Atlas and epitome of diseases of children / by R. Hecker and J. Trumpp ; edited by Isaac A. Abt.

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Hecker, R. 1868-1963.
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Publication/Creation

Philadephia ; London : Saunders, 1907.

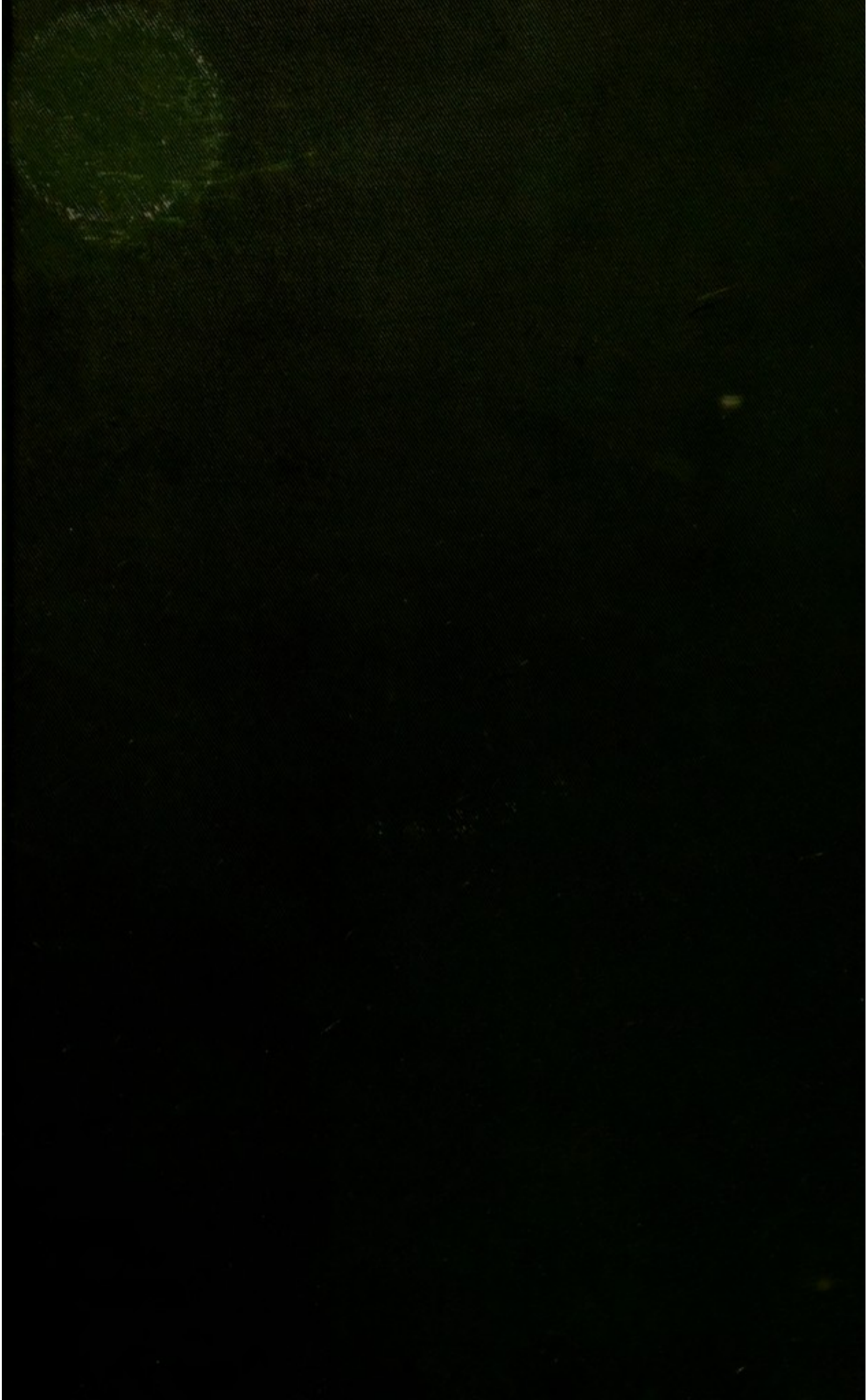
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ATLAS AND EPITOME

OF

DISEASES OF CHILDREN

BY

DR. R. HECKER and DR. J. TRUMPP

of the University of Munich

ROTHERSILLIAN
11 CHANDOS ST. CAVENDISH SQ.
FUND

Authorized Translation from the German

EDITED BY

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PHILADELPHIA AND LONDON

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EDITOR'S PREFACE

THE text of the present volume forms a manual of Pediatrics in small compass. The plates, as will be seen on examination, are very life-like and, as in the other volumes of "Saunders' Hand-Atlases," accurately portray the conditions they are intended to represent.

The additions by the Editor will be found enclosed in brackets. Considerable changes have been made in the sections on Therapeutics, and these have been revised in accordance with the practise in this country. This was called for, especially since many of the climatic measures, health foods, and apparatus are not to be obtained in the United States.

PREFACE

PEDIATRICS is, unfortunately, still the stepchild of our universities. It is true that, owing to its eminently practical importance, it is included as a secondary subject in the general examination for the degree in medicine, but many universities, nevertheless, are still without pediatric clinics and separate chairs for diseases of children. Under these circumstances it is no wonder that the pediatric education of many students and young physicians, who have been obliged to get their knowledge of the subject from text-books, leaves much to be desired. The best text-book cannot take the place of clinical observation, but this deficiency can, to a great extent at least, be supplied by pictorial illustrations, provided they are given in sufficient numbers and arranged in series in a manner adapted to the use of students.

The current text-books on pediatrics do not meet this requirement, and the authors have, therefore, decided to bring out this "Atlas of Diseases of Children" in the hope that it will enable students and young practising physicians who have not had the advantage of clinical instruction to gain a better understanding of systematic text-books, and that the epitome which the Atlas contains may serve as a convenient summary of the subject. We also hope that teachers may occasionally find the book useful in their class work.

In the preparation of the illustrations we have had the benefit of the kindest co-operation from various quarters. Professors Escherich and Pfaundler have kindly allowed us to reproduce a number of splendid illustrations from the collection of the Pediatric Clinic in Vienna and Graz. Professors Heubner and Stoltzner furnished us some valuable microscopic preparations; and, last but not least, the directors of the Münchener Institut, Geheimrat von Ranke (Kinderklinic), Obermedizininalrat von Bollinger (Pathologisches Institut), and Professor Ruckert (Anatomy), very kindly allowed us free access to their Institutions. To all these gentlemen, as well as to Prosector Dr. Hahn, Professor Dürck, Dr. Eggel, and Dr. O. Seitz, who were kind enough to assist us in the preparation of specimens, we take this opportunity of expressing our warmest thanks.

The colored plates were furnished by the artists Messrs. Tersch, Liner, and Dirr, while the task of preparing the engravings was undertaken by Messrs. Tersch and Biehl (Atelier Elisabeth).

Mr. Lehmann, the publisher, has spared neither pains nor expense in perfecting the external garb of this work, and we must not neglect to express our grateful acknowledgment of his invariable willingness to carry out our wishes.

THE AUTHORS.

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DISEASES OF CHILDREN

GENERAL PART

THE fact that certain diseases are peculiar to infants and that many of the diseases of adult life assume an altogether different character in children, depends less upon differences in causation than upon the difference in the region attacked by the disease process. A thorough knowledge of the construction and function of the infantile body is absolutely necessary in order to understand the diseases of children. In general, the body of a child is characterized by its diminutive size, by its diminished resistance, and the consequent hypersensitiveness of the organs, by increase in growth and alterations in form, together with the associated variations of the physiologic functions and reactions of the organs.

ANATOMIC PECULIARITIES

THE FETAL CIRCULATION

The blood circulates through the fetus as follows: From the placenta through the umbilical vein; the latter divides into two branches at the liver, one empties into the portal vein and the other, as the ductus venosus Arantii, into the inferior vena cava. This vessel in turn empties into the right auricle, whence the blood is guided by means of the Eustachian valve through the foramen ovale directly into the left auricle. The blood then flows into the left ventricle, the aorta, the major circulation, and in part through the hypogastric and umbilical arteries, to be aërated in the placenta. The blood from the superior

FIGURE 1

Circulation in the Fetus.—1. Umbilical vein. 2. Branches of the portal vein. 3. Ductus venosus Arantii. 4. Inferior vena cava. 5. Aorta. 6. Hypogastric arteries. 7. Umbilical arteries. 8. Superior vena cava. 9. Pulmonary artery. 10. Ductus arteriosus Botalli.

---

vena cava flows into the right auricle, and passing the blood current from the inferior vena cava, enters the right ventricle and the pulmonary artery ; from here only a small portion of the blood enters the lungs, while the greater portion passes through the ductus arteriosus Botalli into the aorta. Thus the fetus receives none but mixed blood, for together with the blood from the umbilical vein, the liver also receives blood which has already been used from the portal vein. The liver, the head, and the upper extremities are supplied with blood richly laden with oxygen, while the lower half of the body receives blood poor in oxygen. The blood in the lungs is purely venous.

After birth the expansion and congestion of the lungs causes the pressure in the left auricle to lessen and to equal that of the right auricle, on account of which the foramen ovale closes. The ductus arteriosus Botalli receives less blood, and the change in the position of the lungs causes it to become constricted, thrombosed, and obliterated. The three umbilical vessels and the ductus Arantii likewise become obliterated on account of cessation of the blood current after the maternal and fetal bodies are separated.

THE SKELETON

The skeleton during early infancy is soft, easily deformed, and is undeveloped.

Skull.—In the newborn infant the cranial is much larger than the facial portion of the skull ; and, therefore, the palate and nose are very narrow and the facial musculature but poorly developed. Body equilibrium has not yet been established, and the skull when at rest falls backward in the newborn, instead of forward as in

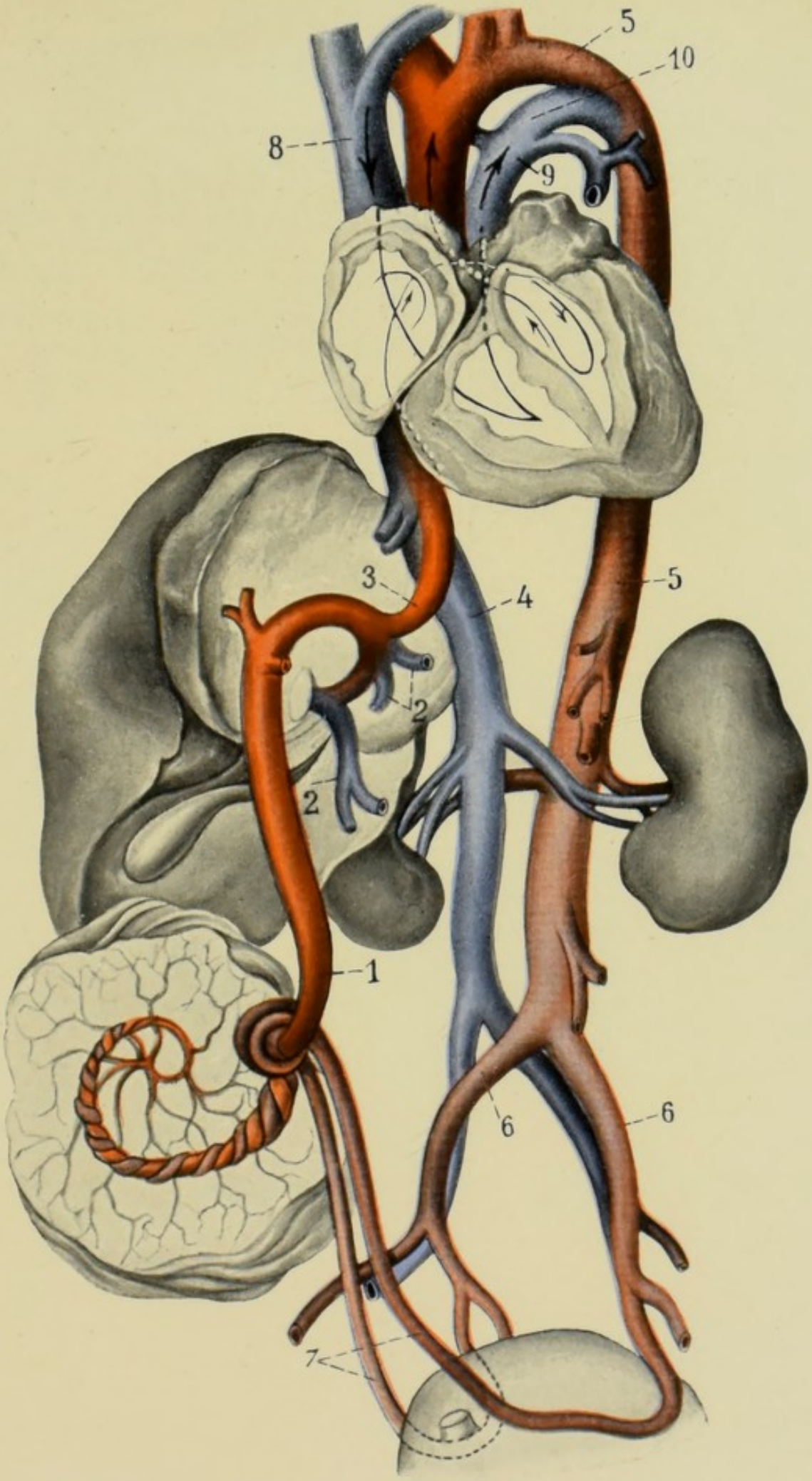


Fig. 1.

adults. The face gradually reaches full development through the growth of the upper and lower jaws, especially the ascending rami of the inferior maxilla and the alveolar processes, and through eruption of the teeth and elevation of the bridge of the nose. The cranium is still wide open at the anterior fontanel; the latter is formed by the frontal and parietal bones, is rhomboidal in shape, with the acute angle pointing forward, and

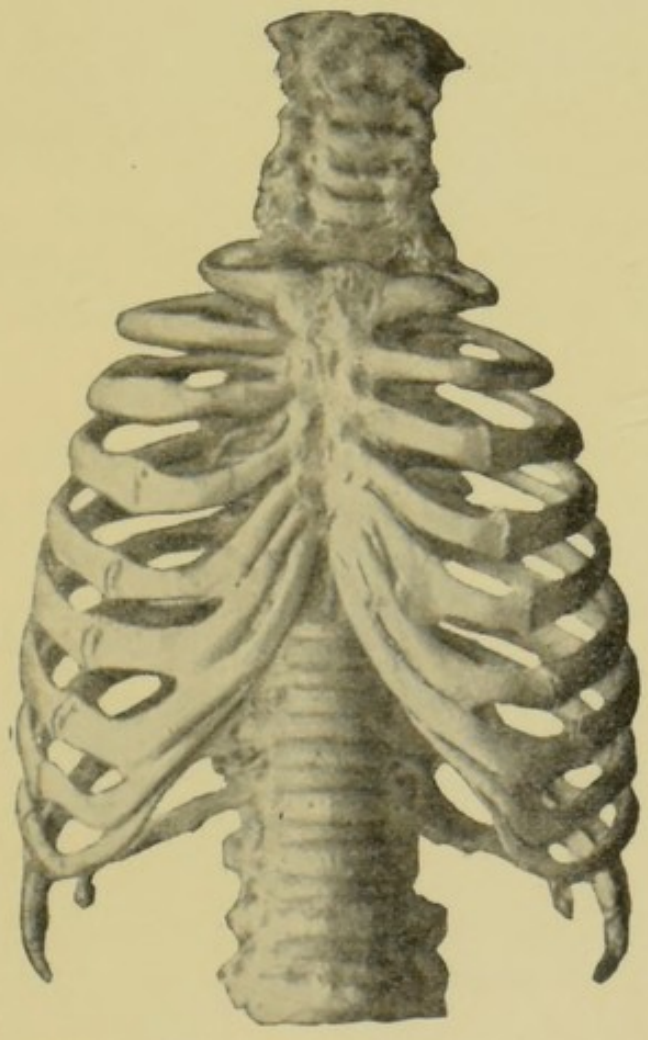


FIG. 2.—Thorax of a newborn infant. Funnel-shaped, horizontal upper aperture; at full inspiration. (From a preparation in the Anatomic Institute in Munich.)

closes in the twelfth to the fifteenth [in exceptional cases as late as the twentieth to the twenty-fourth] month after birth. The small fontanels between the parietal and occipital bones are represented after birth only by a superficial depression, while the anterior and posterior lateral

PLATE 1

The Skulls of a Newborn Infant, of a Six-year-old Child, of an Adult Man, and of an Old Man.—The different relationships between the cranial and facial portions of the skull are shown, also the gradual elevation of the face, the development of the ascending rami of the lower jaw, and the similarity between the infantile skull and that of old age. (From preparations in the Anatomic Institute in Munich.)

PLATE 2

Skulls of a Newborn Infant (from in front and from above), **of a Six-year-old Child, and of a Man.**—Showing the persistence of the fontanels and the sutures, and the development of the facial portion of the skull. (From preparations in the Anatomic Institute in Munich.)

---

fontanels are completely closed. The anterior are formed by the junction of the frontal, temporal, and sphenoid bones; the posterior by union of the parietal, temporal, and occipital bones. The sutures are loose or still gape somewhat.

Thorax.—The thorax, which is circular on cross-section, approaches more nearly the shape of a funnel than that of a cylinder. The anterior wall is higher and a greater distance from the spinal column than in the adult; this is the inspiratory or emphysematous posture. The upper aperture and the ribs lie in a horizontal position. The anterior wall gradually descends and the sagittal diameter becomes smaller on account of the weight of the hanging arm, tension of the abdominal muscles, and the relative diminution of the liver and spleen.

The transverse section loses its circular form and becomes oval in shape, the lateral being longer than the anteroposterior diameter. (Asthmatics possess a permanent infantile form of thorax.)

Pelvis.—The pelvis is still largely cartilaginous and highly movable. The promontories are only partially developed and the position is more horizonal. The development of the pelvis proceeds with apposition at the symphysis, at the lateral processes of the sacrum, and at the synchondroses of the ilium, os pubis, and sacrum, as well as the forward growth of the promontory of the sacrum.

