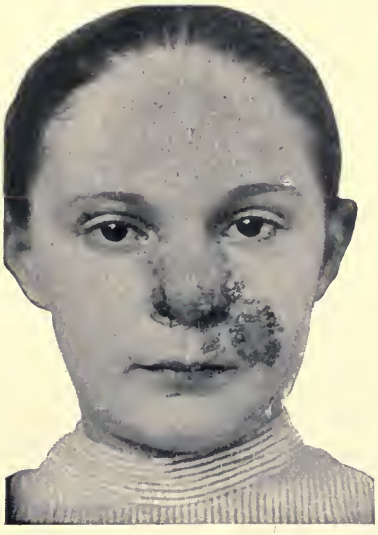


FIG. 233.—Lupus of nose and face. Typical lupus nodules at the edge of the plaques.

crusts be removed, the soft base of the ulcers, often covered with ragged granulations, readily bleeds. If rapidly advancing proliferations of epithelial and connective tissue cells develop at the edges of the ulcer, wart-like new growths appear (*lupus verrucosus*), which on the face often lead to elephantine thickening of the entire affected area. Lupus verrucosus, however, is most frequently seen on the extremities and especially on the extensor surfaces. In this location it may, at times, be of the multiple and disseminated type and with the qualities of an acute exanthem, which is doubtless indicative of a metastatic hematogenous infection.

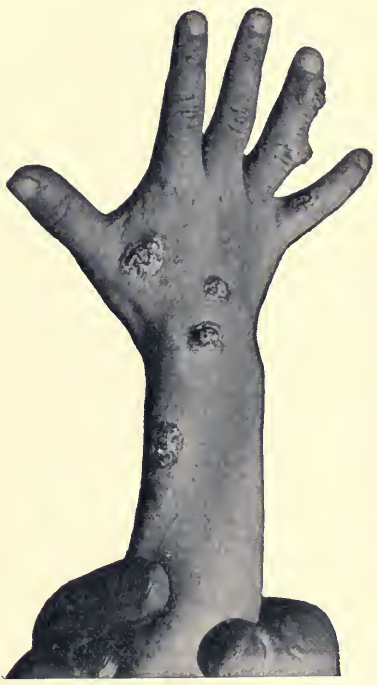


FIG. 234.—Disseminate lupus of skin.

A serious, but quite frequent complication of lupus, is its extension to neighboring mucous membranes where it may be terribly destructive. The distressing facial deformities, the bluish-red shrunken nose, eaten away upon all sides, the nares, enlarged and everted by the destruction of the alæ, suggest the physiognomy of a mummy.

If a differential diagnosis between syphilis and lupus has to be considered, it is to be made by the Wassermann test and the tuberculin reaction.

Treatment.—In the early stages, excision and skin grafting are indicated, chiefly for cosmetic reasons. Lupus, in itself, is not a cause for great anxiety, since there is always an earlier and initial tuberculosis to be considered. Very good results are often obtained by the use of pyrogallol in the form of a ten per cent. ointment. This is to be applied twice a day in a thick layer on sterile lint. If this has the desired effect of producing inflammation and an eschar, the treatment is continued with the use of a solution of aluminum acetate. Usually it will be found necessary to repeat the process several times and, if possible, until all lupus nodules have disappeared. This is followed by inunction with zinc ointment. The Einsen ray gives better results than the Roentgen ray. So far no favorable results have been seen from tuberculin treatment.

FIG. 235.—Lupus vulgaris. (Courtesy of H. H. Hazen.)

SCROFULODERMA

This term is applied to tuberculous skin lesions which begin with nodular infiltration of the derma and which almost invariably go on to purulent softening. Sometimes the nodules disappear spontaneously after persisting for months. Usually they rupture and pus is discharged externally. The nodules frequently arise from a specific infection of a lymph node. In other cases, however, they appear, whether single or multiple, as initial indolent infiltrations of the skin. They vary from one-half to one and a half centimeters in diameter. At first they do not differ in color from the normal skin, but later they become a livid red and of glossy surface. These formations are called scrofulous gummata, but the term is not a very appropriate one since the word gumma should be reserved for syphilitic conditions.

Commonly the scrofuloderma develops in the form of bluish, broken-down, and readily bleeding granulations around the perforate openings of specifically infected lymph nodes, bones or joints. Essentially this represents nothing more nor less than a continuous cutaneous auto-reaction to the influence of the tuberculotoxic pus, voided by the organism, upon its own specifically hypersensitive skin. This reaction is never produced

around fistulous exits by non-specific pus. The fistulous tract, of course, responds similarly and this is why with a persistently increased local hypersensitivity the surrounding tissues show so great a tendency to necrosis and ulceration rather than to permanent healing. It is quite obvious that such areas must leave large and irregular scars which, by means of their blue discoloration, announce their origin even after many years.

In the treatment of scrofuloderma, the wonderful results of direct sunlight have forced the benefits obtained by surgical interference or by

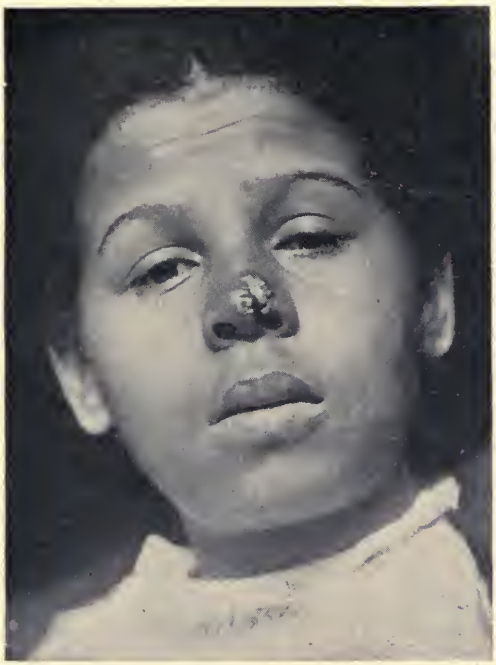


Fig. 236.—Lupus vulgaris. (Courtesy of H. H. Hazen.)

the tuberculin therapy into the background. X-ray therapy has given excellent results.

LICHEN, ACNE, ECZEMA

Since the days of Hebra the classical lichen scrofulosorum has been defined as a superficial exanthem, occurring in a group form, in tuberculous individuals, developing slowly from nodules, varying from a millet-seed to a pinhead in size, of yellowish-brown or livid red color which protrude but slightly above the surface of the skin. These nodules itch very little. They are soon covered by very small scales, and undergo involution after persisting unchanged for months. Such a case is shown in Figure 237, in which, also, the typical localization in the small of the back is to be noted.

Pure cases of lichen scrofulosorum are relatively rare. Usually other

forms of efflorescence are associated in the lichen area, and larger papules, pustules and true eczematous nodules may be scattered through it. In contrast to pure lichen which is always in distinct, sharply circumscribed areas of the size of a hand, these combined exanthemata often cover large surfaces, extending at times over the entire lower portion of the body and especially over the lower extremities.

Therapeutically, the application of cod-liver oil has proven the most satisfactory remedy.