Vertebræ.—Aside from the promontories the subsequent









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curvatures of the spine are still absent or only indicated, and the vertebræ appear to form a straight line. The final shape of the spine is due to the weight of the body and the traction of the muscles. Voluntary raising of

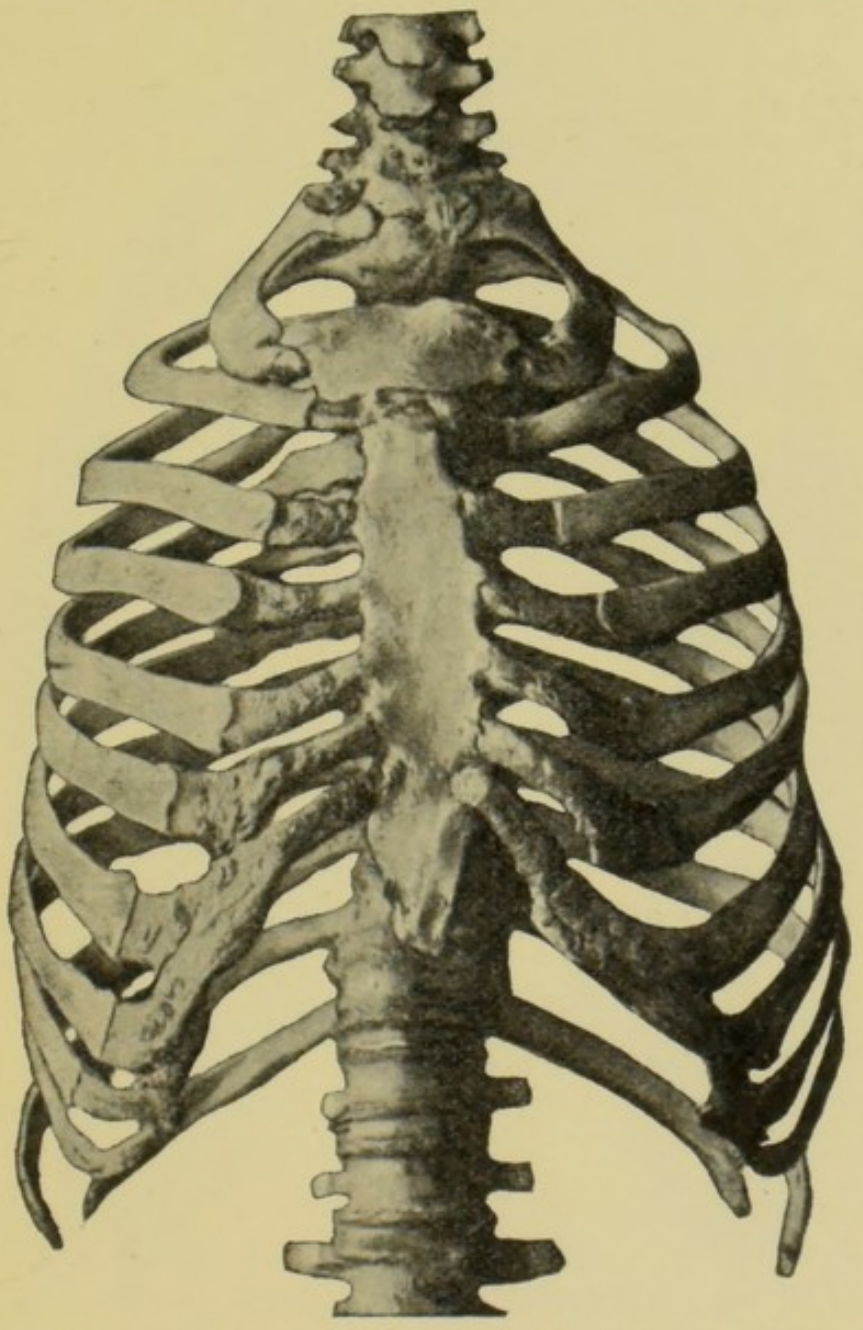


FIG. 3.—Thorax of an adult man. The sternum and ribs have descended; the upper aperture is bent downward; the sternum is nearer the spinal column. (Preparation from the Anatomic Institute in Munich.)

the head in from two to three months forms the cervical curvature; the standing posture at about the twelfth month, the traction of the erector muscles of the trunk,

and the weight of the abdominal organs tend to increase the lumbar curvature. The weight of the body in sitting, the traction of the shoulder and the rectus muscles, help to form the dorsal curvature.

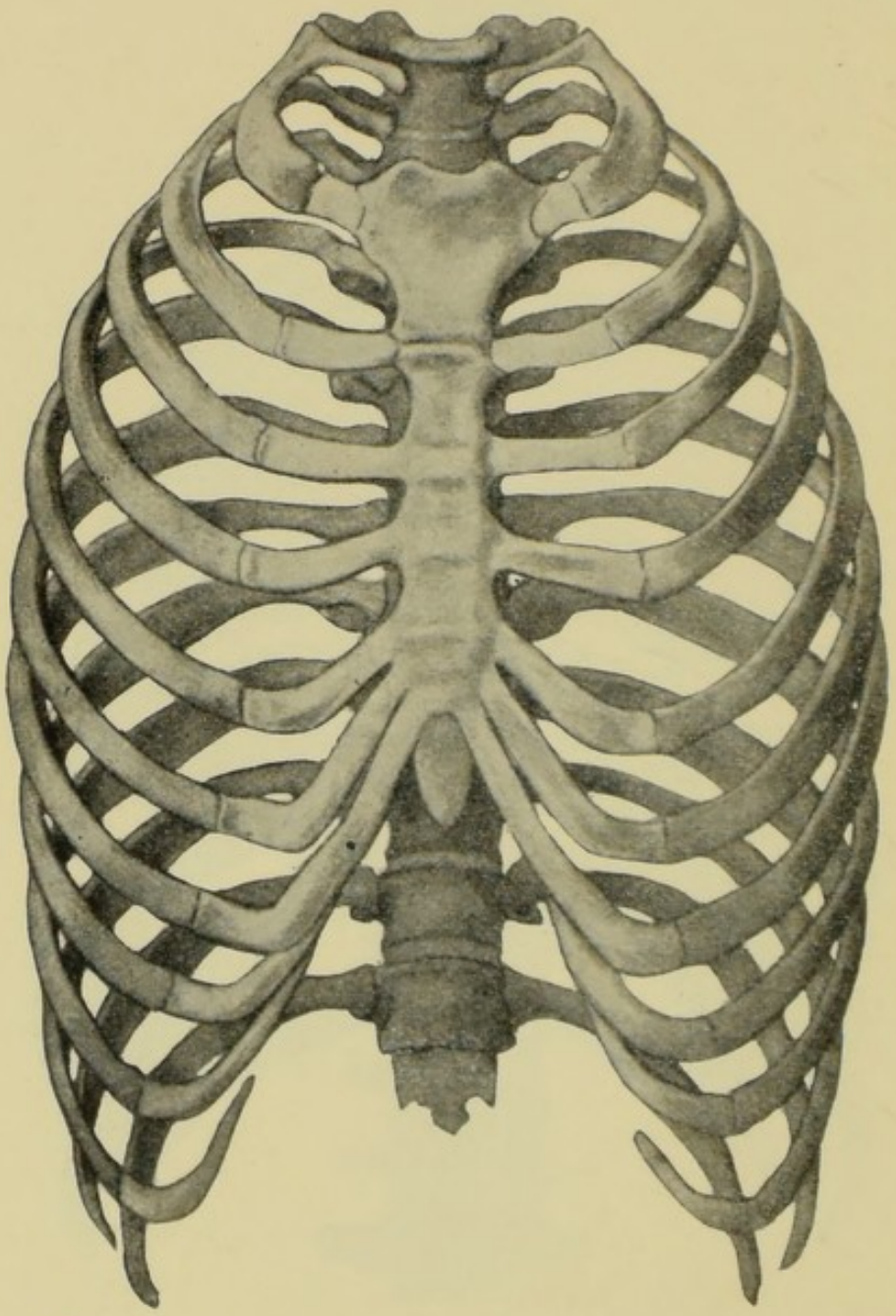


FIG. 4.—Thorax of an adult woman. The descent of the anterior wall of the thorax is even more pronounced than in man. (From a chart in the Anatomic Institute in Munich.)

Extremities.—Aside from the undeveloped state of the neck of the humerus, there are no great differences in form; the foot, which originally is poor in fat, later, in

the crawling and walking period, becomes encased in fat, showing the same skeletal structure as in the adult (Spitzy). The epiphyses of the long cylindric bones, the hand, and the tarsal bones are still cartilaginous; their ossification is not completed until nearly the sixteenth year (Rauber, von Ranke).

The **body surface** is much greater than in adults; to each kilogram [2.2 lbs.] of body weight there are 810 sq. mm. [12.5 sq. in.] of surface in the newborn, 620 sq. mm. [9.6 sq. in.] in infants six months old, 450 sq. mm. [6.9 sq. in.] in seven-year-old children, and 320 sq. mm. [4.9 sq. in.] in adults.

INTERNAL ORGANS

The Thymus Gland.—This organ, occurring only in children, lies in the anterior mediastinum and is concerned in the formation of blood. Its size varies in individuals of the same age; it is from 2 to 7 cm. [.8–1.4 in.] in width and from 5 to 10 cm. [2–4 in.] in length, and weighs in the newborn on an average 12 gm. [191 gr.]. The gland continues to grow during the first year of life, after which it diminishes in size on account of atrophy of the glandular substance, which is replaced by connective tissue, and disappears at the time of puberty.

The Liver.—This organ is relatively larger and heavier than in adults. It weighs in newborn and nursing infants one-twentieth of the body weight, whereas in adults it weighs only one-fiftieth of the total weight of the body. The lower edge runs obliquely from the crest of the right ilium above the umbilicus toward the left to the region of the fundus of the stomach. The left lobe reaches the left anterior axillary line.

The Kidneys.—These are lobulated and comparatively large. The histologic structure of the liver, kidneys, and pancreas at the time of birth is still in a transitional stage of the process concerned in the formation of a definite structure. The liver and kidneys probably still possess for a time after birth the fetal blood-producing function.

FIG. 5.—Median section of a newborn infant. Aside from a slight promontory curvature, the vertebræ still form an almost perfectly straight line. (From a preparation in the Munich Gynecologic Clinic.)

FIG. 6.—A median frozen section through the body of a six-year-old boy. The vertebral column shows a slight cervical and dorsal curvature and a fairly well-developed promontory. The spine is, however, still quite straight, especially in the lower dorsal and lumbar portion. A physiologic lumbar lordosis is distinctly marked in life (J. Symington).

FIG. 7.—Median section through the skeleton of an adult man. The curvatures of the spinal column are fully developed; the anterior wall of the thorax has descended and the pelvis tilted. (Preparation in the Anatomic Institute of Munich.)

The Stomach.—The stomach occupies a more vertical position, the fundus is but slightly formed, and the musculature, especially about the cardia, is but poorly developed. The normal position and shape are developed in the course of the first year. The mucosa, with its rich blood supply, is more sensitive to thermal and chemic irritants, which together with the position of the organ, the small fundus, and weakness of the cardia, all explain the tendency of the infant to vomit. The capacity of the stomach is increased from about 40 ccm. [1.3 oz.] at the time of birth to from 300 to 400 ccm. [10–13.5 oz.] at the end of the first year of life.

The Intestines.—The length of the intestines in nursing infants is six times that of the body, whereas in adults it is only four and a half times the body length. The capacity in the new born is 5000 ccm. [10 pints], in twelve-year-old children 9000 ccm. [18¾ pints], and in adults only 4000 ccm. [13 pints] to 1 kilogram of body weight. The mucosa is sensitive and contains incompletely developed glands. The weakness of the musculature favors constipation and explains the frequent tendency to dilatation and enteralgias. The intestines possess a great absorptive capacity, but relatively deficient digestive power. The colon runs—without an hepatic flexure parallel to the edge of the liver—obliquely from the right iliac crest upward toward the left.

The Nervous System.—The dura is attached to the cranium. The brain is large and heavy and equals 13 to

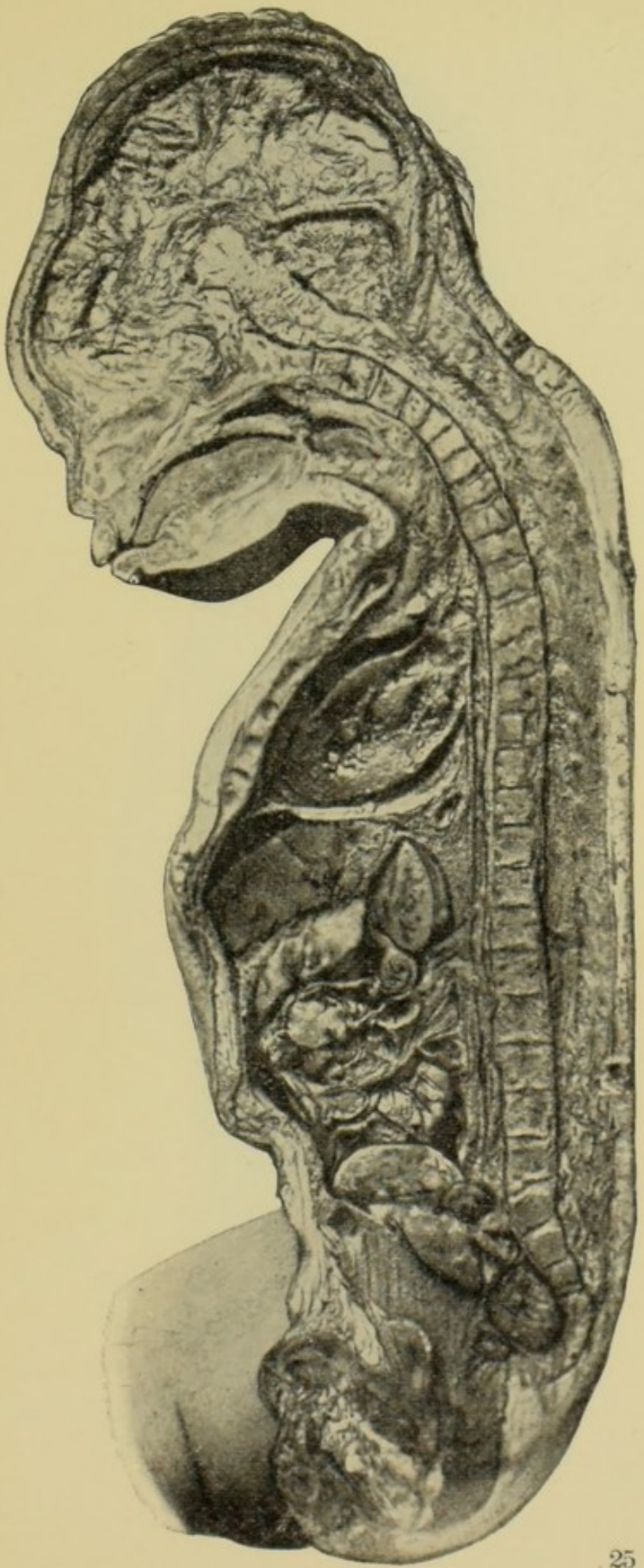


FIG. 5.

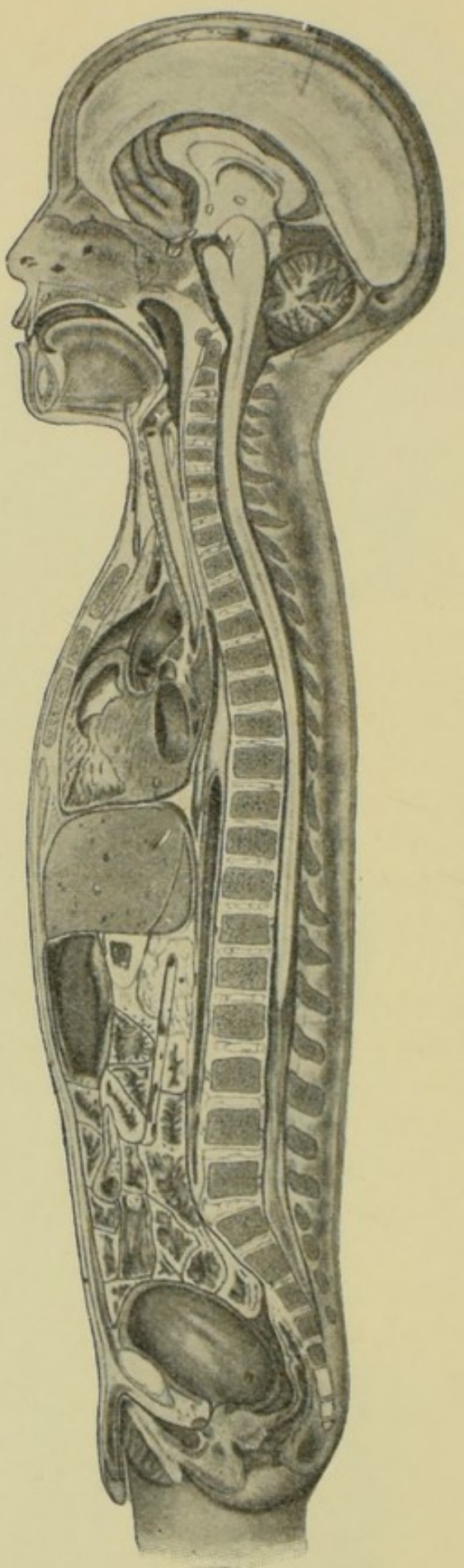


FIG. 6.

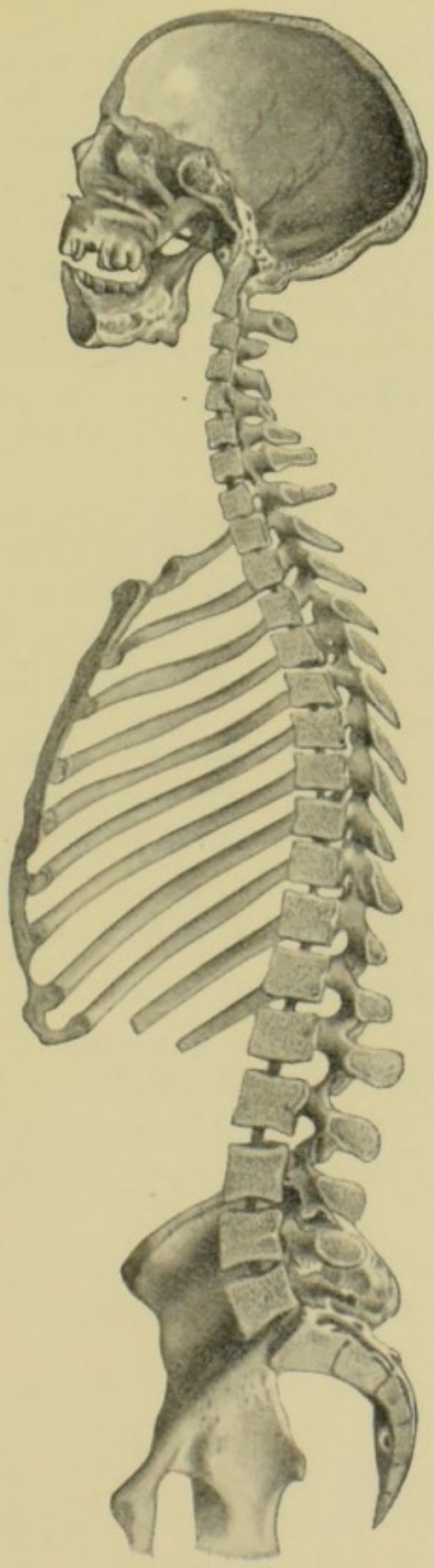


FIG. 7.

14 per cent. of the body weight, in contradistinction to 2.7 per cent. in adults. It grows very rapidly during the first year, at the end of which time it reaches one-half of its permanent weight. The convolutions are but slightly differentiated and there are but few medullated nerve-fibers. The psychomotor subcortical inhibitory centers are but slightly excitable, as are also the peripheral sensory and motor nerves during the first six weeks (Soltmann, Westphal).

The **musculature** is relaxed, pale, watery, and is readily fatigued.

The **adipose tissue** is present in large amounts. In the cheeks, where it possesses distinct anatomic boundaries, it forms a cushion of fat, and is of assistance in the act of sucking (von Ranke). When the baby begins to walk about he loses much of this fat; that of the cheek is the last to disappear.

Female Genitals.—These are not closed. The labia minora, hymen, and urethra are visible, and therefore the infant is predisposed to vulvovaginitis and cystitis.

PHYSIOLOGIC PECULIARITIES

GROWTH IN LENGTH

The average length of a newborn infant is 50 cm. [20 in.], in boys 51 cm. [20.2 in.] and in girls 49 cm. [19.8 in.]. The length of the body at various ages, according to E. von Lange, is as follows:

Age in months	Birth.	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.
Body length (cm.)	49.5	55.2	58.5	61.0	63.0	64.7	66.2	67.5	68.8	69.9	71.0	72.0	73.0
[Body length (in.)]	19.8	22.0	23.4	24.4	25.2	25.8	26.4	27.0	27.5	27.9	28.4	28.8	29.4

The increase in the length of the body is practically the same for both boys and girls during the first two years, but from then until the thirteenth year the female child grows more slowly. After the thirteenth year,

however, because of their earlier period of puberty, girls grow more rapidly and overtake the gain made by the males previous to that time. After its fifteenth year, however, the female child grows less in length than the male.

During the first year the child grows about 23 cm. [9.2 in.]; in the second year, about 10 cm. [4 in.]; in the third year, 8 cm. [3.2 in.], and in the fourth year, 7 cm. [2.8 in.]. In four years it has doubled its growth and in fourteen years trebled it.

Body Length, According to E. von Lange

Age in years.	Boys.	Girls.	Age in years.	Boys.	Girls.
1	73.0	73.0	10	130.7	130.0
2	83.1	83.1	11	135.0	134.6
3	91.5	91.3	12	139.2	140.3
4	99.0	98.7	13	143.8	147.6
5	105.4	105.0	14	149.7	153.8
6	111.2	110.7	15	156.7	157.3
7	116.5	116.0	16	163.5	159.0
8	121.5	120.9	17	167.6	159.7
9	126.2	125.6	18	169.4	159.9

SKULL AND CHEST MEASUREMENTS

Of the skull measurements the fronto-occipital periphery is to be measured over the most prominent portion of the frontal and occipital bones. Aside from this measurement, in the case of cranial disease, the following distances are to be measured: The bitemporal from one external ear to the other; the fronto-occipital diameter from the glabella to the occipital protuberance; the biparietal, from one parietal protuberance to the other. The circumference of the chest is taken midway between inspiration and expiration while the arms are held in a horizontal position.

The circumferences of the skull and chest increase symmetrically until about the fifth year, but after that

time the chest grows more rapidly. It is to be noted that the circumference of the chest usually exceeds one-half of the body length by from 9 to 10 cm. [3.6–4 in.].

Head and Chest Measurements (Heubner)

Age.	Circumference of head.	Circumference of chest.
1 month	35.4 cm. [14.1 in.]	34.2 cm. [13.6 in.]
6 months	42.7 " [17.0 "]	41.0 " [16.4 "]
1 year	45.6 " [18.4 "]	46.0 " [18.4 "]
2 years	48.0 " [19.2 "]	47.3 " [18.9 "]
4 years	50.0 " [20.0 "]	49.0 " [19.6 "]
5 years	50.0 " [20.0 "]	52.0 " [20.8 "]
8 years	51.3 " [20.5 "]	58.0 " [23.2 "]
12 years	52.3 " [20.9 "]	65.0 " [26.0 "]

INCREASE IN WEIGHT

The average weight of a newborn infant is 3250 gm. (extremes 2500–4000 gm. and over), boys weighing a little more than girls. The physiologic loss of weight during the first three or four days equals about 200 gm. This depends upon the lack of proportion between ingestion and excretion; discharge of meconium and urine, excretion through skin and lungs, and the small amount of nutritive material ingested. In from five to eight days the original weight is usually regained.

The weight of the body increases steadily or intermittently with diminishing rapidity.

The daily increase in weight is originally 30 gm.; but after a year only about 10 gm. There is, as a rule, a loss of weight during the ninth month of life (dentition, change of nutrition) and in the first year of school life. At the end of five months the weight is about double the original weight; after a year, three times; after six years, six times; and after the thirteenth to the fourteenth years, twelve times the weight at birth. Bottle-fed infants weigh less than breast-fed children up to nine months of age, but after that time soon regain this loss and later show no difference.

Tables of Weights, According to Heubner

(*a*) First year of life (breast-fed infants).	Gm.
Original weight	3433
End of 4th week	4008
End of 8th "	4907
End of 12th "	5600
End of 16th "	6294
End of 20th "	6824
End of 24th "	7289
End of 28th "	7774
End of 32d "	8175
End of 36th "	8655
End of 40th "	8855
End of 44th "	9232
End of 48th "	9589
End of 52d "	1,0141

(*b*) Between 2 and 18 years of age.	Boys.	Girls.
At the end of 2 years	13.2 kg.	12.0 kg.
At the end of 3 years	15.4 "	14.0 "
At the end of 4 years	16.8 "	15.7 "
At the end of 5 years	19.3 "	17.5 "
At the end of 6 years	21.1 "	19.0 "
At the end of 7 years	23.0 "	20.7 "
At the end of 8 years	24.9 "	22.5 "
At the end of 9 years	26.8 "	24.9 "
At the end of 10 years	29.4 "	26.4 "
At the end of 11 years	32.1 "	29.1 "
At the end of 12 years	34.9 "	33.7 "
At the end of 13 years	38.2 "	37.9 "
At the end of 14 years	42.6 "	42.6 "
At the end of 15 years	51.0 "	47.2 "
At the end of 16 years	57.1 "	48.2 "
At the end of 17 years	62.7 "	49.2 "
At the end of 18 years	66.0 "	50.0 "

The increase in length and weight are not proportionate. During the period of active body growth the increase in height exceeds that of the weight (Axel Key). During the summer there is usually a decided increase in height, but only a slight increase in weight, whereas during winter the reverse holds true (Malling, Hansen).

Table of the Average Height, Weight, Head Circumferences, and Chest Measurements of American Boys and Girls. (From Koplik.)

Years of age.	Sex.	Height.		Weight.		Head circumference.		Depth of chest.		Breadth of chest.		Chest expansion.	
		In.	Cm.	Lbs.	Kg.	In.	Cm.	In.	Cm.	In.	Cm.	In.	Cm.
5½	Boys	41.7	105.9	41.6	18.9	20.1	51.2	4.9	12.3	7.1	18.1	1.3	3.4
	Girls	41.3	104.9	40.7	18.5	19.7	50.2	4.8	12.3	7.0	17.7	1.4	3.5
6½	Boys	43.9	111.9	45.2	20.5	20.2	51.5	5.0	12.8	7.2	18.4	1.6	4.2
	Girls	43.3	109.0	43.4	19.5	19.8	50.3	4.9	12.3	7.0	17.7	1.5	3.8
7½	Boys	46.0	116.8	49.5	22.5	20.4	51.9	5.1	12.9	7.4	18.9	1.8	4 5
	Girls	45.7	116.0	47.7	21.6	20.0	50.9	4.9	12.5	7.2	18.4	1.8	4.5
8½	Boys	48.8	123.9	54.5	24.4	20.5	52.2	5.1	12.8	7.6	19.4	2.3	5.9
	Girls	47.7	121.1	52.5	23.8	20.2	51.2	4.9	12 5	7.4	18.9	2.0	5.0
9½	Boys	50.0	127.0	59.6	27.0	20.6	52.4	5.2	13.2	7.8	19.7	2.5	6.5
	Girls	49.7	126.2	57.4	26.0	20.4	51.9	5.1	13.1	7.0	19.3	2.2	5.6
10½	Boys	51.9	131.8	65.4	29.5	20.6	52.6	5.2	13.2	8.0	20.2	2.7	7.0
	Girls	50.7	131.3	62.9	28.5	20.5	52.0	5.1	13.0	7.8	19.8	2.4	6.0
11½	Boys	53.6	136.1	70.7	32.2	20.8	52.9	5.4	13.8	8.2	20.9	2.9	7.3
	Girls	53.8	136.6	69.5	31.5	20.7	52.5	5.2	13.1	8.0	20.3	2.6	6.6
12½	Boys	55.4	140.7	76.9	34.9	21.0	53.3	5.6	14.1	8.5	21.5	3.0	7.8
	Girls	56.1	142 5	78.7	35.7	20.9	53.0	5.4	13.8	8.4	21.0	2.4	6.2
13½	Boys	57.5	146.0	84.8	38.5	21.1	53.5	5.6	14.3	8.7	22.1	3.2	8.2
	Girls	58.5	148.6	88.7	40.3	21.0	53.5	5.5	14.1	8.7	22.1	2.6	6.6
14½	Boys	60.0	152.3	95.2	43.2	21.3	54.1	5.9	15.0	8.9	22.7	3.3	8.4
	Girls	60.4	153.4	98.3	44.6	21.3	54.1	5.7	14.5	9.0	22.9	2.7	6.8
15½	Boys	62.9	159.7	107.4	48.8	21.4	54.5	6.3	16.0	9.3	23.6	3.3	8.4
	Girls	61.6	156.4	106.7	48.5	21.5	54.6	6 0	15.3	9.5	23.8	2.6	6.5

Table of Weight, Length, Head Circumference, and Girth of Chest from Birth to the End of the Fourth Year. (From Koplik.)

Age.	Sex.	Length.		Weight.		Head circumference.		Chest girth.	
		In.	Cm.	Lbs.	Kg.	In.	Cm.	In.	Cm.
Birth	Boys	19.7	50 0	7.4	3.45	13.8	35.1	12.6	32.0
	Girls	19.3	49.0	7.1	. . .	13.1	33.4	11.8	30.0
6 months	Boys	25.4	64.8	16.0	7 2	16.0	40.5	15.7	39.9
	Girls	25.0	63.6	15.5	7.0	16.4	41.7	15.2	38.6
12 months	Boys	29.5	73.8	21.5	9.8	17.8	45.3	I7.8	45.1
	Girls	28.7	73.2	21.0	9.5	18.2	46.3	19.0	48.3
2 years	Boys	33.8	84.5	30.3	13.8	19.3	49.0	20.0	50.8
	Girls	32.9	82.8	29.2	13.3	18.0	45.6	18.0	48.0
3 years	Boys	37.0	92.6	34.9	15.9	19.3	49.0	20.1	51.1
	Girls	36.3	90.7	33.1	15.0	19.0	48.4	19.8	50.5
4 years	Boys	39.3	98.2	37.9	17.2	19.7	50.3	20.7	52.8
	Girls	38.8	97.0	36.3	16.5	19.5	49.6	20.5	52.2

Daily Increase in Weight in Grams, According to Camerer

Week.	Breast-fed children.	Artificially fed children.
1	20	4
2 to 12	31 to 26	21 to 22
12 to 24	24 to 18	22
24 to 36	15 to 16	13 to 16
36 to 40	9	9
40 to 52	12	12

Condition of the Skin.—In the newborn there is a flesh-red color, and a physiologic yellow discoloration frequently prevails for from two to six days. In the course of two or three weeks a permanent rose-red color develops.

Icterus neonatorum occurs in 80 per cent. of the newborn and progresses without discoloration of the feces and without the usual bile-stained urine. The conjunctivæ are slightly yellow and the jaundice is chiefly limited to the upper half of the body. Children who are decidedly icteric suffer considerable loss of weight during the first week of the disease and show a tendency to disturbances of digestion. Icterus of long duration usually indicates the existence of a pathologic condition (syphilis of the liver, obliteration of the bile-ducts). The etiology is not yet perfectly understood.

The skin shows during the first weeks an increased activity. There is nearly always a certain degree of exfoliation in the second or third week, often accompanied by swelling and redness, especially of the toes and fingers. There is an increased secretion of sebaceous matter from the scalp, with the formation of fatty scales, which are composed of sebum and epithelium. The secretion of sweat is slight, but in the case of rachitic and bottle-fed children may be quite profuse. The nodules which sometimes occur in the skin of the face are due to secretions from the sebaceous glands (Epstein) and disappear in the course of time.

The Blood and its Circulation.—The blood does not reach its definite stage of development until the fourth or fifth

year. The number of the red cells as well as the white corpuscles is increased during the first year. Two-thirds of the leukocytes are mononuclear lymphocytes and only one-third are polynuclear leukocytes (Metchnikoff); it is for this reason probably that little children are susceptible to infections (Heubner).

The blood current is more rapid than in adults; it takes 12 to 15 seconds to complete the circulation in the infant, whereas 22 seconds are required in the adult.

The *pulse* in healthy children often shows irregularity in force and frequency. Even slight affections may cause a disproportionate increase in frequency. The average rate during the first six months is 130 per minute; at the end of the first year, 120; in three years it is 110, and in nine years, 82 (girls 92).

Respiration.—The number of respirations during sleep averages in the newborn 35.3; in the second year, 28; in the third and fourth year, 25. The depth of the inspirations and the length of the intervals are also irregular in sleep. The respiration, on account of the inspiratory position of the thorax during the first year, is chiefly diaphragmatic. Not until the tenth or twelfth year is the adult type reached. The first inspiratory movement follows irritation of the respiratory center by the blood which has become charged with CO_2 during birth.

Sleep.—A child sleeps during the first weeks of life twenty hours per day; at the end of a year, for from twelve to fifteen hours; between the second and third year, eleven to fourteen hours; from the fifth to the seventh year, ten to eleven hours; in the seventh year, ten hours, and in the twelfth year, nine hours. These figures vary, of course, in different individuals. The sucking infant sleeps, as a rule, several hours after nursing.

Temperature.—The temperature of the newborn is from 37.8° to 38.5° C. [101.6°–102.2° F.]. From the second day on the temperature is that of an adult, 37.0° to 37.5° C. [98.6°–100° F.]. It rises during crying spells and when the child is given nourishment, and sinks during sleep and when bathed (about 0.1°–0.5° C. [.1°–.9° F.]).

Heat production and heat dissemination are greater in a child than in an adult. According to Vierordt the ratio is as follows:

1 kg. of body weight of the newborn	130,000 calories.
1 kg. of body weight of one and a half years	91,000 "
1 kg. of body weight of an adult	39,000 "

The greater degree of heat dissemination depends upon the relatively larger amount of surface, the more rapid circulation, and the increased respiration rate.

Excretion of Urine.—The urine is already excreted *in utero*. It is scarce during the first two days of life, and then increases in proportion to the amount of fluid ingested. When 100 gm. of milk are taken, the urinary output amounts to 60 to 70 gm. (Bendix). The daily amount of urine secreted is as follows: first day, 17 gm.; second to third day, 40 to 50 gm.; eighth day, 250 gm.; six months, 500 to 600 gm.; after which it rises to one liter during the years of puberty. The urine during the first days of life contains albumin, tubular casts, and crystals of uric acid. (For examination of urine, see Renal Diseases.)

Special Senses.—*Vision.*—Newborn infants are sensitive to light and cannot bear diffuse daylight until after the second or third week. Light and dark are distinguished after a few days. Objects can be focussed after four or five weeks. Primarily the eye is myopic because of greater curvature of the cornea. [Some authors maintain that the eye is hypermetropic during the first days of life.—Ed.]

Hearing.—Deafness exists during the first twenty-four to thirty-six hours, because of swelling of the mucous membrane of the middle ear. In the course of a few weeks the infant hears noises; during the whole first year a disagreeable sensation is produced by shrill tones and noises. The other special senses are developed, with the exception of the sense of space, which becomes functional with the enlarged experiences of later childhood.

Movements.—The first movements are reflex and auto-

PLATE 3

FIG. 1. **The Upper and Lower Jaws of a Child during the Second Dentition.**—The lower incisor teeth have erupted abnormally previous to the molar teeth. (The upper jaw has been divided in the median line.)

FIG. 2. **Complete Set of Milk Teeth.**—Showing the position of the permanent tooth crowns above and behind the adventitious roots. (From a preparation in the Anatomic Institute of Munich.)

matic. The first voluntary movement is lifting of the head; the ability to grasp voluntarily does not occur until the second or third month; sitting, the sixth month; standing and walking, after one year.

Dentition.—The temporary teeth erupt in the following order:

1. The lower central incisors in from five to eight months.
2. The upper central incisors in from six to nine months (four weeks later).
3. The upper lateral incisors soon thereafter.
4. The lower lateral incisors at the end of the first year of life.
5. The first molars soon thereafter.
6. The canine teeth about the middle of the second year of life.
7. The outer molars from the twenty-second to the thirtieth month.

There is considerable variation in different individuals as to the order and period of eruption. The latter is delayed in rachitis, syphilis, and tuberculosis.

The second dentition begins about the sixth year, and the various teeth erupt as follows:

1. The first molars in the fifth or sixth year.
2. The middle incisors in the sixth to ninth year.
3. The lateral incisors in the seventh to tenth year.
4. The first bicuspids in the ninth to thirteenth year.
5. The canine teeth in the ninth to fourteenth year.
6. The second bicuspids in the tenth to fourteenth year.
7. The second molars in the tenth to fourteenth year.
8. The third molars in the sixteenth to fortieth year.

The first dentition proceeds either without symptoms



Fig. 1.

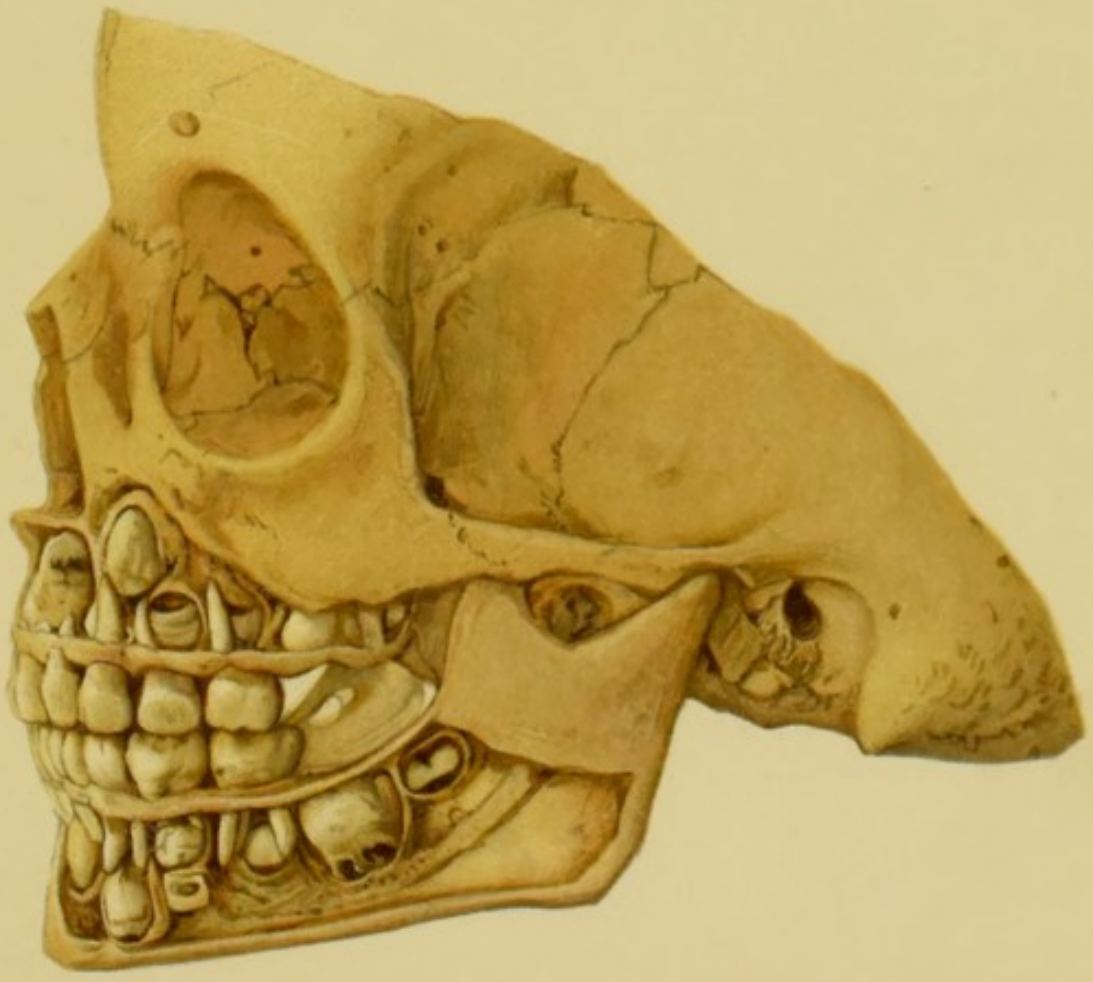


Fig. 2.

or it shows certain phenomena. Thus pains may occur before or during the eruption. There may also be swelling and reddening of the gums, salivation, flushed cheeks, and styes [?]. Symptoms may occur because of an increased excitability of the nervous system: restlessness, crying, convulsions, an irritable cough, frequency of urination, diarrhea, vomiting, eruption of the so-called "tooth-rash" (see Strophulus infantum). [It is very doubtful whether all these symptoms are produced by dentition. We would grant that local discomfort and general restlessness might occur at this time. It does not seem in accord with modern pathologic conceptions to assume that convulsions, cough, vomiting, diarrhea, etc., can result from teething. These—for the most part acute conditions—disappear very shortly under appropriate treatment, though the process of dentition still continues.—Ed.]