Acne scrofulosum is another exanthem, closely related to lichen, and found even more frequently in scrofulous children. Sometimes it is combined with lichen, but oftener occurs alone and is localized chiefly on the

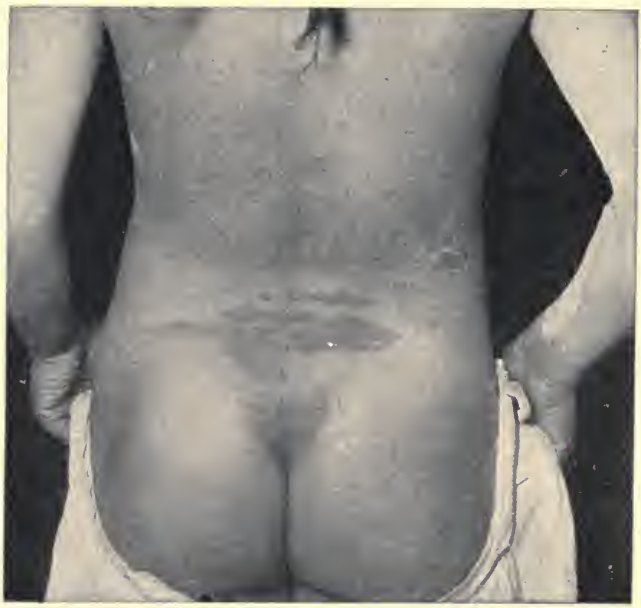


Fig. 237.—Scrofulous lichen. (Children's Hospital, Munich, Prof. von Pfaundler.)

face, the nates and thighs. It occurs in the form of a discrete reddish nodule with a small pustule at the apex surrounded by a red areola. While this is only of the size of a pinhead at first, it grows rapidly to the size of a pea. In the course of its enlargement, the pustule usually dries up and forms a crust. The lesion often leaves, similarly with the papulo-necrotic tuberculide, a flat skin scar which is surrounded by a pale ring of pigment.

As with lichen, so we sometimes see typical acne nodules arising in areas to which tuberculin ointment has been applied. This goes to show that their etiology is not entirely dependent upon bacterial infections.

The same is true of scrofulous eczema Most frequently and very typically it appears in those areas where the skin meets specifically inflamed mucous membranes, as around the eyes, on the eyelids, on the cheeks, about the nostrils, in the concha and the external ear, and particularly in

the fold between the auricle and the scalp. Furthermore, the distinct limitation of these eczemas to their original focus and their slight tendency to superficial spread is characteristic. Their reaction to tuberculin, injected at some other part of the skin, shows their specific character and indicates that, aside from the exudative diathesis, the essential factor to be considered is tuberculosis. Escherich includes all of these skin reactions under the name of scrofulides.

THE SMALL PAPULAR TUBERCULIDE OF INFANCY

This lesion belongs to the group described by Boeck as papulosquamous tuberculides. Hamburger first called attention to the great diagnostic significance of this condition in infancy.

As its characteristic central scale is soon cast off and it usually appears as a small round papule it is permissible to describe the lesion as a small papular tuberculide.

The individual lesion is no larger than a pinhead or at most a small round lentil. It is circular or oval in form, does not itch, and is, at first, either pale or livid red, and later of a light brown color. The absence of any necrosis or ulceration, the existence of a slight depression at the centre of the papule and its glossy appearance when the skin is stretched, are peculiar. The fact that these papules appear in small numbers is especially characteristic. Usually there are not more than three or four papules to be found; at times, indeed, but a single specimen.

The exanthem occurs on the abdomen, the chest or shoulders or on the extremities. Its localization is not typical. This diagnostic sign may put the physician upon the right track at once. Confusion with small hemorrhagic or cachectic strophulus is, of course, possible. For this reason it is always well to apply a cutaneous tuberculin test before making a final diagnosis

THE PAPULO-NECROTIC TUBERCULIDE

This form occurs most frequently on the extensor surfaces of the extremities. Occasionally it is found also on other parts of the body, as on the buttocks or on the auricle.

Its lesions arise from the deeper layers of the skin. On the surface they form large nodules varying from one-fourth to one-half a millimeter in diameter. They soon show a distinct tendency to necrosis and ulceration at their apices. At this stage a dirty brown crust formed is on these summits, the removal of which leaves a crater-like base which bleeds as readily as a lupus. The papules are never very close together; indeed, the crop is scattered but much more numerous than in the small papular form of tuberculide.

Very often the papulo-necrotic tuberculide, accompanied by scrofulous lichen, appears after measles and usually about fourteen days after the exanthem has disappeared. That it persists for a long time is due chiefly to repeated new crops of lesions which keep up the process. The scars left by necrotic tuberculides are very typical. They appear as round white spots, somewhat below the surface of the skin and are surrounded by a pigmented areola.

SUPPLEMENT

EXUDATIVE DERMATOSIS

URTICARIA PIGMENTOSA

(XANTHELASMOIDEA)

Definition.—Urticaria pigmentosa is a rare affection usually beginning in the first year, characterized by the gradual development of wheals,

FIG. 1.—Urticaria pigmentosa. (Courtesy of H. H. Hazen.)

which are followed by pigmented lesions either macules, papules or nodules having a marked tendency to persist.

Symptoms.—While the disease usually begins in the first few months of life, cases are now recorded appearing in young adults. Wheals are the first lesions to appear, rapidly followed by tan or brownish-red pigmented lesions which may be either macules, papules or nodules. All types may appear in the same case. The lesions are most abundant on the trunk, particularly on the back and buttocks, and the neck. The extremities may also be involved. In size the lesions range from a pea to a bean and are usually numerous. Irritation produces wheals on the unaffected skin. Where the pigmented lesions are, itching may be intense or slight. The disease usually runs a persistent course tending to disappear spontaneously at puberty.

Differential Diagnosis.—The itching and wheals suggest urticaria but the pigmented lesions are distinctive. Cases of generalized xanthoma while similar in appearance fail to produce wheals upon irritation. The microscopic picture with mast cell infiltration is also characteristic.

Etiology.—A definite or specific cause is unknown, it is likely that it depends upon a congenital disturbance.

Prognosis.—Life is not affected and the disease is likely to finally disappear.

Treatment.—Treatment should be directed to the amelioration of itching when present, using antipruritics as in urticaria. Every effort should be made to improve the general condition paying particular attention to proper diet. There is no treatment which affects the courses of the disease.

HERPES SIMPLEX

(Herpes Febrilis, Herpes Labialis, Cold Sores, Fever Blisters)

Definition.—Herpes simplex is an acute, grouped eruption of vesicles appearing most frequently at a muco-cutaneous junction.

Symptoms.—The eruption is proceeded by burning and tingling and perhaps some redness and swelling. The vesicles are at first tiny, tense, glistening and from two or three to a dozen in number, filled with clear fluid which may become red from hemorrhage or purulent from infection. After a few days they become flaccid and dry into a crust which shortly falls off leaving a pinkish surface beneath. The eruption may appear around the mouth or nostrils, on the face, eyelids or in the mouth. As a rule the attack lasts only a few days but recurrent varieties occur, sometimes extending over a period of years. The disease is designated according to location as herpes labialis, progenitalis, etc.

Differential Diagnosis.—The diagnosis usually offers little difficulty, although it may be confused with zoster when unilateral and with impetigo, particularly, when infection takes place.

Etiology.—There is no specific cause but the eruption is frequently excited by gastro-intestinal disturbances, colds, acute exanthemata or other febrile affections. In those disposed, even slight local irritation may precipitate an attack.