The resorption of the milk teeth begins with the disappearance of the last deciduous teeth by the crowding onward of the permanent teeth. The latter lie almost completely developed back of and underneath the milk teeth, and on account of insufficient space are frequently pushed upward or downward. Thus the particularly large canine tooth is displaced as far as the infra-orbital foramen.

DIGESTION

The oral apparatus of the suckling functionates only as a pump; chewing and salivation are impossible on account of the weak muscles of mastication, the lack of teeth, and the insufficiency of saliva. The oral cavity of a newborn child is dark red in color and dry. Only traces of the diastase ferment are present, but appear in appreciable quantities after the second month. The infant can, therefore, ingest nothing but liquids.

Pepsin, hydrochloric acid, and rennin are secreted in the stomach. The casein of the milk is precipitated, and the free hydrochloric acid becomes united with the casein, which is partially dissolved. From the sugar of milk

lactic acid is formed. Aside from the above action hydrochloric acid has a bactericidal one also. The nutriment leaves the stomach of artificially fed children in from three to four hours, while in breast-fed children it passes out in from one and a half to two hours, that organ serving only as a collecting and preparatory station. The fate of the food in the intestines through which it passes in six to eight hours is as follows: The pancreatic juice and the bile alter its reaction and color; the albumin digestion continues; the casein becomes peptonized; the fats are split up, saponified, and absorbed; the sugars and dissolved salts are absorbed, as are also the albumins.

In the large intestine the water and the remaining unabsorbed dissolved substances are absorbed. The bacteria, which are normally present in the large and small intestines (Bacillus acidophilus—Moro; B. coli and B. lactis aërogenes—Escherich), are important for the purpose of proteid digestion, fermentation of milk-sugar, protection against invading pathogenic micro-organisms, and to excite peristalsis. The undigested portions of the food and the residue of the digestive juices pass out as feces and eventually form pathogenic products. The stools of the first few days—the meconium—are blackish green in color, odorless, acid, and composed mainly of the digestive agents excreted by the intestines. Microscopically they contain cylindric epithelium, mucoid bodies, fat globules, cholesterin, and minute hairs.

Next follow transitory stools, which partake of the color of meconium and milk stools. The normal breast stool is golden yellow in color, of the consistency of soft paste, frequently somewhat nodular, almost odorless, and slightly acid in reaction. The normal stool of babies fed on cows' milk is paler and of a lighter yellow color, it possesses the consistency of paste, and is formed. As a rule it has an acrid odor, slightly acid or alkaline in reaction, and is discharged in larger quantities. After the ingestion of a meal, especially cereal foods, the stools become brownish in color. The constituents of the stools are 85 per cent. water, casein, fatty acid salts, mucus,

sodium chlorid, cholesterin, and bilirubin. A microscopic examination shows micro-organisms, fat, epithelial cells, and vegetable débris.

NOURISHMENT

NATURAL FEEDING

The only form of nutriment which is fully adequate for the nourishment of a child's body is that derived from its *mother's milk*. Such nourishment should be attempted in every case, excepting when the mother is suffering from pronounced tuberculosis, for it is also of advantage to her (favors prompt involution of the genitals, protection against conception, and has a tendency to improve the nourishment of her own body). When there is really insufficient milk, artificial feeding should be accompanied by at least several attempts to feed from the breast, as this *mixed feeding* is of greater value to the child than a purely artificial diet.

Constituents of Human Milk.—Water, albuminoids, fat, sugar, and salts. Aside from these elements, human milk contains also unknown antitoxins and immune bodies, as well as a number of ferments (Escherich, Moro), which are of importance for internal metabolism.

After a period of nursing the following characteristics are noted:

Colostrum is the name given to the milk during the first eight days. It is richer in albumin and salts, but poorer in fat, and contains larger amounts of the so-called colostrum bodies—*i. e.*, fatty degenerated mammary epithelium (see Fig. 8, *b*).

The *milk of the first two months* shows an increase of albumin, salts, and colostrum bodies, and gradually comes to resemble the permanent milk.

Permanent milk remains nearly constant as to its constituents during the whole period of lactation; it no longer contains colostrum bodies, and the fat globules

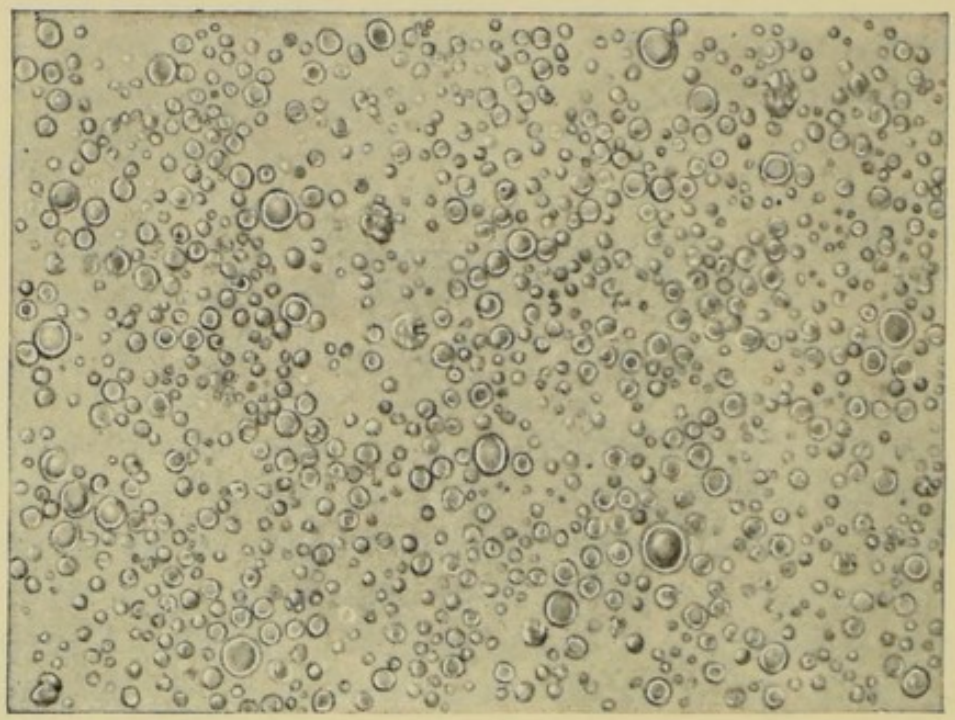
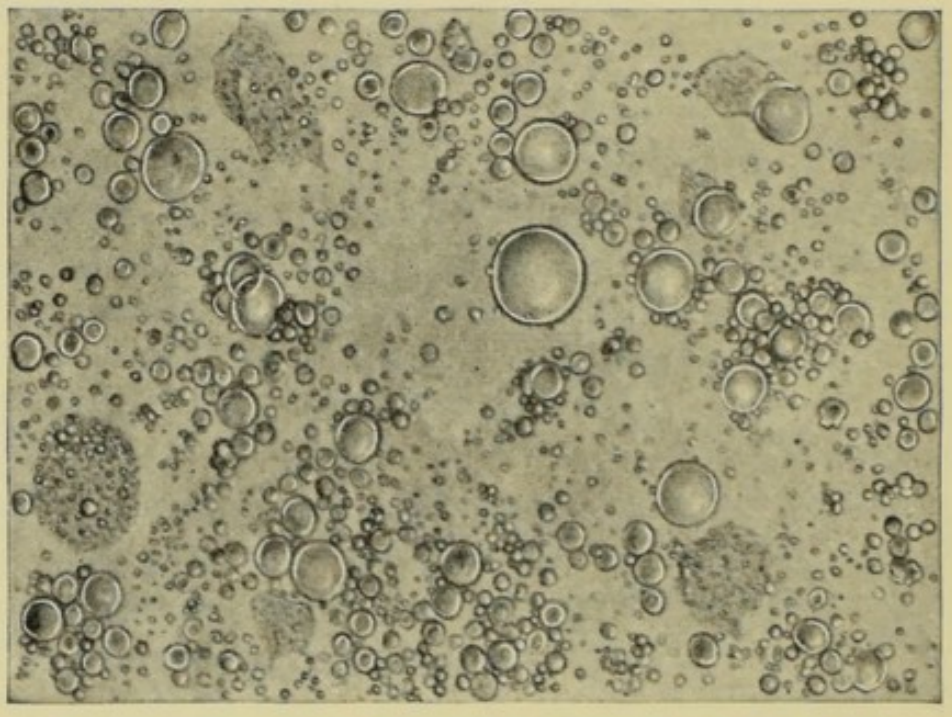


FIG. 8.—(*a*) Maternal milk. The fat globules are of varying size, but show, as a rule, equal subdivision ; no colostrum bodies.



(*b*) Colostrum. The fat globules are unequally divided, agglutinated in certain areas, and show marked differences in size (large fat vacuoles) ; the colostrum bodies may be recognized as pale gray areas partially covered with fat.

are of varying size, but nearly similar in form (see Fig. 8, *a*).

Comparison of Various Milks (Heubner and others)

100 gm. milk contain in grams:

Source.	Albumin.	Fat.	Sugar.	Salts.	Other nitrogen-containing and unknown bodies.
Human	0.9	3.52	6.75	0.197	0.6
Cow	3.0	3.55	4.51	0.7	0.3
Goat	2.8	3.40	3.80	0.95	
Mare	1.9	1.00	6.33	0.45	0.5
Ass	1.63	0.93	5.60	0.36	

The portion of milk which is first drawn is more watery than that which is obtained at the end of a nursing.

The number of meals daily during the first few weeks should be seven; later, six or five.

The size of the individual meals, according to Feer, are:

Weeks.	2.	4.	8	12.	16.	20.
Average amounts	90	110	140	150	160	170
Maximal amounts	140	160	215	240	260	270
Capacity, according to Pfaundler	. . .	90	100	110	125	140

The difference between the capacity and the amount of milk ingested is due to the fact that the milk passes into the duodenum during nursing.

During the first two days only a very small amount is drunk. The amount of milk taken daily increases in the first few weeks from 10 gm. to 400 to 500 gm., and then continues to increase.

The amounts drunk by a breast-fed child, according to Bendix and others, are:

At the end of	1st	week of life	250 grams.
At the end of	2d	" "	500 "
At the end of	3d	" "	550 "
At the end of	4th	" "	600 "
At the end of	8th	" "	800 "
At the end of	12th	" "	850 "
At the end of	16th	" "	860 "
At the end of	20th	" "	930 "
At the end of	24th	" "	1000 "

The *amount of food required* by a nursling, as has been recently determined, depends upon the energy which such food can produce (Heubner). The number of calories, per 1 kg. of body weight, which are obtained from the food are indicated as the "energy-quotient" (Heubner). This energy-quotient during the first half year equals 100.

According to Rubner the number of *calories* produced by various forms of nourishment are as follows:

	In 1 liter.
Human milk (depending upon the amount of fat present)	from 614–724 calories.
Cows' milk	from 690–724 "
Two-thirds milk (according to Heubner)	from 480–724 "
One-third milk with sugar	from 340–724 "
Buttermilk (according to de Jager)	from 698–724 "
Liebig extract (according to Keller)	from 808–724 "
Allenbury's milk mixture	from 546–724 "
Asses' milk (from Dresden)	from 502–724 "
Flour soup (5 per cent.—from Rudemann's meal)	from 195–724 "

The following *example* shows the manner of figuring out the amount of food required:

A child weighing 7 kg. requires 700 calories; the amount of human milk required (see table) $= \frac{700}{650}$ gm. (1.07 liters). The amount of cows' milk required $= \frac{700}{690}$ gm. (1.01 liters).

For practical purposes it should be remembered that a healthy child must receive daily during the first three months about one-sixth, and during the second and third months about one-seventh, of its body weight of human milk (Heubner).

In other words, the amount of food ingested daily

during the first week of life should be 10 per cent. of the body weight; in the second to fourth week, 16 per cent.; in the second month, 17 per cent. After this period the percentage is about 1 per cent. less every month (Oppenheimer).

Breast Feeding.—The breasts should be prepared during the pregnancy by washing and massage of the nipples. The child is applied to the breast for the first time on the first or second day. In case it is hungry and the milk has not yet appeared a teaspoonful of camomile tea may be given [warm water answers the same purpose]. If the nipples are hard to grasp a nipple-shield or breast-pump may be employed. At the beginning the child should be put to the breast every two hours, later, every three hours. The child should become accustomed to feed at regular intervals.

It is best to confine the nursing to one breast until satisfied. The rest following nursing should continue for from four to five hours. The beginning of menstruation, or slight disturbances in the health of the child, should not interrupt the nursing. The mother should be given no special diet, but should continue her accustomed food as long as the appetite remains good. She may take as much milk as possible, but without compulsion. Alcohol must be avoided, her occupation regulated, and she must take regular exercise in the open air.

The child should not be weaned until the sixth month and, if possible, not during the hot season. It should be prepared gradually throughout the course of weeks by the administration of artificial meals (milk mixtures, broths, and, under certain circumstances, bouillon).

Indications for Weaning.—Pregnancy, acute febrile diseases of the mother, insufficient milk (the child's weight failing to increase and the occurrence of constipation), unsuitable milk (the presence of colostrum bodies in the permanent milk, fat corpuscles, bacteria), and chronic dyspepsia of the infant. When there is a relative insufficiency of milk the mixed feeding should be continued as long as possible.

When the mother is unable to nurse the child, artificial feeding should be resorted to, and not until this fails should a wet-nurse be obtained. The requirements of a good wet-nurse are: Good health; it is especially important that she should be free from tuberculosis and syphilis, of other acute or chronic diseases, and be capable of producing a large amount of good milk. Her condition may also be judged by the health and body weight of her own child, and by a careful observation of the increase in weight and the amount of milk taken by the child to be nursed. A chemic examination of the milk is of value. Pressure upon the glands should cause the milk to appear in several streams. As regards the quality of milk, the age of the gestation period is of little significance, yet it is advisable for safety's sake not to accept a wet-nurse previous to six weeks after confinement, and for social reasons, not after three months. The offspring of the wet-nurse should not be brought into the house. [There are several reasons why a wet-nurse should be permitted to have her own baby with her:

(1) The moral question of consigning her own baby to an institution, probably to its death. To this there can be only one answer.

(2) The wet-nurse's baby frequently stimulates the breasts and keeps up the supply of milk.

(3) If she does not care for her own baby, it is doubtful if she ought to be trusted with another's baby.

(4) She is more contented and in a better mental state, in consequence her milk is more likely to be normal. She will have no valid excuse to make visits or absent herself from her charge. For this reason she is at home when she is needed, and her food and morals are under control.—Ed.]

ARTIFICIAL FEEDING

Artificial feeding, even under the most favorable circumstances, is not an absolute satisfactory substitute for the mother's breast. For this purpose cows' milk is the

best, partly because the milk of other animals is too expensive and partly because of a difference in constituency.

Cows' milk differs from human milk in the following respects: The presence of dirt and bacteria; it contains three times the quantity of albumin; by the chemic union of the albuminoid bodies; by the comparatively larger amounts of dissolved albumin (relation of casein to albumin in cows' milk = 10 : 1, in human milk = 10 : 12); larger amounts of salts and less sugar; larger curds of casein; greater acidity, on account of which less hydrochloric acid is set free in cows' milk to prevent fermentation. The casein of cows' milk is not more indigestible than that of human milk (Heubner, Bendix).

In preparing an infant's food an attempt is made to compensate for these differences by obtaining milk, if possible, which is clean and free from bacteria, and converting it both chemically and physically to correspond to human milk, and thus obtain perfect equalization.

FREEING THE MILK OF FOREIGN MATERIAL AND BACTERIA

To obtain clean milk: Judicious feeding and care of the animals; exclusion of diseased cows, and cows tested with tuberculin; mixed milk is better than the milk from one cow, because of the greater dilution of the injurious substances. The cows should be milked under clean and sanitary circumstances. The *milk-slime* should be separated by means of a centrifuge or an aseptic filter. The milk is to be rapidly cooled and kept cool until delivered; centrally located model milk establishments for the distribution of milk. When it is impossible to obtain raw milk free of bacteria, the latter should be destroyed either by simply boiling in covered vessels, set aside to cool rapidly and kept cool, or by steam sterilization at home in a *Soxhlet apparatus* (sterilization of from ten to fifteen minutes insures a durability of from two to three days). Another method is to *pasteurize* the milk, that is, heating it to 68° to 70° C. [154.4–158° F.] in an apparatus like

that of Oppenheimer, Kobrack, or by the agitation method more recently described by Gerber.

Aside from the ease of sterilization, the Soxhlet method has the additional advantage of preparing a whole day's supply in individual bottles securely protected and ready for use.

Results of Sterilization.—Sterilization gives the milk an unpleasant taste and disagreeable odor, and alters its composition in all respects, destroying the ferments and immune bodies. It creates a monotonous diet; in artificially fed infants the tendency to anemia and disturbances of metabolism so frequently observed may be traced to it.

EQUALIZING THE CHEMICOPHYSICAL DIFFERENCES ACCORDING TO VARIOUS METHODS

Diminishing the *albumin content* by dilution or partial precipitation. The casein of cows' milk is probably not less digestible than that of human milk, but on account of the increase of albumin, the digestion of the albuminoids is more likely to be interfered with in sucking infants than favored (Heubner).

The *loss of fat and sugar* in the dilution is compensated for by the addition of either sugar of milk or cream (Biedert), or of sugar of milk alone (Heubner), or of nutritive sugar [Nahrzucker] (Soxhlet), until the mixture possesses an equal percentage of ingredients, or (Biedert) an energy potential equal to that of human milk (Heubner).

For practical purposes we proceed as follows: As a diluting fluid a thin decoction of oatmeal or barley is employed, to each 100 grams of which 1 coffeespoonful of sugar of milk or 2 coffeespoonfuls of nutritive sugar (or Liebig's powdered extract) are added. Begin with one-third milk and two-thirds of this solution, and gradually increase to one-half of each. In two or three months a two-thirds mixture is employed. In from six to eight months only milk is employed. The increase in concentration of the food depends upon individual conditions.

Preparation of *Heubner's two-thirds mixture* (two-thirds

milk, one-third of 12 per cent. solution of sugar of milk): 1000 parts contain 18 of albumin, 24 of fat, 70 of sugar, 47 of ash = 640 calories. This represents approximately 1 liter of human milk. There is used of this mixture at first $\frac{3}{4}$ liter, then 1 liter, and finally 1200 gm. in twenty-four hours.

These figures are only approximately correct, it being impossible to present a scheme of feeding which would serve in all cases.