Treatment.—When seen just previous to the eruption of vesicles the attack may be aborted by painting the area with collodion. Frequent

application of spirits of camphor serve to relieve and possibly shorten the course. When the vesicles have ruptured, antiseptic salves to prevent infection are indicated, 10 per cent. boric acid in lanolin and petrolatum is particularly useful, as it matters not if the child gets it into the mouth or eyes in rubbing. If infection has taken place ammoniated mercury ointment is serviceable. When the lesions occur around the corners of the

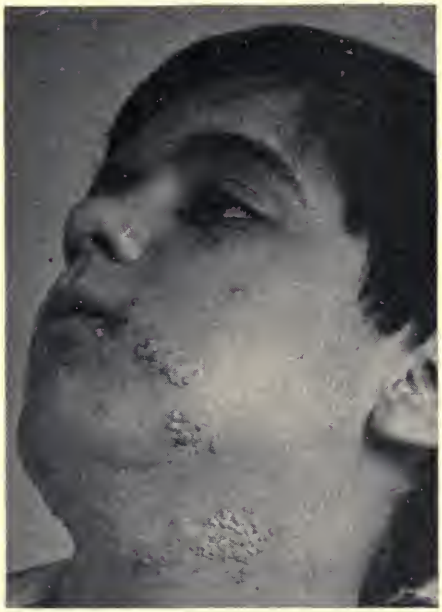


FIG. 2.—Herpes simplex. (Courtesy of Howard Fox.)

mouth and result in fissures, these may be painted with tincture of benzoin or healed by mechanically drawing the parts together and holding with adhesive tape or collodion.

HERPES ZOSTER

(ZONA, SHINGLES)

This disease occurs very frequently in children. It is characterized by a unilateral grouped vesicular eruption on a reddened base, appearing most frequently on the trunk. It is self-limiting in its course and usually measures need be taken only to protect the lesions and prevent infection. A ten per cent. boric acid salve is practical for this purpose.

PEMPHIGUS

The term pemphigus was formerly applied to almost any bullous eruption and until comparatively recent date, there was confusion between pemphigus neonatorum and true pemphigus particularly. The term is now limited to a definite group of diseases characterized by the spontaneous

development of bullæ. Pemphigus occurs in four varieties, pemphigus vulgaris or chronicus, pemphigus acutus, pemphigus foliaceus and pemphigus vegetans.

Pemphigus Acutus

Pemiphigus acutus is applied to a rather rare acute form of the disease usually of a definite toxic nature occurring most frequently in butchers and others particularly exposed to infection and frequently of fatal termination. It is exceedingly rare in childhood.

Pemphigus Foliaceus

Pemphigus foliaceus is a type where exfoliation persistently follows the bullæ until it is general. This is also rare in childhood.

Pemphigus Vegetans

Pemphigus vegetans is exceedingly rare, occurs usually in the middle-aged and is characterized by vegetative growths following the bullæ with their excoriations.

Pemphigus Vulgaris

This is the type, especially, which is understood when the term pemphigus is used It is a disease, usually chronic, characterized by the primary development of crops of bullæ.

Symptoms.—The eruption may or may not be preceded by a slight period of malaises and fever, a few blebs arise which either dry or break and

Fig. 3.—Pemphigus vulgaris.

crust. The lesions are tense or flaccid, and contain serum, pus or blood. The bullæ usually appear in crops, have no inflammatory halo, and are followed by slight pigmentation. Mucous membranes are commonly involved. With the appearance of the eruption there may be a rise of two to four degrees in temperature, considerable prostration, nephritis, enterocolitis and a slight leucocytosis with a relative increase in the neutrophiles.

Course.—The disease may last for months or years or may terminate in one of the other types. There may be little effect upon the general health.

Differential Diagnosis.—Once the disease is established there is little question of the diagnosis because of the non-inflamed base and slow course of the disease.

Etiology.—While micro-organisms have been found there is no proof of their being the causative agent. The cause is actually unknown.

Treatment.—Arsenic pushed to the limit is sometimes of benefit. Everything should be done to better general conditions and focal infection should receive special attention. Locally dusting powder generously applied is the most satisfactory, ointments are ordinarily not well borne. The permanganate bath is of value.

Prognosis.—Patients may survive for years but most of them finally succumb. The benign form which is occasionally seen and which disappears after a few months is a questionable pemphigus, most authorities are agreed that pemphigus is always fatal.

EPIDERMOLYSIS BULLOSA

This is a rare hereditary and familial disease characterized by the formation of bullæ as a result of trauma. The general health is unaffected. It

FIG. 4.—Epidermolysis bullosa. (Courtesy of Richard L. Sutton.)

is not affected by treatment. The first signs appear shortly after birth. It is presumably due to a malformation of the elastic tissue.

HYDROA VACCINIFORME

This disease is also known as herpes æstivalis or recurrent summer eruption. It occurs only in childhood and usually in boys. The eruption

consists of vesicles or small bullæ which occur on the exposed surfaces such as the face, hands and forearms. These break and crust leaving depressed

Fig. 5.—Hydroa vacciniforme. (Courtesy of Richard L. Sutton.)

scars. It is due to some malformation of the skin which makes it susceptible to light. There is no cure for the disease but it spontaneously disappears at adolescence.

INFLAMMATIONS OF THE SKIN

Dermatitis Venenata

Definition.—An acute erythematous or vesicular inflammation caused by an external chemical irritant. Under this title are usually described various types of dermatitis due to chemicals such as mustard, cantharides, croton oil, mercury, iodine, turpentine, etc., and vegetable irritants such as rhus toxicodendron (poison ivy). The latter may be taken as a type inasmuch as it is most common.

Symptoms.—In slight cases only a dry erythema occurs. More frequently it produces an acute vesicular dermatitis and occasionally a violent inflammation with marked redness and swelling accompanied by the formation of large tense bullæ. The exposed parts are naturally most frequently affected, such as the hands, feet and face although other parts may be involved especially around the genitals and anus. Not infrequently with lack of care or wrong treatment a more or less general dermatitis complicates the situation. The eruption usually develops immediately or within a few hours after exposure. There is usually considerable burning and itching and in severe cases constitutional symptoms such as fever of 101° or 102° F.

Diagnosis.—Characteristic eruption on exposed parts with history of exposure.

Treatment.—If the diagnosis is unquestionable, radical treatment may be used, such as washing the affected parts with strong soap and water with a brush or scrubbing with 50 to 90 per cent. alcohol to remove the irritating oil. This treatment is followed by calamine lotion or a bland ointment.

With a dermatitis in which the diagnosis is not so clear, less radical measures are better and there is less danger of increasing the inflammation. Solutions of lead are presumed to precipitate the oil. The following is best:

Plumbi acitatis	5.0
Pulvis alumin	1.0
Aquæ dest	100.0

M. ft.
Sig.—Dilute with 5 to 10 parts of water and apply on gauze.

This application should be used cold on thick compresses of gauze and kept moist. Oil silk may be used over the gauze for twenty-four hours to hasten maceration and rupture of the vesicles and bullæ. When the eruption is dry, treatment may be changed to bland protectives such as zinc paste or the following is exceedingly useful and practical:

Zinci oxididi	
Amyli	
Glycerinæ	
Aquæ	āā 30.0

M. ft.
Sig.—Paint on several times a day.

This application has the advantage of "sticking on" and if dusting powder is applied on top it does not soil the clothing as much as an ointment.

DRY SCALY INFLAMMATORY DERMATOSES

Pityriasis Rosea

(herpes tonsurans maculosus, pityriasis circinata)

Definition.—Pityriasis rosea is a disease of the skin, characterized by the development of symmetrically distributed macules or patches, which are round or circinate in outline, slightly scaly and of a pinkish-red color. It occurs at all ages but only occasionally in childhood.