The Administration of Milk.—At the beginning it should be given after two- and then after three-hour intervals. The number and size of each feeding are regulated as in the case of breast-fed children; the amount is, as a rule, somewhat larger.

Chief Danger: Overfeeding.—The rubber nipple must be daily boiled and preserved in a solution of boric acid and dried. The milk-bottles must possess smooth walls. The temperature is to be personally observed.

Feeding with gruel after three or four months once daily. In the course of time fruit juice may be given. Other highly recommended preparations of milk are mainly used when the above mixtures of milk and sugar cannot be digested. The most important of these preparations are:[1]

Group I.—Milk mixtures containing a diminished amount of albumin and an increased amount of fat.

Biedert's cream mixture. Receipt: Diminishing amount of albumin by diluting with water; gradual increase of same by addition of milk and substituting fat and sugar by cream and sugar of milk.

Natural cream mixture No. I. consists of $\frac{1}{8}$ liter of cream, $\frac{3}{8}$ liter of water, and 18 gm. of sugar of milk. No. II. consists of the same and $\frac{1}{16}$ liter of milk; the addition of milk increases then until $\frac{3}{4}$ liter is reached, after which water and sugar of milk are decreased. The preparation of cream "Ramogen" is more convenient; it can be regulated by the addition of water and increasing the amount of milk.

[1] As grouped by Bendix.

Drenckhan's milk.

Gärtner's milk.

Lehmann's vegetable milk (formula for cream mixtures, combined with vegetable albumin and fat, employed as an addition to milk).

Condensed (Swiss) milk, with high percentage of cane-sugar.

Monti's Vienna infant's milk (milk thinned with whey).

Group II.—Diluted milk mixtures containing an increased amount of fat, and in which the albuminoids are predigested and held more or less in solution.

Backhaus' milk, No. I.: Addition of cream from which a portion of the casein has been dissolved by trypsin. No. II.: Milk-fat. No. III.: Pure milk.

Voltmer's mother's milk (conversion of the casein of cows' milk into peptone by the addition of the pancreatic ferment; otherwise its constituents are analogous to that of human milk).

Loefflund's peptonized milk.

von Dungern's milk rennet (addition of a knife-pointful of "pegnin" to 200 gm. undiluted milk).

Group III.—Diminution of the albumin content by dilution, increase of fat, substitution for the deficit of albumin by a soluble albuminate or peptone.

Rieth's albumose milk (substitution of the casein by a non-coagulable form of albumose, which is derived from egg-albumin by means of heat). Addition of cream and sugar. Hartmann's somatose milk is similar in character.

Hempel-Lehmann's milk (dilution of cows' milk until it contains 75 per cent. of casein. The addition of a yelk—phosphorus and iron—and the white of an egg, enriched with fat and sugar of milk).

Group IV.—Mixtures poor in fat, but rich in sugar (especially malt sugar).

Liebig's soup (conversion of wheat flour into maltose); a more recent form of this preparation is Keller's malt soup (50 gm. of wheat flour and 650 water, 100 gm. of Loefflund's malt-soup extract and 350 milk); both mixtures being cooked together.

Allenbury's infant's food No. III. (malted food).

Liebe's neutral food (Dresden).

Soxhlet's nutritive sugar (a wheat flour converted into dextrin and maltose), to which has been added a certain amount of acids as well as digestive salts and table salt.

Brunnengräber's (Rostock) powdered malt.

The useful forms of flour for children are classified as follows :[1]

Simple prepared flour, over 5 per cent. fat : Knorr's and Weibezahn's prepared oatmeal.

Infant foods (prepared from milk and diastasized flour or malt) containing a high percentage of fat : Faust and Schuster (4.5 per cent. fat), Nestlé (5.1), Rademann (6), and Muffler (6.4).

Infant foods containing a low percentage of fat : Soxhlet's Liebigsoup ; Mellin (0.3 per cent.), Kufeke (0.8), Opel's nutritive zwieback (1.3), Ridge (1.3), Neave (1.7).

Foods containing large amounts of easily soluble starches are : Mellin, Theinhard, Nestlé ; containing large amounts of starches soluble with difficulty : Neave, Ridge, Weibezahn, Rademann, and Kufeke.

In two and a half years the following articles of diet may be gradually given : Veal broth and also well-cooked veal, apple sauce, various fruit juices, biscuits, purée of carrots, cauliflower, spinach, fresh strawberries, finely sliced apples. The chief form of nutrition from the second year on must still be milk, supplemented by a mixed diet of meat, vegetables, potato broth, fruit, zwieback, butter rolls, easily digested farinaceous foods, etc. Regular meals of milk should be continued as long as possible (the milk-bottle must not be withdrawn too soon).

EXAMINATION AND HISTORY

The examination of children follows the same rules as in the case of adults. As in each individual case different characteristics are met with, it is advisable to follow a certain routine in order to avoid errors.

[1] Author's division.

ANAMNESIS

The following is in a general way a reproduction of the plan outlined for the Children's Hospital of Munich:

The number of the child? Diseases and deaths of parents, brothers, and sisters? Previous premature births? Form of feeding to date: Breast? cows' milk? broth? beer? meat diet? Sleep. Digestion. Domestic conditions, sanitary state of the home, care and attention of the child. Hardening? Source of the milk, handling, and method of administration (long tube, nipple, or graduated bottle).

Previous diseases of the child, especially disturbances of digestion, convulsions, diseases of the lungs, eruptions, enlargement of the glands, ocular and aural discharges, infectious diseases, course and treatment of such diseases (exact data).

Present Illness.—Beginning, course, and treatment up to date (always give data).

In case of special diseases greater details are required. Thus one must consider:

In Nervous Diseases.—The variety and duration of labor when child was born; past trauma? Whether there was any nervous disease, suicide, alcoholism, etc., amongst the relatives. Physical and mental development and character of the child; the form and duration of cold-water treatments, etc., if such were undertaken at any time in the past. The presence of adenoids.

In Rachitis.—Form of nourishment, as above. The course of dentition. Learning to stand and walk. Sweats in occiput; outcry upon being lifted. Restlessness? Diarrhea? Age of parents; hereditary syphilis.

In Hereditary Syphilis.—Former premature births, stillbirths, period and nature of same. Healthy children during intervals? From one or more fathers? Possible syphilis in parents. *Concerning the child itself:* Full-time baby? Snuffles? Eruptions?

In Diseases of Metabolism.—Character of the infant's feeding—if milk, how long sterilized and until what age

was it administered? (Anemia, Barlow's disease.) Hardening with cold water? (Anemia.) In dysthyroidal conditions, myxedema, infantilism, cretinism, etc., inquire into the existence of the following conditions in ancestors, brothers, or sisters: Goiter, idiocy, obesity, gout, gallstones, excessive genital hemorrhages, sensitiveness to cold, cold hands and feet.

In Diseases of the Respiratory Tract.—Colds, exposure to wet, former diseases of respiratory organs, hardening procedures, measles, whooping-cough as an excitant of latent tuberculosis. Possibility of aspirated foreign bodies? Chronic pulmonary diseases in relatives.

In Diseases of Digestive Tract.—In sucklings determine accurately the former modes of feeding—broths, milk—source, especially in bottle-fed children, and preparation and method of administration of the milk. Infant's milk? Feeding of the cows. Mixed milk, or milk from one cow, possibly from one infected with bovine tuberculosis. At what time delivered at the house (possibility of standing in the sun on delivery wagon for hours)? Is the milk simply boiled, sterilized, pasteurized, or administered raw? Length of sterilization? Method of preservation, whether kept near sick room? Mode of mixing milk and character of ingredients. Total amount of liquids taken daily? Amount of pure milk? Size of individual meals; intervals between drinking. Form of bottle, of nipple; size of the holes of the latter. The care of the whole feeding apparatus.

THE PROPER METHOD OF EXAMINING A CHILD

In recording a certain case the following plan should be followed: I. Temperature, pulse, respiration. II. Nourishment and development. III. Skin. IV. Glands. V. Apparatus of locomotion. VI. Nervous system and organs of special sense (sensorium, reflexes). VII. Pharynx, nose. VIII. Organs of circulation. IX. Respiratory organs. X. Abdominal organs (stomach, liver, spleen, intestines, and genitalia). XI. Urine. XII. Stools.

At first, before disturbing the child, a general inspection is made; if the infant is awake delay the examination until it has become accustomed to the physician's presence

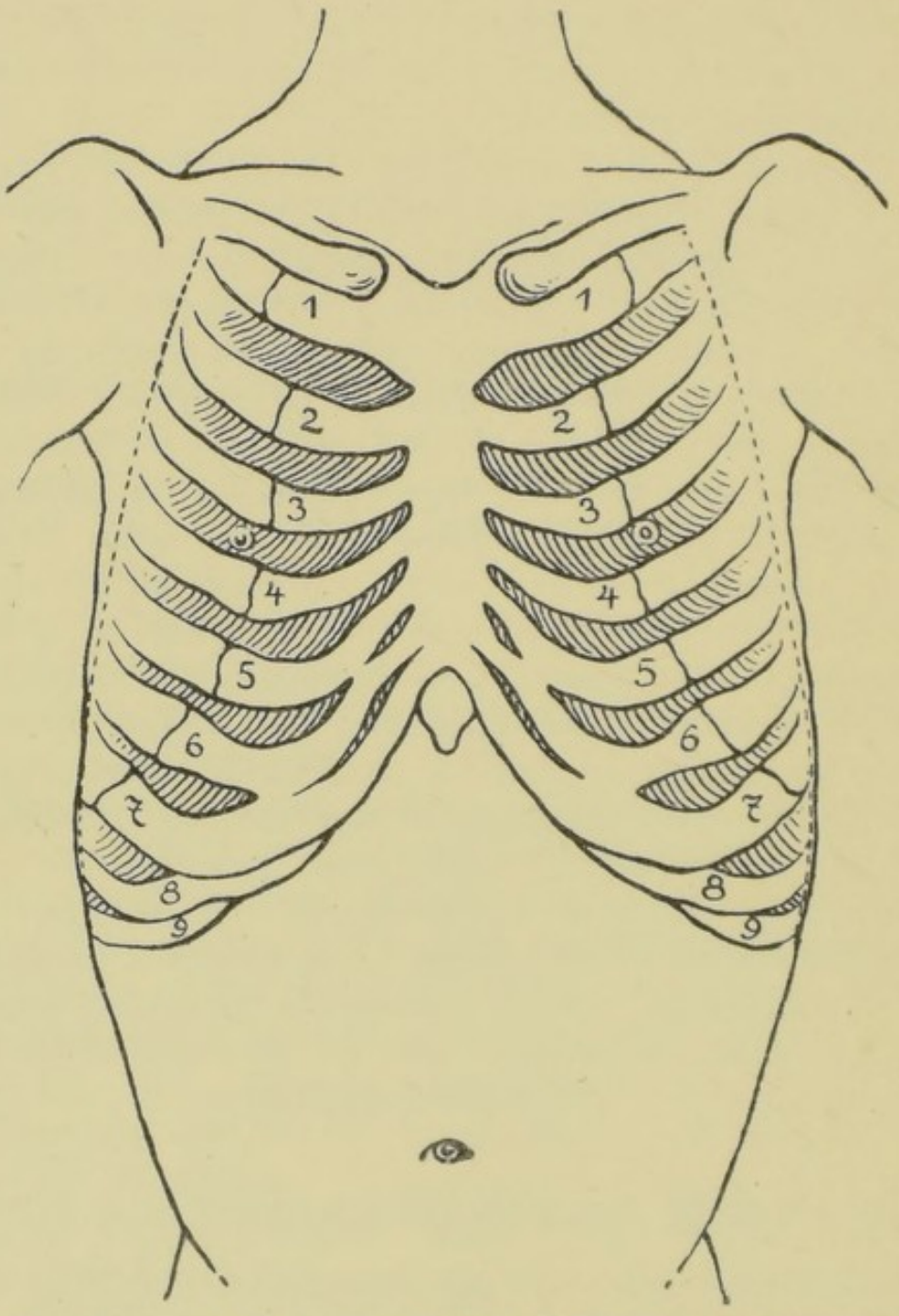


FIG. 9.—Diagram for recording the findings in the examination of children up to four years of age. (According to the exact cadaver measurements of Trumpp.)

and employ the time asking the most important questions, and in observing the child with as little annoyance as possible.

Note: A ceaseless drawing up of the legs with loud cries and painful distortion of the face in sleep indicates pain in the abdomen; rubbing the head on the pillow points toward craniotabes or otitis media; in the latter case, there is usually high fever. The failure of voluntary motion when the child is awake is a sign of weakness, stupor (opiates), or imbecility. Characteristic appearance of the face in meningitis (sickly, painful, squint); in whooping-cough (puffiness in the neighborhood of the eyes, protrusion of eyeballs); in vegetative adenoids (somewhat stupid, open mouth, obliteration of nasolabial folds).

After the pulse and respiration have been controlled, inspection, which plays a very important rôle in children, is undertaken. This is followed by thermometry, palpation, auscultation, percussion, mensuration, and examination of the excretions. The respiration can only be studied when the child is asleep or absolutely quiet. We note its frequency, depth, regularity, and character, which is snorting in case of vegetative adenoids, impeded in tonsillitis, gurgling in retropharyngeal abscess, contraction of the jugulars in stenosis of the upper, and contraction of the costal arch in stenosis of the lower, air-passages. The pulse is likewise best observed when the child is asleep. It is to be noted that a considerable increase in the rate may follow the slightest cause, even in healthy subjects. The observation includes mainly the frequency, regularity, tension, and size. The pulse is slower than normal at the commencement of meningitis, during the convalescence from infectious diseases, and in weakening conditions. The pulse is accelerated in fever, in excitement, and in the terminal stage of meningitis. The pulse is irregular (also unequal) in certain intestinal diseases—influenza, diphtheria, myocarditis, and in meningitis, even at the beginning.

While the child is still in bed the temperature is taken. This is always done, without exception, per rectum, and is determined by passing, high up into the rectum, the thermometer which has been cleansed with alcohol and

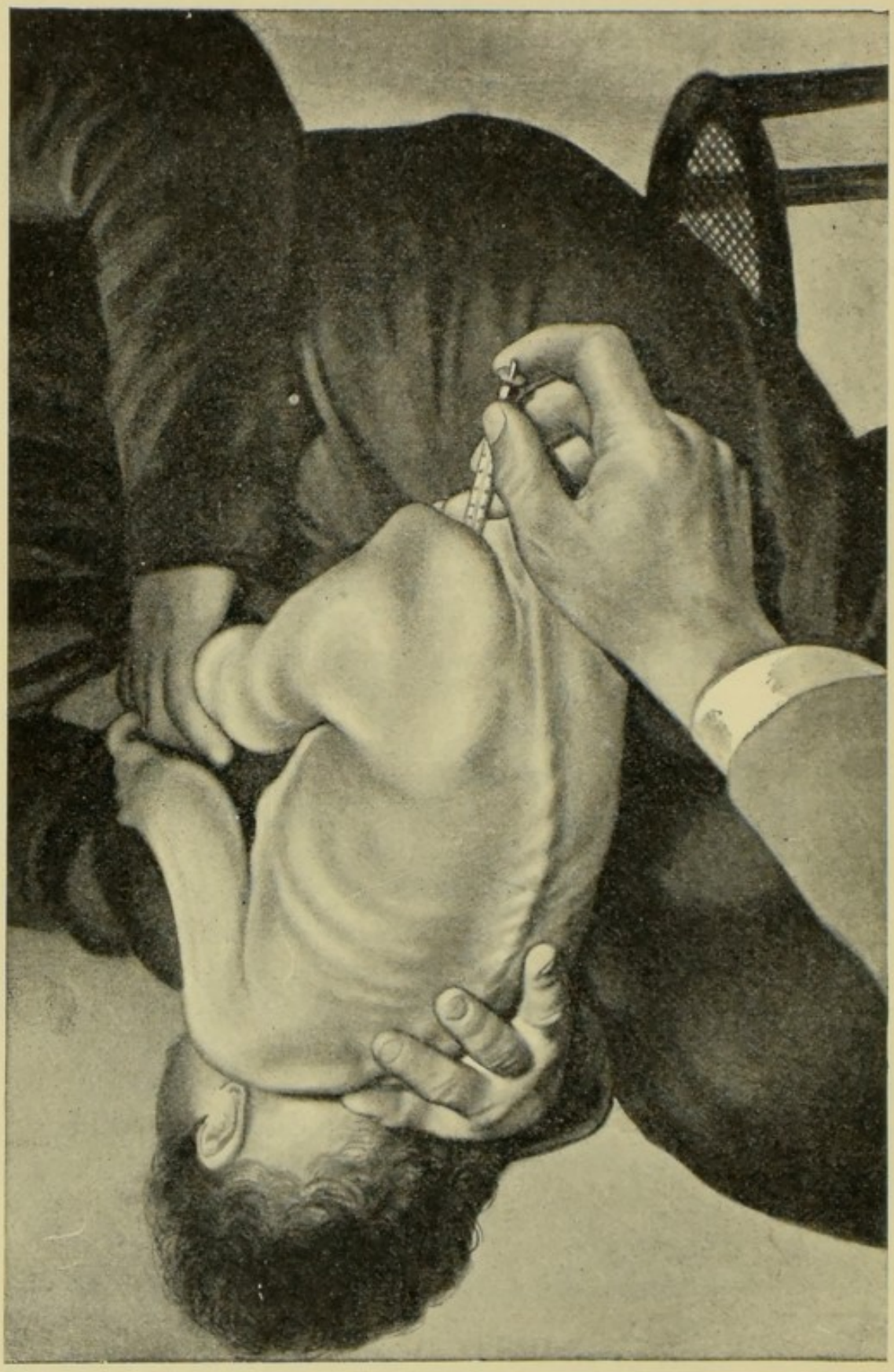


FIG. 10.—Introduction of a thermometer in the rectum of an infant.

lubricated with fat, soap, or water. This act may be accomplished with greater ease by drawing the legs of the

child tightly against the abdomen. So-called "minute" or "half-minute" thermometers are to be employed, and the first result controlled by a second test.

For further examination it is absolutely necessary to remove the child, undressed, from the bed. The infant is held either on the lap of its mother or on an upholstered table with its face toward the light.

INSPECTION

We next note the general appearance and state of nutrition, expression of the face, posture, and abnormal movements. In inspecting the skin observe the peculiar cyanotic pallor of pneumonia; the pale to dirty yellow, somewhat shiny color in hereditary syphilis; the sickly and dry condition in diseases of the thyroid gland; the cyanosis of laryngeal stenosis, miliary tuberculosis, and heart failure; the edema in nephritis; minute extravasations of blood as unfavorable prognostic signs in certain diseases, especially in those of the intestines, in diphtheria. Various diseased states must be distinguished from insect bites, which are red with central points, sudamina, and the erythema which follows applications.

The skin and the pad of fat in the region of the anus point to the general state of nutrition; intertrigo in severe diarrhea; rhagades in chronic constipation and in syphilis.

Papules in eczema and syphilis. Also observe the general form of the body and of its different parts (deviation of the vertebræ, deformities, contractures), and also the posture and gait.