Symptoms.—There is usually a single lesion that appears first which is known as the "mother spot," this is followed in a few days by other pink-red papules. The lesions tend to enlarge and become patchy and scaly. They are confined largely to the trunk but may appear on the face, shoulders or thighs. Central healing takes place making circinate lesions. The eruption usually disappears in two to four weeks.

Etiology.—Unknown.

Diagnosis.—Distinguished from ringworm by lack of fungus. From psoriasis by its fine bran-like scales, location and lack of infiltration.

Fig. 6.—Pityriasis rosea.

Treatment.—Salves containing salicylic acid and sulphur.

Acidi salicylicii	1.0
Sulphuris lac	3.0
Zinci Oxidi	20.0
Amyli	20.0
Glycerinæ	30.0
Aquæ Dest	30.0

M. ft.

Sig.—Apply several times daily.

PSORIASIS

While psoriasis is one of the most common skin diseases, it is only, occasionally seen in children, although cases even in infancy are reported. The eruption appears in the form of variously sized and shaped macules or

papules, which almost immediately show scaling. The scales are characteristically silvery and rather adherent, particularly the underlying ones. The

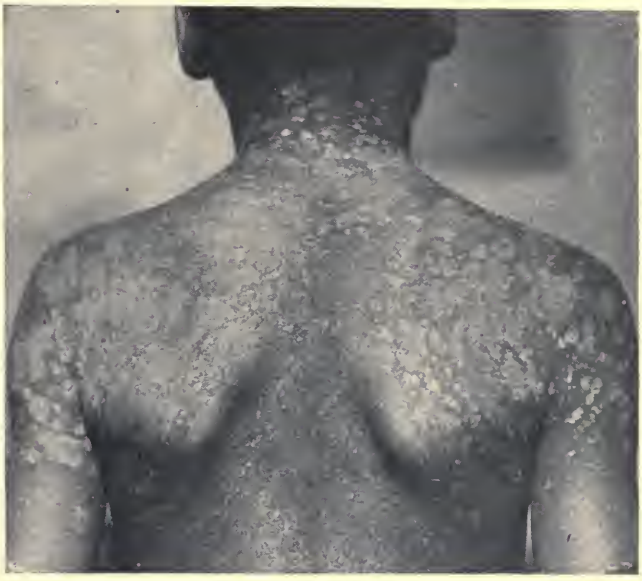


FIG. 7.—Pityriasis rosea. (Courtesy of H. H. Hazen.)

extensor surfaces of the arms and legs, especially the elbows and knees and the scalp are most frequently involved. It is likely to run a chronic course

FIG. 8.—Psoriasis.

with relapses from time to time. While a large amount of investigation has been made, the etiology continues to be unknown. Treatment consists of putting the patient in the best possible condition; focal infection, gastro-in-

testinal disturbances and diet being important factors. Locally, daily baths and ointments are indicated. The list of drugs advised in psoriasis is almost endless. For the more acute cases salicylic acid and sulphur 3 and 5 per cent. or ammoniated mercury 5 to 10 per cent. are valuable. In old chronic cases with a few plaques, pyrogallic acid or chrysarobin 10 to 15 per cent. are good, both produce a dermatitis and neither should be used on the scalp. Ultra-violet light and X-ray properly applied will cause disappearance of the lesions. Recently, raying of the thymus gland has been reported successful by Foerster. Arsenic was formerly used and it is frequently of service, it is questionable, however, with the frequent recurrences, whether it is advisable as a routine, on account of the danger of arsenical keratoses following its unrestricted use.

INFECTIOUS DISEASES OF THE SKIN

Granuloma Pyogenicum

Granuloma pyogenicum is a term applied to a slow developing, usually pedunculated tumor one-fourth to one-half inch in diameter, arising upon the site of an injury. The lesions are usually single, the size of a pea or bean and bright red in color. They occur most frequently upon exposed surfaces.

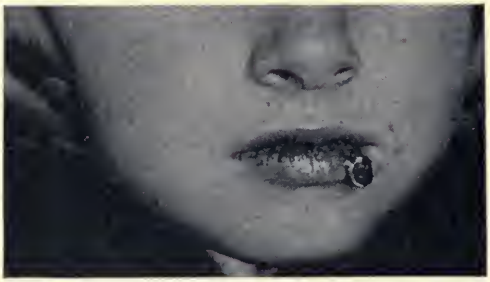


Fig. 9.—Granuloma pyogenicum. (Courtesy of Richard L. Sutton.)

The surface may be smooth or cauliflower-like with purulent secretion in the crypts. It is presumably due to infection with staphylococcus. The lesion persists indefinitely and unless properly removed, recurs. Removal with electric cautery is the best. The tumor may be excised and the base thoroughly cauterized with zinc chloride.

FAVUS

(Tinea Favosa)

Favus is a fungus disease, usually limited to the scalp, occurring primarily in childhood but not always disappearing with adolescence, as other forms of tinea. It is seen infrequently in this country and usually in immigrants. It may appear on the body or nails. The characteristic lesion starts as a papule around the hair follicle, followed by a dirty yellowish crust

with concave surface. There is loss of hair. Diagnosis is made by characteristic lesion and microscopic demonstration of the fungus. Treatment

Fig. 10.—Favus.

consists of epilation by X-ray followed by antiseptic applications to bald scalp. Precautions should be observed to prevent the spread. Baldness produced by the disease is permanent.

TINEA TRICHOPHYTINA

(Ringworm, Trichophytosis)

Tinea trichophytina or ringworm is a local infectious disease of the skin due to the trichophyton fungi. The various conditions produced by these fungi may be classified according to the nature of the fungus or according to the part involved, the latter is by far the most practical, particularly in this work where only the ones of importance to the pediatrist are to be described.

TINEA TRICHOPHYTINA CORPORIS

(Tinea Circinata; Ringworm of the Body; Trichophytosis Corporis)

Symptoms.—Ringworm of the non-hairy skin begins as a flattened pinkish papule with a tendency to spread peripherally and clear up in the centre. As the lesions enlarge they become ring-shaped, usually with a sharply marked border and pinkish or brownish furfuraceous centre. Combined lesions form gyrate figures. Spontaneous healing may take place. Subjective symptoms are not often present. The uncovered portions of the

body are most frequently involved. The deeper parts of the skin may be-

FIG. 11.—Tinea circinata. (Courtesy of Howard Fox.)

come involved, giving a papulo-pustular lesion, carbunculoid in appearance.

TINEA TRICHOPHYTINA CRURIS

(TINEA CRURIS: ECZEMA MARGINATUM: DHOBIE ITCH)

This variety, together with that occurring occasionally in the axillæ and on the hands and feet is usually due to the epidermophyton inguinal. The cruris type is rarely seen in children, it does not differ from the others except in location and the tendency to vesicle or pustule formation along the border. The condition caused by infection with this fungus on the hands and feet is seen in children as an eczematoid dermatitis. It occurs as a vesicular eruption, frequently grouped and sharply marginated on the backs of the hands, between the fingers and similarly on the feet. Occasionally the palms and soles are also involved. Between the fingers and toes the skin becomes boggy from perspiration and peels with fissure formation.

Diagnosis.—Ringworm of the body is to be differentiated from pityriasis rosea; seborrhœic dermatitis and psoriasis. Ringworm occurs more frequently on the uncovered parts, pityriasis rosea is usually limited to the trunk, ringworm is more frequently limited to a few lesions, pityriasis rosea begins with one lesion, the "mother spot" and later shows quite a general eruption pretty well covering the trunk, individual lesions are almost identical, only microscopic finding would definitely differentiate them. In sebor-

rhœic dermatitis, usually the sternal and interscapular areas are the principal ones involved, scales are greasy and there is no tendency to vesicle or pus formation. Psoriatic lesions are always dry, may be ring-shaped but the scales are characteristically silvery, bleeding points occur upon scraping scales away. There is nearly always involvement of elbows, knees, or scalp. The eczematoid ringworm of hands and feet is frequently impossible to differentiate from eczema. When both hands and feet are involved it is more likely ringworm, theoretically one might depend upon the microscope but practically the fungus is frequently so hard to find that this is not safe. Considerable work is being done on this condition but it still remains in considerable confusion. Specimens for the microscope are made by taking scrapings of the scales or the tops of vesicles, applying 30 per cent. sodium hydroxide to dissolve the tissue and in one-half hour and two hours the spores and fungi may be seen.

Etiology.—Infection may occur by direct contact or through the use of contaminated articles, hats, underwear, combs, brushes, etc. The infection exists in the domestic animals and is frequently contracted from them.

Prognosis.—The prognosis of ringworm of the body is always good.

Treatment.—On the body, the condition is usually soon cleared up, with ointments of ammoniated mercury 5 to 10 per cent., sulphur 5 to 10 per cent. or Whitfield ointment, salicylic acid 3 per cent. and benzoic acid 5 per cent. Aqueous solutions of sodium hyposulphite 10 per cent. alone or followed by a solution of tartaric acid 3 per cent. On the hands and feet the lesions are frequently more rebellious, the above mentioned Whitfield ointment may be used until peeling takes place and then followed by zinc paste or crude tar ointment may be used:

Crude tar..... 4.0
Zinc oxidi..... 4.0
Amyli.....30.0
Petrolatum Flav...30.0
M. ft. Ung.
Sig.—Apply 2 or 3 times daily, cleaning off with sweet oil.

ONYCHOMYCOSIS

(Ringworm of the Nails)

Ringworm of the nails may occur primarily or secondarily. The nails are a dirty grayish or yellowish color, appear worm eaten and thickened. Fungus is found with difficulty in scrapings. The nails should be scraped frequently and Whitfield ointment or pure crude coal tar applied.

TINEA TRICHOPHYTON CAPITIS

(Tinea Capitis: Tinea Tonsurans: Ringworm of the Scalp)

Symptoms.—Ringworm of the scalp is definitely a disease of childhood, spontaneous involution taking place at puberty. It starts as a small scaly patch or reddened hair-perforated papule. The base is reddened but the scales are whitish or grayish in color. There is no tendency to central

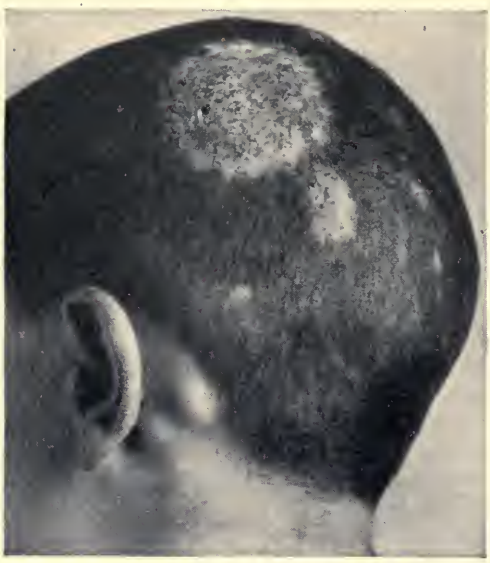


FIG. 12.—Tinea capitis. (Courtesy of Howard Fox.)

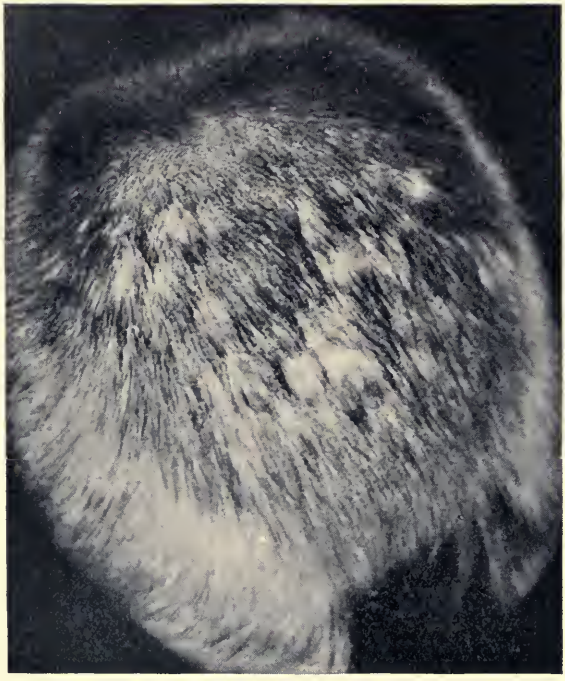


FIG. 13.—Tinea capitis.

involution. The hair becomes lustreless and dry and many broken hairs are seen in the partly bald patch. Occasionally there is a pustular folliculitis.

Diagnosis.—The condition as described above is characteristic and the diagnosis is easily confirmed by examining hairs treated with sodium hydroxide solution.

Prognosis.—The prognosis depends upon the treatment, with older methods of epilation and treatment, usually recovery was a matter of several years. Epilation with X-ray followed by applications of iodine is the modern accepted treatment and the time is reduced to a few months.

Treatment.—The older methods consisted of forcep spilation and the application of various antiseptic salves. Nowadays the entire scalp is epilated with X-ray, the hair falling out from one to three weeks after exposure, depending upon the method used. When the hair is out, the scalp is painted several times with tincture of iodine. The hair grows in again in from two to three months. If necessary epilation can be repeated. Obviously this treatment should only be undertaken by an expert. Precautions should be taken to prevent spread of the infection to others, the child wearing a skull cap.

DISEASES OF THE APPENDAGES OF THE SKIN

(Miliaria)

Miliaria is a sweat eruption produced by the retention of sweat in the sweat follicles. It occurs as miliaria crystallina and miliaria rubra. The lesions of the first are non-inflammatory and the second inflammatory.

Miliaria crystallina or sudamina is characterized by an eruption of numerous tiny discrete acuminate vesicles. The vesicle walls are very thin and rupture easily. It is produced by excessive sweating and is without subjective symptoms. It is usually confined to the trunk.

Miliaria rubra (prickly heat, heat rash) is an eruption of miliary vesicles and papules at the mouths of the sweat follicles. The lesions are minute, superficial, thin-walled, vesicles, upon an inflamed base. The contents are at first clear and later opalescent. There may also be numerous very fine papular or papulo-vesicular lesions. The lesions are usually very numerous giving to the whole area where they occur a pinkish appearance. The sides of the neck, chest and back are the usual sites but other parts may be affected. There is always burning, pricking, itching and marked discomfort. There is frequently an accompanying dermatitis and when this occurs in the folds in adipose individuals it may lead to an intertrigo. The diagnosis is not difficult when the associated sweating or overheating together with the characteristic lesions are taken into consideration. Miliaria crystallina requires no treatment other than bathing and powdering. Miliaria rubra is frequently very disturbing and requires treatment to relieve the itching, otherwise scratching and rubbing are likely to produce further trouble. Sponging with dilute alcohol (60 per cent.) and pow-

dering may be sufficient. Calamine lotion applied frequently is satisfactory. A practical and satisfactory application is:

Zinci oxide		Glycerinæ	
Amyli	āā 30.0	Aquæ dest.	āā 60.0

M. ft. in unguent.
Sig.—Apply several times a day.

This application may be smeared on several times a day and powder dusted over it. Salves should not be used. The eruption rarely lasts more than a few days unless the causes are permitted to continue. Every effort should therefore be made to keep the child cool by means of cool baths and light clothing.