Inspection of the oral cavity is not to be neglected in any case, but should be delayed until the end of the examination. It is to be performed with the infant in the lap or arms of the mother. The physician stands either in front of or behind the child and presses the tongue with a spatula or spoon until a swallowing movement occurs, which brings the posterior pharynx into view. If it is impossible to open the mouth because of the

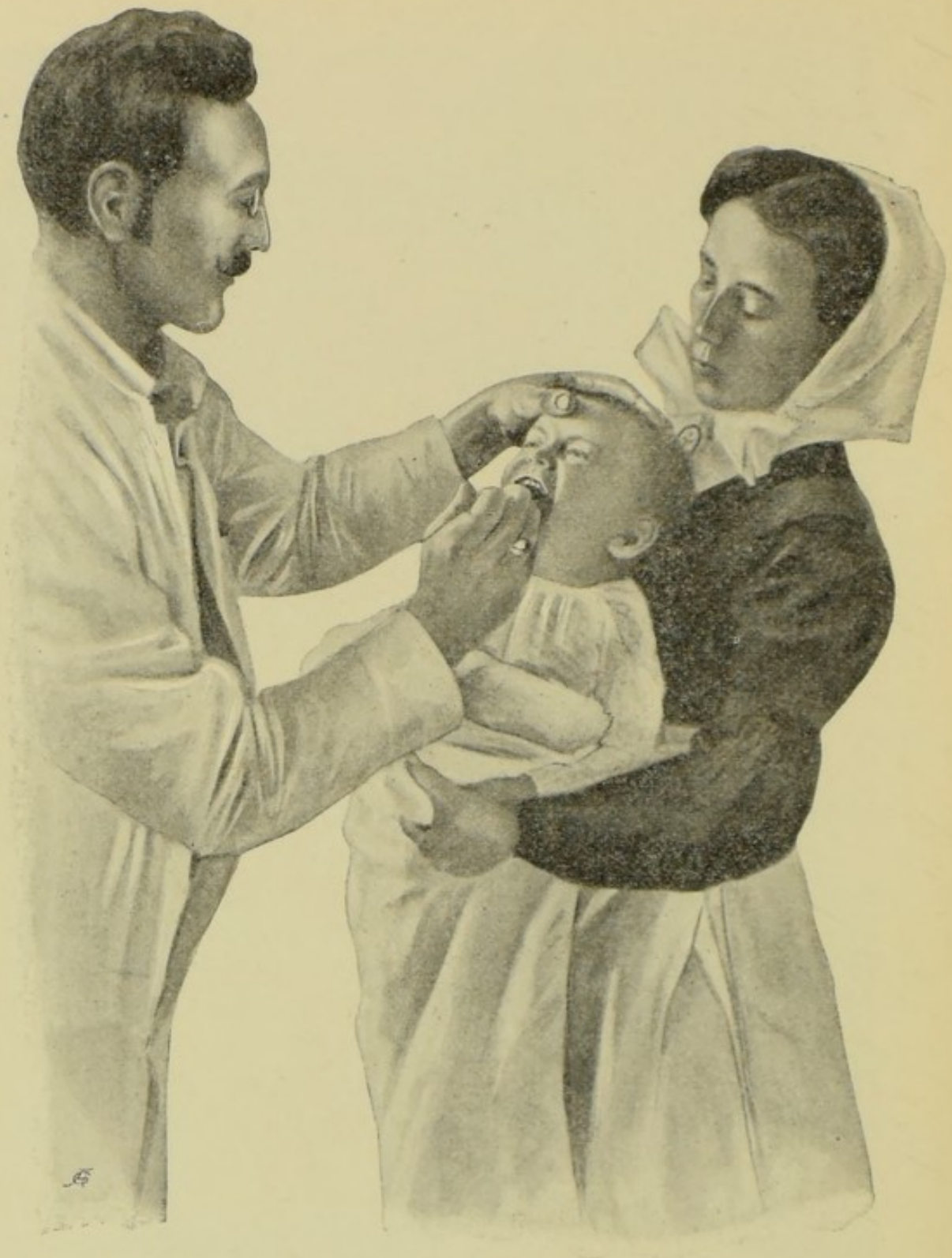


FIG. 11.—Inspection of the oral cavity of a small child. The hands are firmly fixed. The physician's left hand holds the head and guides it toward the light. The inspection may also be performed as in Fig. 12, the nurse holding the infant on her lap.

strong resistance offered by the child, we should patiently try to enter the mouth back of the molar teeth. Holding the nose shut in order to force the child to open its



FIG. 12.—Inspection of the oral cavity with the physician standing back of the child. This position is a better protection against the cough, but requires more practise to see.

mouth is of no avail. It will only serve to excite the child, who can finally, even without opening the mouth, breathe through the tooth spaces. During the short time which is at our disposal, we note the color and any eruption that may be present on the hard and soft palates, the condition of the tonsils and posterior wall of the pharynx, and any deposit which may be on these parts.

The presence of a tonsillar or retropharyngeal abscess must not be overlooked under any circumstances. If it is suspected, the oral cavity must be palpated. Inspection of the teeth with reference to their number, development, form, and position should take place either before or after viewing the pharynx.

PALPATION

By passing the flat hand over the surface of the body a superficial idea may be obtained of its temperature (but only the thermometer should be trusted). The skin is tested as to its moisture or dryness. It is moist when a fever is on the decline, dry in profuse watery diarrhea, in disease of the thyroid gland, and in increasing fever. If on inspection any discoloration or change in form is noticed, it is further examined by palpation. Edema and papular exanthems can only be determined positively in this manner; erythema and roseola can only be distinguished from hemorrhages by their disappearance upon pressure. Then follows a systematic palpation of the whole body from the head downward. From the large fontanels, where the size of the opening, the degree of tension, and the character of the borders are noted, the hand passes to the occiput. The latter is grasped in both hands and with pressure exerted by the fingers we detect softened areas (craniotabes). The fingers passing over the two lateral fontanels exert pressure upon the tragus and the posterior auricular region, to exclude severe affections of the ear (otitis media, mastoiditis). Next the palpating hand examines the lower jaw, the posterior and lateral cervical region, as well as the two



FIG. 13.—Palpation of the spleen with the left hand from above. By means of light pressure with the ball of the hand upon the thorax, the spleen is forced further downward. (Observe the position and fixation of the child.)

FIG. 14.—Palpation of the liver from above. The organ may be pressed downward with light pressure exerted by the ball of the hand. Palpation may also be performed from below.

supraclavicular fossæ and the lymph-nodes which are found in that neighborhood. Note: Acute enlargement of the glands in diphtheria, scarlet fever, stomatitis, and abscesses; chronic enlargement in scrofula, eczema of the head, caries of the teeth. Enlarged supraclavicular nodes are of importance in latent tuberculosis.

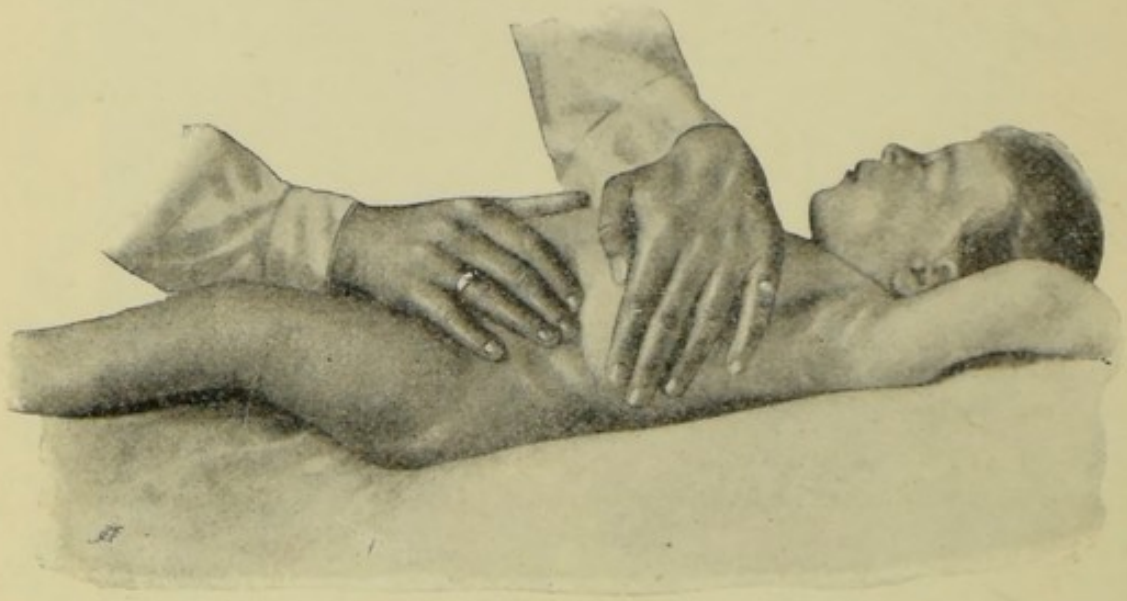


FIG. 15.—Bimanual palpation of the spleen from below upward. Counterpressure is exerted upon the spleen with the left hand.

In the thorax the borders of the cartilages of the ribs are felt and physiologic enlargement distinguished from the rachitic swelling. In palpating the abdomen the child should lie upon its back. The hand, which should be relaxed and held as flat as possible, is slowly and gently pushed inward. The abdomen is retracted in atrophic and cachectic conditions and in meningitis; it is enlarged in rachitis and in many diseases of the intestines. Tenderness exists in the region of the colon and in inflammatory processes of the lower section of the intestines. The ileocecal area should always be examined for tenderness and resistance. Ileocecal gurgling is a frequent phenomenon in children. To detect free liquids in the abdominal

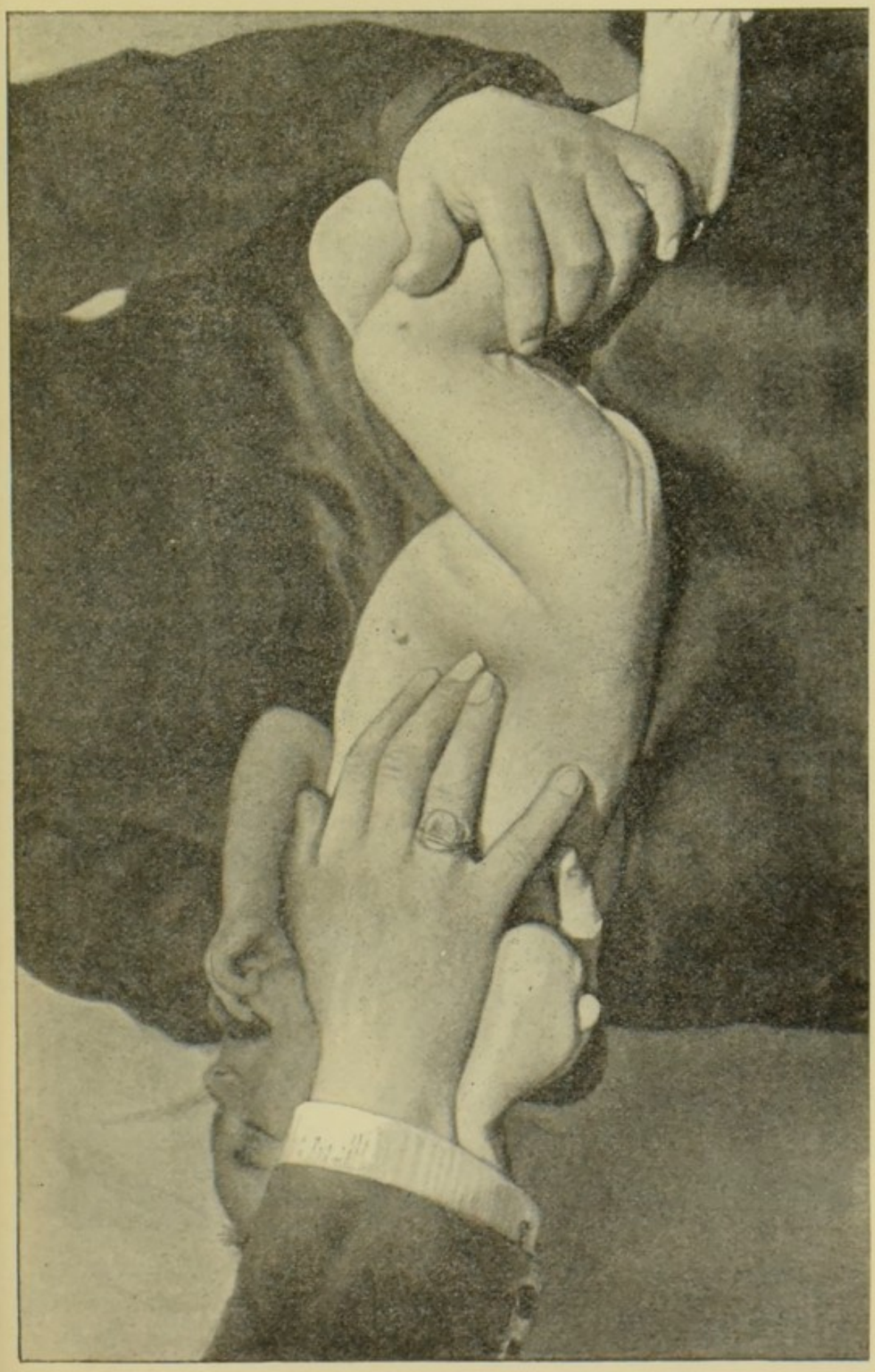


FIG. 14.

cavity gently tap with the right middle finger and receive the wave with the point of the left finger.

The spleen is palpated in two ways (compare Figs. 13 and 15). Be careful not to mistake the lower ribs for that organ. The same conditions hold true for palpation of the liver. Finally, the extremities are palpated for swellings, bony exostoses, tenderness, abnormal or diminished motility, paralyses, spasms, etc.

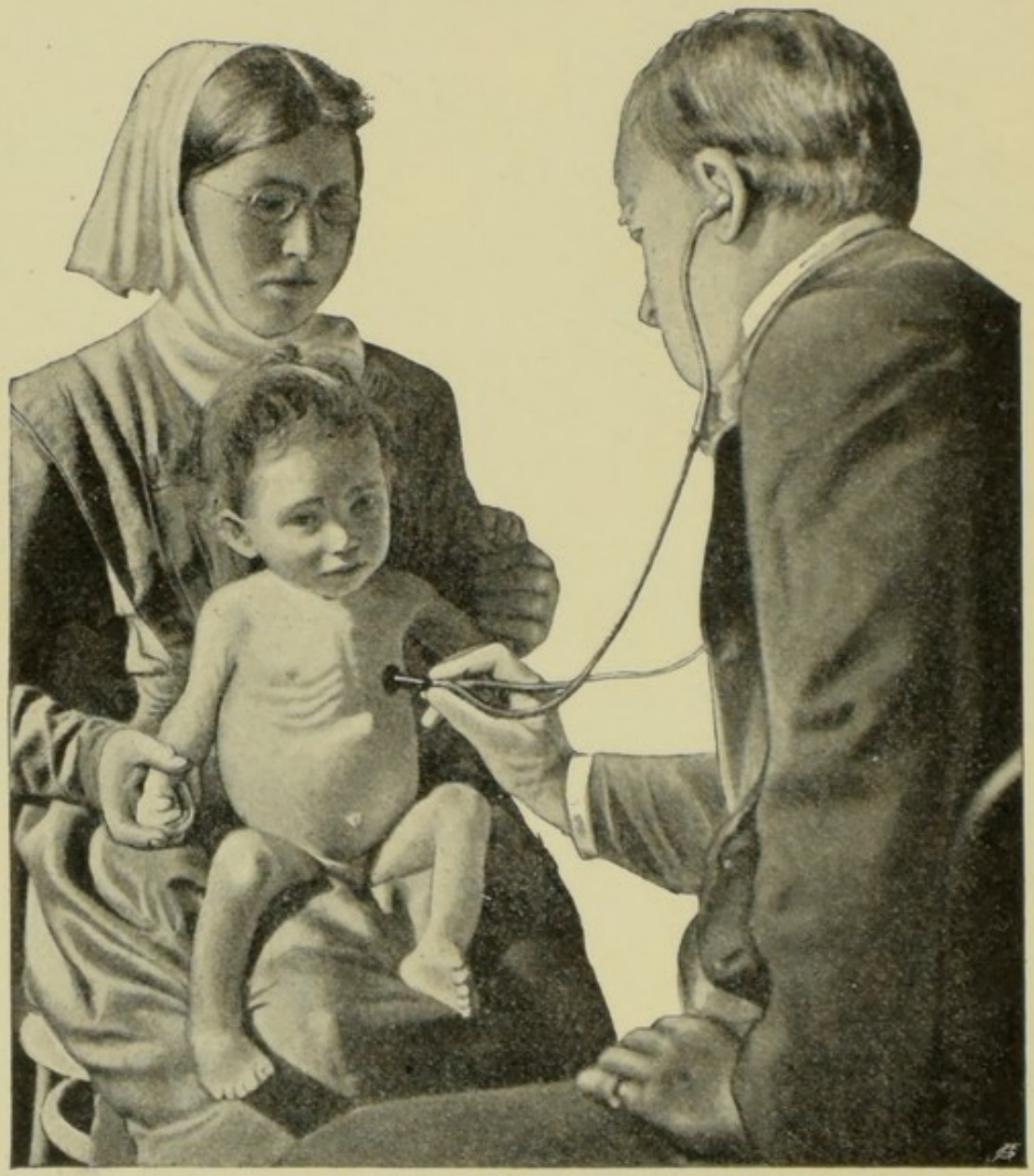


FIG. 16.—Auscultation with the double stethoscope. Advantages: The area to be auscultated is visible, the child is less disturbed, and there is greater magnification of the sounds. Disadvantages: Neighboring sounds are more distinctly heard, and it is necessary to renew the tubes in the course of time.

AUSCULTATION

It is desirable that the child be kept as quiet in this procedure as in percussion, but it is not an absolute necessity. The crying is of use in determining the vocal resonance and fremitus. The respiratory murmur itself can in that case only be heard during the short inspiration. Whether auscultation precedes percussion or vice versa, depends upon which is the most unpleasant to the child. (It is wise to proceed gradually with the least pleasant examinations.) Auscultation of the lungs may be performed in various ways: At first with the bare ear—for thus the sounds are purer and clearer—and then, to control the results of that method, by means of a stethoscope. The binaural stethoscopes are almost exclusively used in this country, and are to be recommended (Fig. 16). It is to be noted that normal bronchial breathing is heard on either side of the spinal column, and that frequently crepitant râles are heard at the beginning of the examination on account of the forcing of air into parts which were previously atelectatic.

Auscultation of the heart should always be performed with the stethoscope, for it cannot very well be outlined or localized in any other way. (For the normal relationship of a child's heart, see Diseases of the Heart.)

PERCUSSION

Either finger or hammer-pleximeter percussion. The latter is to be done only with a light Curschmann's hammer. Various forms of finger percussion are distinguished, all of which are best employed one after the other.

Direct Percussion of Bones.—The point of the middle finger percusses anteriorly the clavicles at symmetric points, and posteriorly the spines of the scapulæ.

Palpatory Percussion.—The four fingers of the right or left hand percuss directly the posterior wall of the thorax in symmetric areas. These two methods of percussion are useful in making a rapid examination.