MILIUM

(Strophulus Albidus, Acne Albida)

Milia are small whitish tumors of the skin formed by hardened sebum beneath the epidermis. These pinhead to small pea-sized papules usually occur beneath the eyes. They may also be seen on the scrotum. There are usually only a few and they remain discrete. Milia are common in young adults and not infrequently seen in infants. In the latter vigorous washing with soap and water will dispose of them. Later in life it is necessary to puncture the lesion and express the contents.

COMEDO

A comedo (blackhead) is a plug of dried sebaceous material which fills the opening of a sebaceous follicle. They are more frequently seen where the sebaceous glands are most numerous as on the forehead, nose, nasolabial furrows, chin, chest, and back. They are almost constantly associated with acne vulgaris and constitute a definite part of that disease.

GROUPED COMEDONES IN CHILDREN

Groups of comedones occurring in children upon parts subjected to pressure and warmth have been described by Corcker. They are seen on the cheeks in nursing infants and on the forehead, temples and occiput of older children.

ACNE NEONATORUM

An acne occurring on the forehead and nasal folds of infants has been described by Kraus. Both comedones and pustules are present and they appear to have the characteristics of a true acne due to a disturbance of sebaceous secretion.

ACNE VULGARIS

Acne vulgaris is characterized by an eruption of inflammatory lesions, situated in the sebaceous follicles consisting of comedones, papules and pustules. The lesions are found where the sebaceous glands are numerous, face including forehead, nose, cheeks and chin, the chest and back. There is usually little difficulty in the diagnosis.

Several types are described depending upon the predominant lesion such as acne simplex, acne indurata, acne papulosa and acne pustulosa. Usually

there is a mixture of all lesions. Superficial lesions produce no scarring but the deep-seated lesions of acne indurata do produce scars and this is one important reason for treatment. The disease is most pronounced at adolescence but may occur in younger children. In the superficial types, a salve of salicylic acid and sulphur with plenty of soap and water suffices. The salicylic acid is used 1 to 3 per cent. with precipitated sulphur 3 to 5 per cent. If many comedones are present they should be mechanically removed. In the pustular and indurated types the lesions need to be surgically opened in addition. Mixed staphylococcus vaccines, 150 million to 400 million, four days to a week apart, help. Ultra-violet ray given to the point of producing a dermatitis is of decided assistance. In severe cases the X-ray is the best agent of all but must be used expertly and care exercised not to overdose on account of later developing telangiectases. All local treatment should be accompanied by regulation of diet, care of constipation, removal of focal infection and adherence to all measures productive of good hygiene. All cases can be cleared up, but the natural tendency is to relapse and there is usually a need for considerable care for several years.

ALOPECIA AREATA

Alopecia areata is characterized by the sudden loss of hair in patches

FIG. 14.—Alopecia areata totalis. (Courtesy of Howard Fox.)

of various sizes and shapes. There may be one or many areas. Recurrence

is frequent. To the pediatrist the usual alopecia areata is of little moment, since in the young the hair usually grows in of its own accord. Particular attention, however, is called to that type which tends to become universal with the loss of eyebrows, eyelashes, axillary and pubic hair, and even lanugo hair. When the onset of such a condition is sudden, it is more than likely to be permanent. I have seen a number of such cases, have attempted various kinds of therapy and always with no result. In no case in my experience has a general examination disclosed anything wrong. In the ordinary case, stimulation with alcoholic lotions or application of ultra-violet rays, hastens the growth and tends to insure a growth of the normal colored hair instead of white or gray.

BENIGN EPITHELIAL GROWTHS

(Adenoma Sebaceum)

Adenoma sebaceum is a disease characterized by the development on the face of tumors of the sebaceous glands. It begins in early childhood as

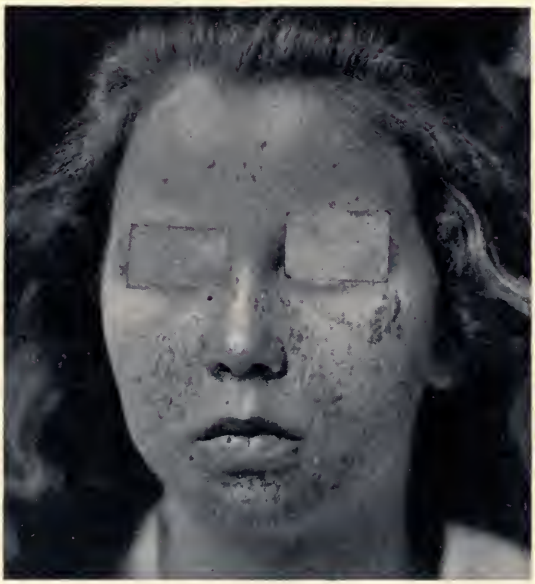


Fig. 302.—Adenoma sebaceum. (Courtesy of Richard L. Sutton.)

bright red, yellowish or brownish pinhead to pea-sized papules on the cheeks, forehead, nose and chin. The color is due to minute telangiectases. There are frequently seen in conjunction, moles, nævi and other developmental defects. Patients are frequently of low mentality. The lesions persist, being most abundant at puberty. Treatment is by destruction with elec-

tric needle or cautery. Where lesions are abundant, I have had good results from freezing thirty to forty seconds with carbon dioxide snow.

Fig. 16.—Adenoma sebaceum. (Courtesy of H. H. Hazen.)

HYPERTROPHIES

(Verruca Vulgaris)

Verruca vulgaris or common wart is seen almost entirely in childhood. It is now accepted that they are mildly infectious and contagious, although nothing is known as to the infecting organism. The lesions occur chiefly on the hands and are hypertrophic epithelial growths. Of the many caustics so frequently recommended and tried, almost all are to be condemned as they frequently lead to infection and bad scarring. Magnesium sulphate and mercury internally have been reported as successful. The former in sufficient dosage to produce two or three movements of bowels daily and the latter as protiodid in proper dosage according to the age. I prefer local treatment. Roentgen ray and radium applied just short of producing erythema are painless, almost invariably successful and leave no scar. The high frequency spark is successful but painful and children will scarcely stand it. CO_2 snow can be used successfully and is not exceedingly painful, the lesion is pared down as much as possible and an area larger than the wart frozen about one minute with good pressure, a blister results with the wart on top. A dressing should be applied to prevent infection while healing. Raying is particularly to be advised when the warts occur around the nails.

VERRUCA PLANÆ JUVENILIS

This type of wart is seen usually in infancy and childhood. The lesions are flat, numerous, yellow and glistening and appear chiefly upon the face, neck and backs of the hands. They are very small, sometimes barely visible.

This is the type in which the internal treatment is more often successful. Light cauterizing with a saturated solution of trichloracetic acid or the application of 10 to 15 per cent. salicylic acid in collodion is usually successful.

KERATODERMIA PALMARIS ET PLANTARIS

(Symmetrical Keratodermia of the Extremities, Congenital Keratoma of the Palms and Soles, Ichthyosis Palmaris et Plantaris)

Definition.—This condition is a familial, often hereditary, symmetrical hyperkeratosis of the palms and soles.