The Usual Method of Percussion, Finger on Finger.—This is not performed as in adults—with relaxed wrist-

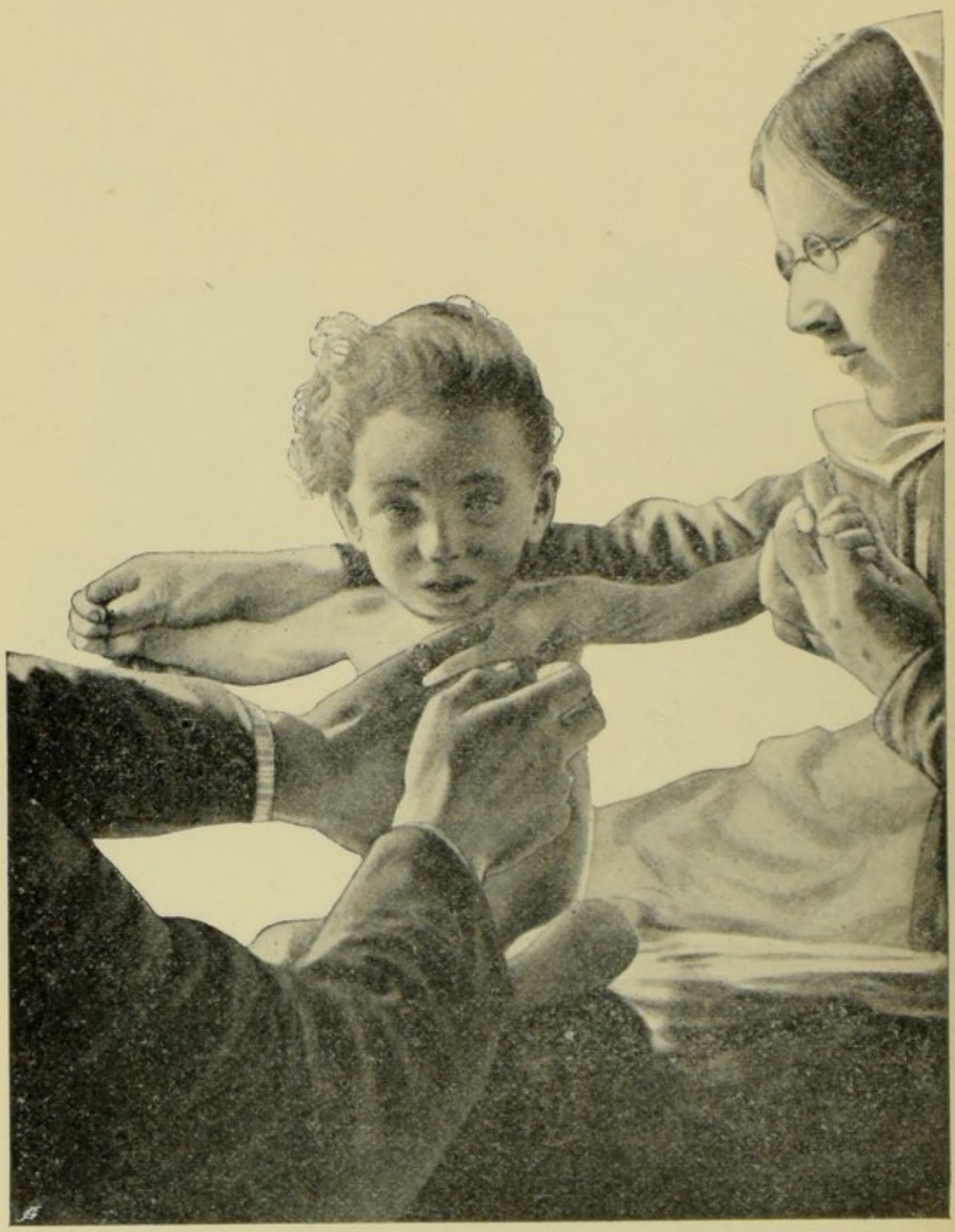


FIG. 17.—Manner of holding and fixing a child in anterior percussion. The same holds true for percussion of the back. The body should be in as symmetric a posture as possible.

joint and with an elastic and hammering movement—but with light pressure of the percussing fingers and, as in pal-

pation, with the middle finger of the left hand applied closely and as lightly as possible, palpating at the same

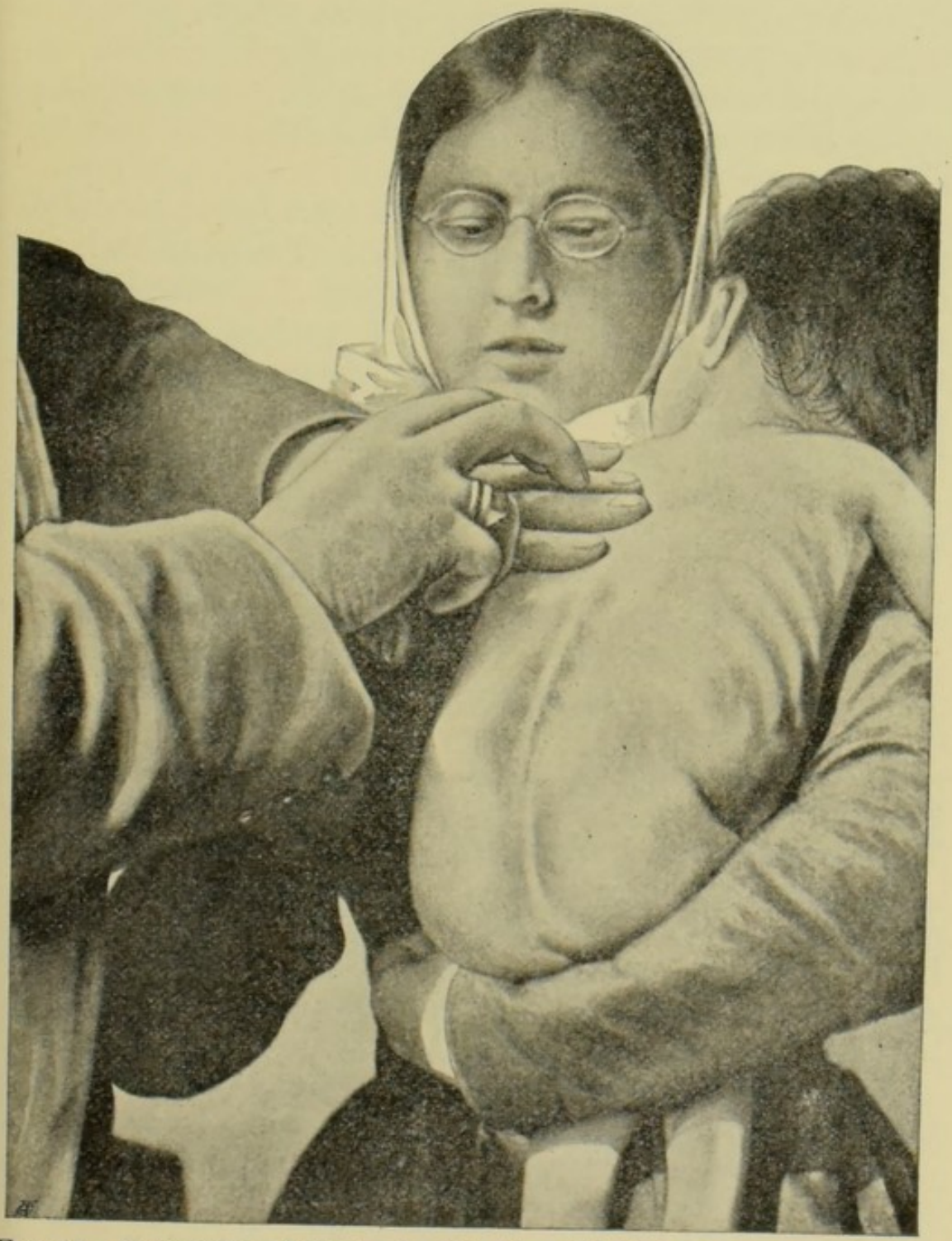


FIG. 18.—Percussion of the back while the child is held in the arms of the nurse. Position as symmetric as possible.

time. Heavy blows include too large portions of the body in the resonance and must be absolutely avoided.

For percussion the child's body must be held as symmetrically as possible, since slight asymmetries of posture cause changes in the sound elicited. The finger which acts as the plexímeter must be always placed on symmetric parts. Since in children the sounds are decidedly influenced by the respiration, it is impossible to always percuss both sides during at least one whole respiratory period.

It should be borne in mind that the lower border of the right lung is higher because of the liver, which is not rarely mistaken for pulmonary dulness. On the left side remember the close proximity of gastric tympany to the edge of the lungs. Examination of the axillary regions is of the greatest importance; bronchopneumonic foci are frequently detected.

Finger-nail upon finger-nail for very light percussion, especially for the spleen and thymus.

Auscultatory percussion by the simultaneous application of the stethoscope or phonendoscope.

MENSURATION

For weighing the child, either a decimal or one of the so-called "infant's scales," fitted with a bowl, is employed. The spring scales, to which the child is hung in a bag, are expensive and unreliable. The spring kitchen scales are useless. Infants should be weighed once every week, larger children, every month. For the physician the scales are indispensable in estimating deviations from normal development.

Linear Measurement.—Infants are held stretched on a table in the dorsal position and measured from heel to crown. Large children are measured standing against a wall or with a vertical measuring staff, which is supplied with a movable transverse arm. The linear measurement is also of value in determining normal and abnormal development, especially in rachitis, hypothyroidism, hereditary syphilis, etc.

THE SECRETIONS AND EXCRETIONS

The secretions from the *conjunctivæ* are examined for diphtheria bacillus and gonococci; the secretion from the *nose*, for influenza or diphtheria bacilli. With a platinum wire, which has been previously brought to a red heat, minute drops are secured from the secretion and rubbed upon the cover-glass; the *sputum* (removed with a cotton swab or aspirated) is examined for its macroscopic characteristics, and microscopically for elastic fibers, influenza, diphtheria, and tubercle bacilli. Deposits in the *mouth* or *pharynx* are removed in minute particles, either with a platinum rod which has been passed through a flame, with sterile forceps, or with a small cotton tampon.

For these as well as for the succeeding examinations proceed as follows: At first examine the unstained preparation mixed with a drop of water and then dry and stain it.

The **urine** is examined for its amount, specific gravity, color, reaction, cloudiness (bacteriuria, cystitis, phosphaturia), albumin, sugar, blood, biliary coloring-matter, indican, and the diazo-reaction. It is obtained from infants either with specially constructed vessels[1] or by means of catheterization (especial care is required in the case of boys). It may be obtained from a sleeping male child by simply catching it in an ordinary reagent glass. For determining the exact quantity of urine excreted the apparatus of Bendix and others may be employed.

The **feces** are examined macroscopically for the amount, color, consistence, odor, reaction, abnormal ingredients, mucus, serum, pus, blood, and remnants of food; microscopically for bacteria, pus-corpuscles, fat, starch (colored blue with Lugol's solution), fungi, tissue cells, amebæ, parasites and their ova.

The **cerebrospinal fluid** is examined as to color, reaction, specific gravity, amount of albumin, sugar, bacteria, and pus-corpuscles. It is obtained by means of Quincke's lumbar puncture. The child lies upon its side or

[1] Compare Diseases of the Kidneys.

assumes the sitting posture; the spinal column is curved forward as much as possible; a sterile needle about 7 cm. [2.8 in.] long is introduced with the point turned slightly upward in the middle line between the 4th and 5th or the 3d and 4th lumbar vertebræ to a depth of from 2 to 4 cm. [.8–1.6 in.], until the point of the needle is freely

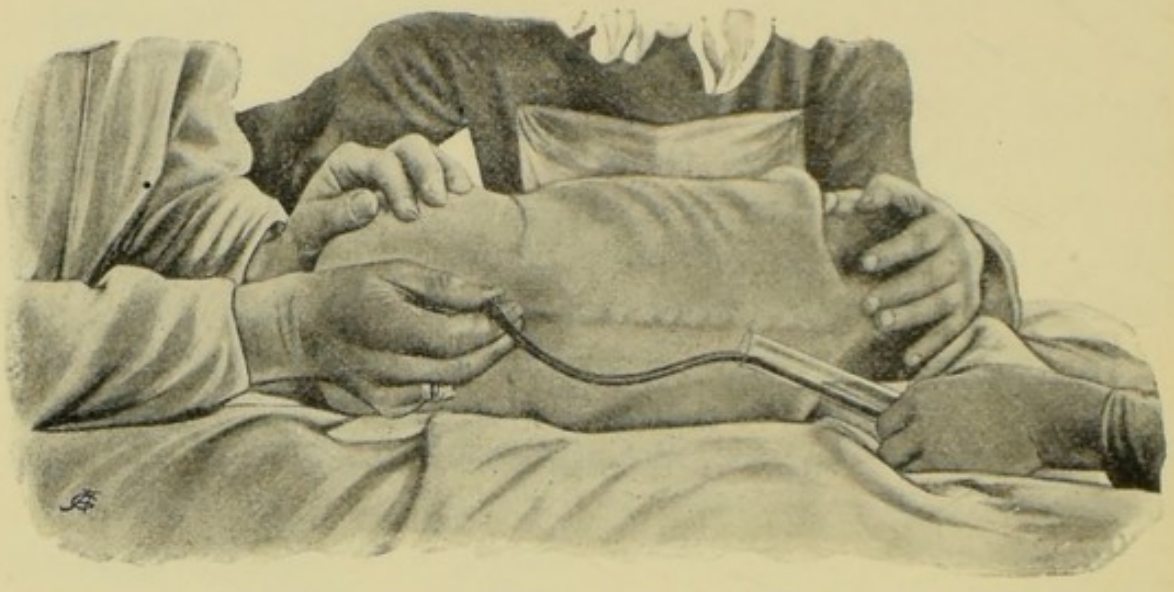


FIG. 19.—Quincke's lumbar puncture. A line drawn from one iliac crest to the other will cross the spine between the 4th and 5th lumbar vertebræ. The puncture may be performed while the child is in the sitting posture.

movable. The pressure of the spinal fluid is measured by connecting the needle by means of a thin rubber tube to a manometer. The normal pressure equals that of from 40 to 130 mm. [1.6–5.2 in.] of water. The operation is free from danger if it is performed in an antiseptic manner.

GENERAL MANAGEMENT OF DISEASE IN CHILDREN

DIETETIC TREATMENT

The most important factor is careful attention to the diet and hygiene. Breast-fed children when sick should continue to receive nourishment from the breast if practicable. Hand-fed children should in acute cases have their diet reduced. In disturbances of digestion

some bland fluid should be given which practically requires no digestion, such as water, dilute tea, or a very thin cereal water; albumin-water (the white of one egg beaten up and stirred in $\frac{1}{4}$ liter of cool water, then strained and sugar added); watery flour broths; 5 to 10 per cent. solutions of nutritive sugar.

Drinks for sick children are cold water, with or without fruit juices, toast-water (toasted wheat bread over which hot water is poured, with the addition of sugar and, if desired, lemon-juice), sugar-water, almond-water, and cold teas.

As a non-irritating diet are recommended: Milk diluted or with the addition of other elements, flour broth, infant's food soups. For larger children give vegetable and potato broths, jellies, fresh minced meat, buttermilk, zwieback, soft eggs, light pastry, fruit, gelatin, and gruels.

As a diet capable of giving strength in chronic sickness give foods rich in fat and sugar, cream, cereals, minced chicken, squabs, veal, ham, sausage, calves' brains, broths, beef-tea, eggs, chocolate, extract of malt, cocoa, and cold oatmeal gruel. In case of weakness give compressed meat juice, meat jellies, beef-tea, strong bouillon, tea, champagne, wine, and port wine.

To stimulate the appetite give undiluted Valentine's meat juice, meat gravies, caviar, and sardines. Of medicines give wine of cinchona, the compound tincture of cinchona, and ichthalbin.

The remaining hygienic directions, which in general are like those observed in adult life, are best given in greater detail, for especial attention should be paid to cleanliness of the mouth, eyes, nose, and skin; preventing wetting of the bed; the clothing; the temperature (15° to 18° C. [59°–64.5° F.]).

HYDROTHERAPY

Sick children are treated with hydrotherapeutic procedures to meet the following indications: To reduce the

fever, to increase the conductivity of the skin, to regulate the circulation, and to increase absorption. Water is employed as a counter-irritant, especially for the nervous system, respiration, and pulse; and also as a sedative.

The following baths are recommended:

Hot baths (37° to 40° C. [98.6°–104° F.]) as a prelude to sweating processes, to increase the temperature of the body (as an analeptic in diseases of the intestines), and to relieve the action of the heart.

Warm baths (33° to 35° C. [91.4°–95° F.]) for cleanliness; at the beginning of febrile diseases; for a symmetric distribution of heat, and as a sedative.

Cool baths (31° to 27° C. [87.8°–80.6° F.]) to diminish fevers; to stimulate and deepen respiration; in nervous diseases, especially with simultaneous vigorous rubbing.

The most useful additions to baths are: Aromatic solutions of camomile, fennel (1 to 2 handfuls in a linen sac over which hot water is poured); wheat-bran, oak-bark as an astringent (3 to 5 handfuls boiled in a sac); salt or brine (cooking salt or sea salt, 200 gm. to a pail of water); mustard as a strong stimulant (5 teaspoonfuls of black mustard in a linen bag over which boiling water is poured, and allowed to stand for several minutes); sublimate (0.5 to 1.0 gm. per bath); peat soil or peat salt (15 kg. of the former and 30 to 40 gm. of the latter); sulphur (15 to 25 gm. sulphuret of potash dissolved with hot water in a bag).

Applications.—Cooling applications made of linen cloth and covered by a larger woolen cloth, without interposing an impermeable layer. These are applied most frequently to the chest and trunk, more rarely to the whole body, for the purpose of diminishing the temperature, as a sedative and to increase conduction; care must be taken not to let them remain in place longer than from one-half to two hours. A satisfactory antipyretic action is obtained at the beginning by changing the application every quarter hour and, later, every half hour.

Hydropathic applications are made with linen, water-tight material, or woolen cloth. The action is a sedative to pain and resorbent; they may remain in place from

three to ten hours. The water used in all of these applications has a temperature of from 16° to 20° R. [70°–77° F.]—"room temperature."

Hot Stupes.—Linen cloths boiled in water, removed with spoons, wrung out in a second linen cloth, and then rapidly applied, covered with a woolen cloth and left in place from a quarter of an hour to an hour. These are employed in sepsis and diseases of the heart and kidneys.

Mustard Poultices.—One liter of boiling water poured on ½ pound of powdered mustard, stirred until the mustard odor arises, soak a linen cloth in the mustard-water which has been poured off the above mixture, and apply it to the body, allowing it to remain in place one-half hour. Follow it by washing with cool water, and later by applying cool applications. A vigorous counter-irritant in bronchopneumonia.

Cold sprays of 18° to 22° R. [72°–81.5° F.] are used upon the neck and chest, usually a warm or cool bath, for the purpose of stimulation, especially of the respiratory center; also used independent of a bath in hysteria, epilepsy, and enuresis.

Cold-water bathing, also bathing sometimes with brandy or eau de Cologne, are of use to refresh the body and to stimulate metabolism.

To **stimulate perspiration** employ either hot baths or dry hot packs, or pack the whole body; the latter is done by placing three or four jugs containing hot water in the bed wrapped in wet cloths. This treatment is supplemented by the administration of hot lemonade or tea.

MEDICINAL TREATMENT

Medicinal treatment is to be resorted to only to meet exact indications. The most convenient form is either the liquid or powder, although small pills, capsules, and granules uay be used. Tablets must be crushed before use. The taste is always to be considered, and to disguise it give simple syrup or sugar-water to each individual dose. Bad-tasting medicaments must not be given in the food, for the latter will thus partake of the disagreeable taste;

insoluble powders may be given in a thick gruel or cocoa; bromid or iodid solutions in cold milk; quinin in slightly sweetened cocoa; bromoform in the yelk of an egg; castor oil heated in a warm spoon or dusted with sugar, or in bouillon or raspberry juice.

Dosage.—As a general rule, give as many twentieths of the average adult dose as the child is years old (Neumann).

Measurements.—One coffeespoon = 5 gm.; 1 children's spoon = 8 to 10 gm.; 1 tablespoon = 15 to 20 gm.; the most useful are the graduated medicine-glasses.