Symptoms.—The palms and soles show patches of thickened epidermis up to an eighth or quarter of an inch, which are smooth, yellow and sharply marginated. While the condition is often hereditary, it may not appear for a number of years. There are usually several cases in the same family.

Course.—The disease is permanent.

Differential Diagnosis.—Appearance and history are usually sufficient to assure the diagnosis. A similar condition may appear in psoriasis and arsenical keratosis must be considered.

Treatment.—In the true cases treatment is palliative only and consists of the application of oils or oily salves. Diachylon ointment applied thick on lint serves well. X-rays may be tried.

ICHTHYOSIS

(Xeroderma, Fishskin Disease)

Fig. 17.—Ichthyosis. (Harlequin Fetus.) (Courtesy of Richard L. Sutton.)

Ichthyosis is a congenital defect of the skin characterized by harshness, dryness and scaling, due to an increase in the horny layer and a deficiency of the secretions. While ichthyosis is a congenital defect it does not ordinarily become apparent until the second or third year, thereafter remaining unchanged until puberty when improvement may take place. There is a true congenital type which occurs very rarely. It is manifested at birth, the surface being covered by thick, scale-like plates marked in all directions by fissures in the horny epidermis. The skin is immobile. Such cases are seen usually in children prematurely born and they usually die within a few days from inanition or loss of heat. It is usually impossible for them to nurse on account of im-

mobility of the lips. A condition which simulates true ichthyosis is due to an accumulation of vernix caseosa, which continues to be produced for a few days after birth. The skin is encrusted, tense, fissured and of a brownish-red color. These cases may die of inanition unless the crustations are softened and removed with oily applications and the body temperature

FIG. 18.—Ichthyosis. (Courtesy of Richard L. Sutton.)

artificially maintained. There is also another rare condition in which the child is born with a membrane covering the skin which resembles collodion. This peels off in sheets followed by further desquamation and normal skin. It is suggested that this is an example of the persistence of the epitrichial layer usually cast off at the seventh fetal month.

Ichthyosis or ichthyosis simplex is a general condition involving the entire skin to a greater or less degree, while ichthyosis hystrix is limited to irregular circumscribed areas. The condition varies in intensity, from a

slight roughening of the skin to cases in which the horny epidermis consists of thick plates, resembling fish scales. It is usually worse on the extensor surfaces of the arms and legs. The hair and nails are usually dry, the hair lustreless and the nails brittle. There are no subjective symptoms but the condition of the skin makes it susceptible to certain types of dermatitis, due to the lack of secretions, these inflammations are more likely to arise in

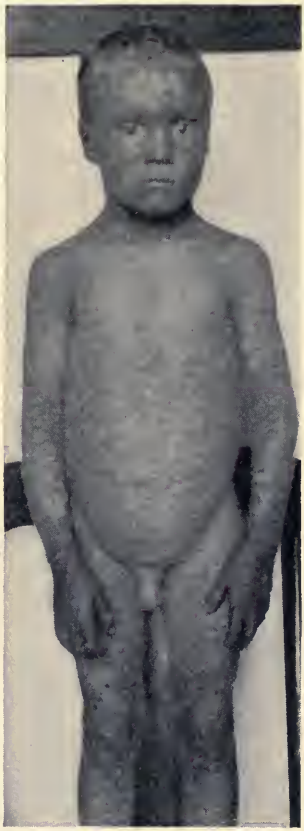


FIG. 19.—Ichthyosis.

the cold weather. The affection is hereditary and frequently more than one case is seen in a family. While ichthyosis cannot be cured, the patient can be greatly benefited with proper care. Some patients show thyroid disturbance and are greatly improved by thyroid extract. Not more than a grain a day should be given to start with, the dose to be increased according to tolerance.

Proper care consists of a daily bath and inunction with a bland fat. In severe cases tincture of green soap is necessary as well as keratolytic salves such as one containing salicylic acid 5 to 10 per cent.

ATROPHIES

(Xeroderma Pigmentosum)

Xeroderma pigmentosum is a rare disease of the skin beginning in early childhood, occurring in families and characterized by the appearance of

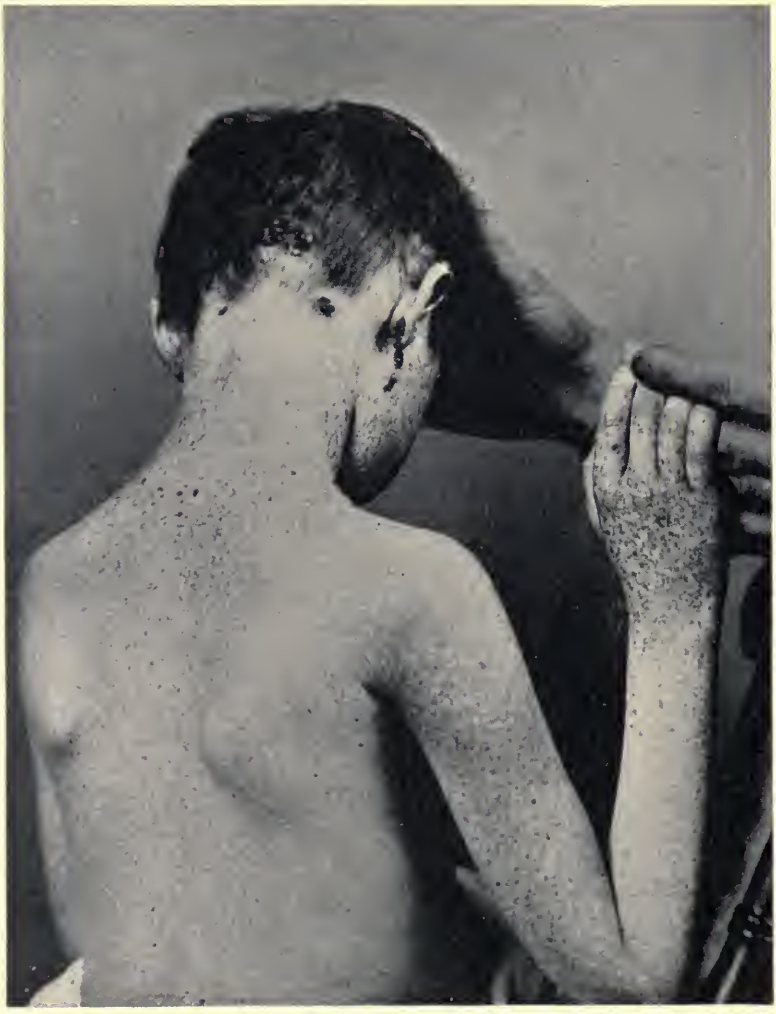


Fig. 20.—Xeroderma pigmentosa. (Courtesy of H. H. Hazen.)

freckles following which there are atrophic changes and later malignant growths. The disease may show its first manifestation by erythematous spots in infancy but later freckles appear becoming more numerous and more pronounced. The skin in a few years takes on the appearance of extreme age with atrophy, a waxy drawn appearance, telangiectases and

keratoses appear which rapidly become malignant. The lesions are limited to the exposed surfaces and aggravated by light. It is undoubtedly due to

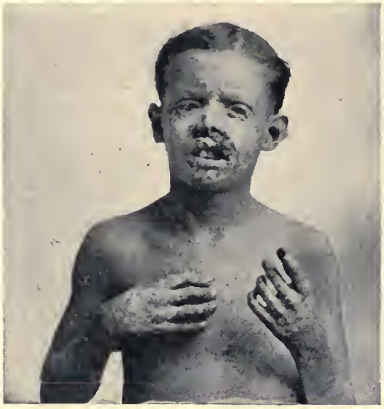


FIG. .21—Xeroderma pigmentosa. (Courtesy of Richard L. Sutton.)

a congenital defect. The disease usually starts in the first year or two and reaches its height at six to eight, while death occurs within two or three years with frightful distortion and disfigurement. Occasionally malignancy does not develop.

FIG. 22.—Molluscum contagiosum. (Courtesy of Howard Fox.)

DEGENERATIVE NEOPLASMATA

MOLLUSCUM CONTAGIOSUM

(MOLLUSCUM SEBACEUM, EPITHELIOMA CONTAGIOSUM)

Molluscum contagiosum is a disease of the skin, with small epithelial waxy rounded tumors, usually umbilicated and multiple in number. While the disease is rather uncommon it occurs more frequently in children than in adults and particularly in those of the poorer class, or in institutions. There are usually from two or three to a dozen lesions and these are seen most frequently on the face, eyelids, nose, scrotum or backs of the

hands. The lesions usually remain separate, but may group, each remaining distinct. They are usually the size of a pinhead or a small pea but on rare occasions, lesions an inch or two in diameter are seen. Individually the tumor is distinctly raised, constricted at the base, almost pedunculated, waxy in appearance and umbilicated with an opening in the umbilication from which cheesy material may be expressed. They are mildly infectious but the organism is not known. The characteristic lesions offer little difficulty in diagnosis. Untreated they tend to persist months or even years. Treatment consists of puncture and expression of the contents. Usually a puncture which draws blood is sufficient without expression.

XANTHOMA

(Fibroma Lipomatodes, Xanthelasma)

Xanthoma multiplex (xanthoma tuberosum) is a rare disease but may occur in children. The lesions are rather rounded, pea to bean-sized, from a yellowish to a brownish-red in color and when seen in children are usually multiple and general in distribution. They are usually rather hard and once formed persist with practically no subjective symptoms. Only radical treatment is successful, the cautery or strong (25 per cent. to 4 per cent.) salicylic acid either in plaster or collodion.

NEVUS

(Birth-mark)

There are two kinds of nevi, pigmented and vascular. The former are frequently classified among skin hypertrophies and the latter among tumors of blood-vessels. Since the pediatrist is concerned only with types and little with the diseases sometimes classified with blood-vessel disturbances, it seems more practical to consider all nevi under one heading.

NEVUS PIGMENTOSUS

(Pigmented Mole)

Pigmented nevi are circumscribed lesions of the skin in which there is increased pigmentation. They may or may not be covered with downey or coarse hair. The lesions more often appear on the face, neck, trunk, thighs, buttocks and genitalia, but may appear anywhere. They vary in size and shape and may be smooth and flat or raised and warty. Their occurrence may be single or multiple and disseminated or grouped. They are rarely found at birth but make their appearance during infancy. No subjective symptoms are present. There is no tendency to spontaneous disappearance. Pigmented nevi or moles are important for two reasons, first cosmetic and

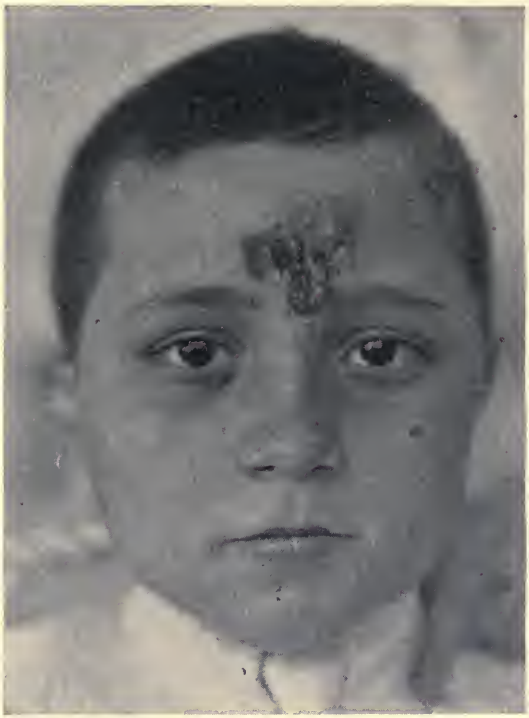


FIG. 23.—Nevus pigmentary. (Courtesy Richard L. Sutton.)

FIG. 24.—Nevus pigmentary. (Courtesy of Howard Fox.)

second on account of their well-known tendency to malignancy, the pediatrist is not concerned with the latter as malignant changes rarely occur before forty.

Treatment.—Small brown moles may be removed by fulguration or electric needle. With the exception of trichloracetic acid, no acids or other caustics should be used on account of the danger of unnecessary scarring. The small lesions may easily be removed by painting lightly with a solution of trichloracetic acid, almost no scarring results and I have never seen any hypertrophic scars follow its use, such, as are frequently seen after nitric acid. The solution should be made by adding just enough water to the crystals to dissolve them. The dark blue or black moles should not be interfered with except in the most radical manner since danger of malignancy is great and when it occurs it is usually widely disseminated and incurable. The larger lesions may be treated with electric needle and hairs removed by the same agent. Freezing from twenty to sixty seconds with carbon dioxide snow is successful. The X-ray skilfully used will give a good result, trial by any one but an experienced therapeutist is, however, not to be recommended. Radium is less dangerous. Excision does not give as satisfactory results as the other methods mentioned.

NEVUS VASCULOSUS

(Nevus Sanguineus, Nevus Flammeus, Moths Mark, Birth-mark, Port-wine-mark)

A vascular nevus is a congenital hyperplasia of the cutaneous vascular system. These nevi may be flat or raised, single or multiple, large or small with the color depending upon their structure. There are three types of vascular nevi.

First, flat nevi consisting merely of dilated capillaries (the port-wine-mark).

Second, hypertrophic nevi, angiomata made up of a net-work of large dilated vessels.

Third, angioma cavernosum, a cavernos nevus which usually enlarges.

The color in all types depends upon whether the blood is arterial or venous. They are usually sharply circumscribed and compressible. More frequently these nevi appear on the face, head or neck, but no part of the body is exempt. Flat or hypertrophic nevi may be present at birth or make their appearance shortly after, they usually increase in size for a time and then remain fixed. The large angiomata sometimes ulcerate following trauma and a cure results. There is of course in such a case definite danger of alarming hemorrhage. Other than this there is no tendency to spontaneous disappearance. Most nevi are amenable to treatment. The raised ones may be frozen with CO_2 snow ten to thirty seconds, flat ones do not respond to this. The flat type may be treated with the Kromayer quartz lamp (ultra-violet light). This and other types may be treated with

FIG. 25.—Angioma. (Courtesy of Howard Fox.)

radium or X-ray; in either case inasmuch as the cosmetic result is usually of great importance, the work should be attempted only by one who is experienced.

TELANGIECTASIS

Telangiectases are acquired dilated capillaries. There may be many or few and they are usually seen on the face. They frequently occur in the form of a central red dot with radicating dilated capillaries constituting the so-called spider nevus or nevus araneus. These may be treated by using the electric needle, with one and one-half milliamperes of current, the negative pole, in the central dot. On account of the pain with small children it is easier to use a needle cautery and just lightly puncture at the point.

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