In the use of different remedies (narcotics), especially during the nursing age, great foresight must be practised, and it is always safest to first give a small test-dose.

Aside from administration by the mouth, medicines may also be given by subcutaneous injections, by enemata (in amounts from 15 to 25 gm., lukewarm), for gastric and intestinal irrigation; externally in inunctions, gargles, painting, insufflations, etc.

Expectorants.—Radix ipecacuanhæ in infusions, 0.2 to 0.3 : 100.0; liquor ammonii acetatis, 1 to 2 per cent., as a supplement to mixtures; sodium bicarbonate, 2 per cent.; radix senegæ, 3 to 5 per cent., in decoctions (for irritating coughs); acidum benzoicum, 0.03 per dose, with an equal amount of camphor; apomorphinum hydrochloricum, 0.005 to 0.03; terpin hydrate, 0.1 to 0.25; creosotal, 3 × 3 to 10 gtt.; vapor inhalations and damp-cloth hangings.

Laxatives.—For nursing infants, magnesia, just enough to cover the point of a knife to a dose; powdered magnesia with rhubarb, a like dose; syrup mannæ, syrup of cascara sagrada, or syrup of rhubarb, coffeespoonful doses; castor oil in coffeespoonful doses; calomel, 0.001 to 0.04. For older children give castor oil in doses of from a coffeespoonful to a tablespoonful; tamarind; infusion sennæ comp. in children's spoonful doses; purgen and baby purgen, one or two tablets, califig in coffeespoonful doses; folliculi sennæ (5 to 10 leaves boiled for one-half minute); Barber's sagrada tablets; the neutral salts.

Astringents.—Alumin. acetic, internally, .5 per cent.; externally, 2 to 4 per cent.; silver nitrate, .04 per cent., internally; 3 per cent. decoctions of calumba roots; bismuth salicylate, 3 to 5 per cent.; tannigen, 0.05 to 0.5 per dose; tannalbin, 0.25; tincture of veratrum, 0.1 : 5.0; diluted spirits, 3 to 10 gtt. every hour; 5 per cent. decoctions of the leaves of uva ursi; 5 per cent. infusion or the fluidextract of the leaves of jambul.

Emetics.—A coffeespoonful of the infusion of the root of ipecacuanha, 1.0 to 2.0 : 50.0, every ten minutes; powdered ipecac, 1.0 to 2.0 : 50.0; syrup of althæa; apomorphin hydrochlorate, 0.0008 to 0.003, subcutaneously.

Narcotics.—Aquæ amygdalæ amaræ, 1.0 : 100.0; codein phosphate, $\frac{1}{10}$ to $\frac{1}{2}$ mg., for nursing infants, later 0.005 to 0.05; heroin hydrochlorate, $\frac{1}{2}$ to $\frac{1}{3}$ mg. per dose; tincture of opium, not before the third month; at one year, $\frac{1}{3}$ drop per day; at two years, 1 gtt.; from two to four to ten years, 1 to 2 to 5 drops per dose; morphin hydrochlorate should not be given until after the third year, 0.001 per dose; chloral hydrate, 0.1 to 0.5 by mouth, 0.2 to 1.0 by rectal injections; extract of belladonna, 0.001 to 0.003 per dose; bromoform, 1 to 6 drops three times daily; atropin sulphate, 0.0002 to 0.0003, subcutaneously.

Nervines.—Potassium bromid, 0.3 per day, depending upon the age (it is best to combine this with sodium and ammonium bromid and sodium bicarbonate; the latter is given in double doses). Erlenmayer's bromid water, $\frac{1}{4}$ to $\frac{1}{2}$ to 1 bottle daily; Sandow's effervescent bromid salts; the muriate of quinin, 0.1 to 0.3; tincture of valerian, 20 to 40 gtt. per day.

Alterants.—Iron (see Anemia, p. 145); arsenic (ibid.); iodin, externally, as tincture of iodin; iodovasogen, potassium iodid ointment, for internal use (see Scrofula); mercury (see Congenital Syphilis).

Stimulants.—Liquor ammonii anis., 2 to 5 gtt.; sweet spirits of niter, same dose; camphor, 0.01 to 0.03, internally; camphor and ether, subcutaneously; wine, champagne, and inhalations of oxygen.

Diaphoretics.—Pilocarpin, $\frac{1}{2}$ to 3 mg., subcutaneously, in double doses internally.

Diuretics and Cardiants.—Calcium acetate, 1 to 2 per cent.; diuretin, 0.05 per dose; infusion of the leaves of digitalis, 0.3 : 100.0, from 1 teaspoonful to 1 children's spoonful every three hours; tincture of strophanthus, 1 to 3 drops; caffein, 0.1 per dose.

For the remaining remedies refer to the different diseases.

Psychic Treatment.—This is not a very practical therapeutic remedy; suggestion, advice, persuasion, deception, and threatening have some effect. These are of practical value in chorea, enuresis, during the convalescence of whooping-cough, hysteria, and disturbances of speech.

Mechano-electric Therapeutics.—General and local for stimulation of metabolism, to strengthen diseased or paralyzed muscles, to mobilize joints, and to scatter an exudate. The galvanic and faradic currents are employed for the same indications; also for nerve stimulation, as in enuresis and hysteria.

DISEASES OF THE NEWBORN

GENERAL LOSS OF VITALITY AND PREMATURE BIRTH

Lack of development in size, weight, and function of the body at the time of birth usually exists in prematurely born infants, but may also occur in full-term babies whose parents are unhealthy, or who themselves are already diseased.

Symptoms.—Abnormally small and underweight children show prematurity in the nails, skin, and genitalia; their respiration and pulse can hardly be detected; they sleep constantly; react slowly to external stimulation; nurse little or none at all; the temperature varies between 30° and 28° C. [86° and 82.4° F.]; the umbilical stump is slow in healing. The face is small and weazened, the voice weak, and the body very sensitive to every change in temperature. Such infants usually die from scleroderma, pneumonia, or asphyxiation. The possibility of life exists only when the weight at birth is at least 1000 gm.; by means of careful nursing it is possible then to save one-half, and when the weight at birth is greater, 80 per cent. of such children continue to live.

Treatment.—The application and maintenance of heat. The infant is wrapped in wool, surrounded by hot-water bottles, and kept in a warm room at a temperature from 18° to 20° R. [72.5°–77° F.]. The following specially prepared brood chambers may be employed: A chest with movable glass covers, which is lined with peat moss and heated bricks placed underneath or upon the floor; a bath-tub with double walls between which hot water flows (Credé); thermophore; incubator with appliances

FIGURE 20

I.–V. Incubator Room for Three to Five Infants. Escherich-Pfaundler System.—(Original in the Clinic of Pfaundler.)

Explanation: The framework of the incubator room is constructed of iron; the sides and roof are lined with plates of cork, while the remainder is simply glazed. The walls are coated on both sides with enamel paint. The size of the room is large enough to accommodate a nurse or attendant, so the infants need never be removed and exposed to the injurious effects of a different temperature. The heat is obtained from a system of cast-iron tubes (I., *a*), which are of solid capacity. These tubes are connected with the hot-water pipes of the house. An extra stove (A) is set up in the wash-room as a reserve. The temperature of the room is regulated as may be desired between 28° to 34° C. [82.4°–93° F.] by means of the ventilation apparatus (F); an electric contact-thermometer registers the gross temperature changes.

The fresh air enters the room through a shaft (B) from without the building (Park), passes through a cotton filter over the heating pipes, and finally over a moistening apparatus which can be regulated (two basins with oblique floors filled with water). By filling the latter to a certain height, the atmosphere in the room may be so regulated that it will hold a relative moisture of 60 per cent. The psychrometer shows then the difference between the temperature of the two thermometers to be 5° C. [9° F.]. The bed shared in common by the infants rests on a perforated metallic plate (G), which is surrounded by a railing. The floor of the room and of the incubator is composed of xylolith. The other arrangements of the room are shown on the plan (I.).

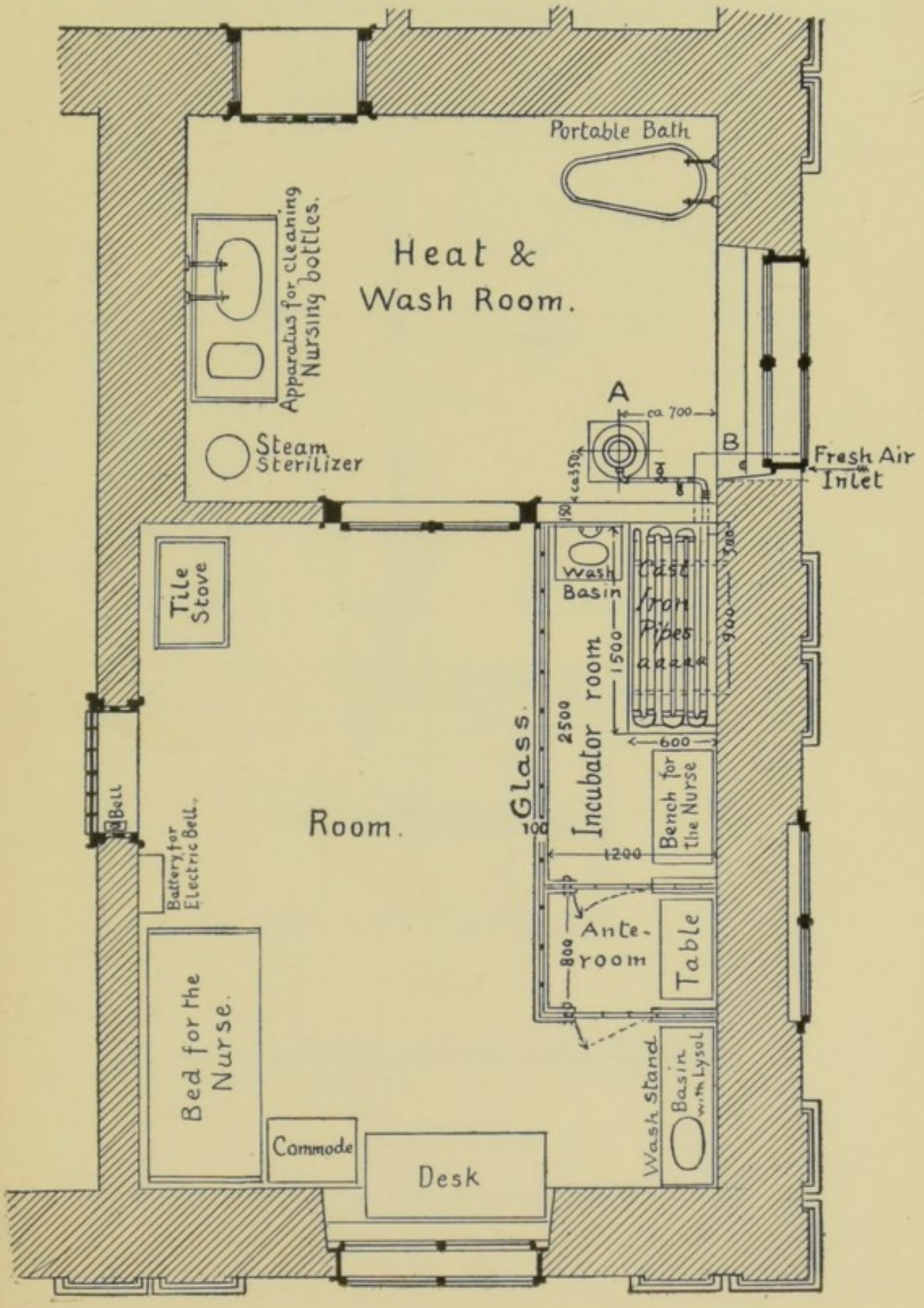
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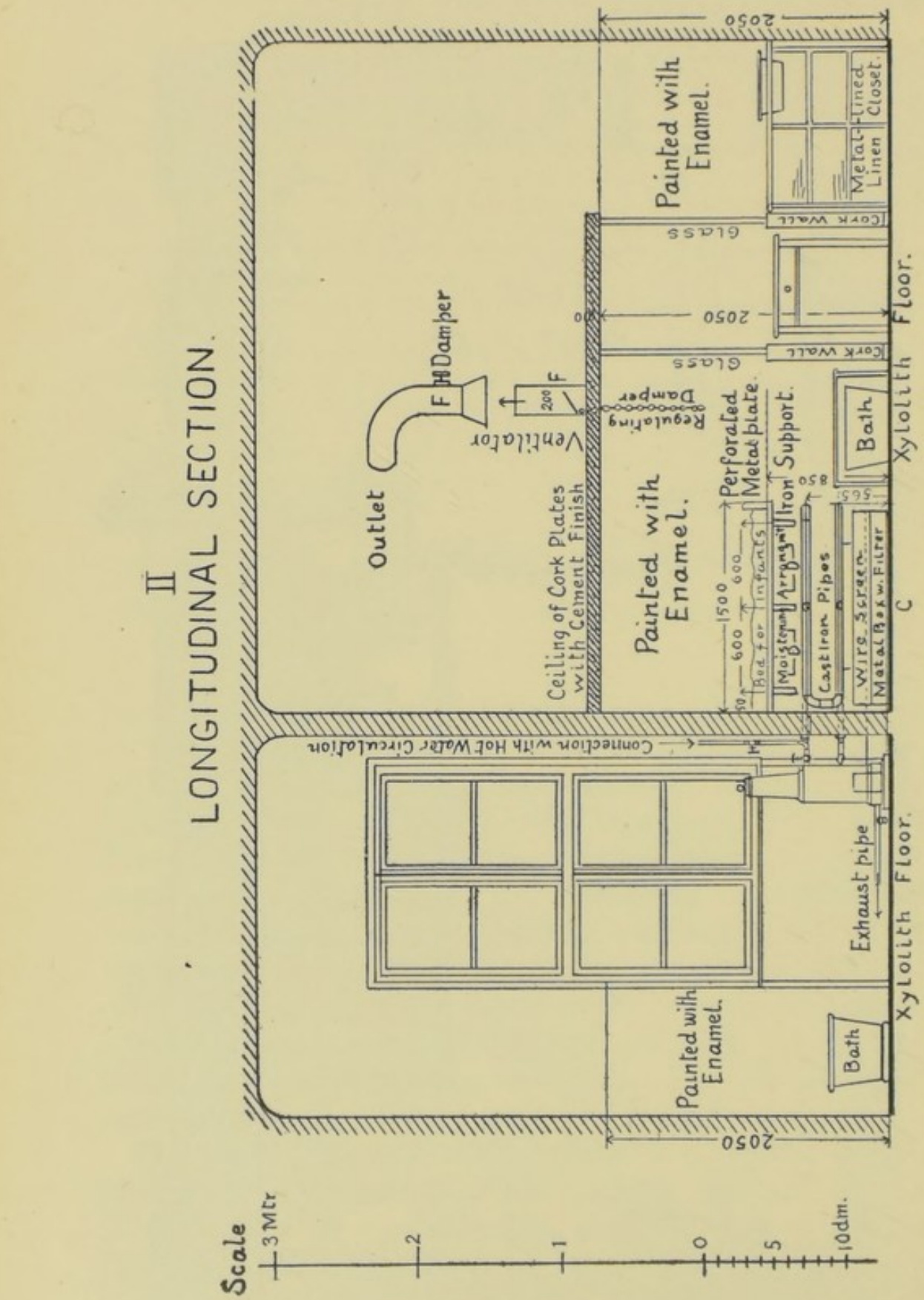
for regulating warmth and moisture, and supplied with an apparatus for heating and for discharging the air which is breathed (Lion, Rommel, and others); the temperature in the incubator should be about 30° C. [86° F.]. The incubator which offers the greatest hygienic advantages is that invented by Escherich and Pfaundler, which, however, is only adaptable for hospitals. Children with poor vitality should be frequently moved and repeatedly carried about every day in order to prevent atelectasis. [Specially improvised rooms and cabinets are in use as incubators for premature children. The details of their construction need not be gone into here.—Ed.]

Feeding.—Mother's milk, which is squeezed from the breasts, is administered with a spoon through the nose. If such milk is unobtainable, give the milk mixtures of Backhaus, Voltner, Biedert, etc., [modified milk with a low per cent. of fat, sugar, and proteids, *e. g.*, fat, 0.25 per cent.; sugar, 4 per cent.; proteids, 25 to 50 per cent.].

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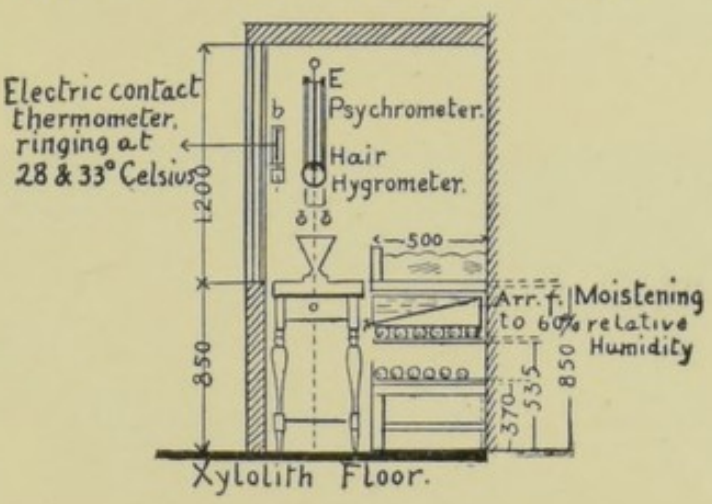
GROUND PLAN.




II
LONGITUDINAL SECTION.
Outlet
Ventilator
F
Damper
200
Regulating Damper
Ceiling of Cork Plates with Cement Finish
Painted with Enamel.
Perforated Metal plate
Iron Support
Bed for Infants
Cast Iron Pipes
Wire Screen
Metal Box w. Filter
Bath
Cork Wall
Glass
Metal Lined Closet.
Linen
2050
1500
600
850
565
C
Xylolith Floor.
Connection with Hot Water Circulation
Exhaust pipe
Scale
3 Mtr
2
1
0
5
10dm.

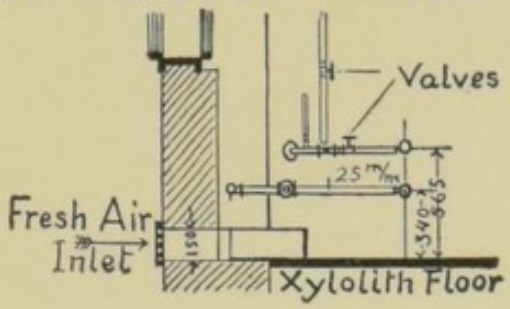
III

SECTION THROUGH INCUBATOR ROOM.



IV

SECTION THROUGH WINDOW PARAPET.



V

GROUND PLAN OF CIRCULATION SYSTEM.

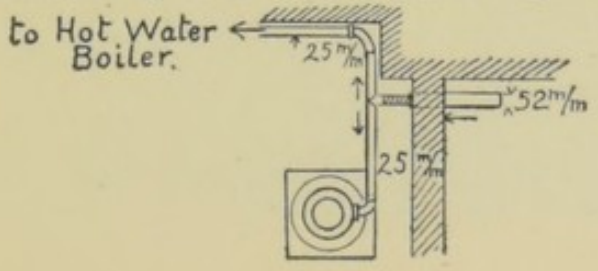


PLATE 4

Congenital Umbilical Hernia.—The hernial sac is filled with intestines and a portion of the liver. The amnion is discolored and is becoming gangrenous. It shows thickened folds at the junction with the abdominal walls. (Escherich's Clinic, Vienna.)

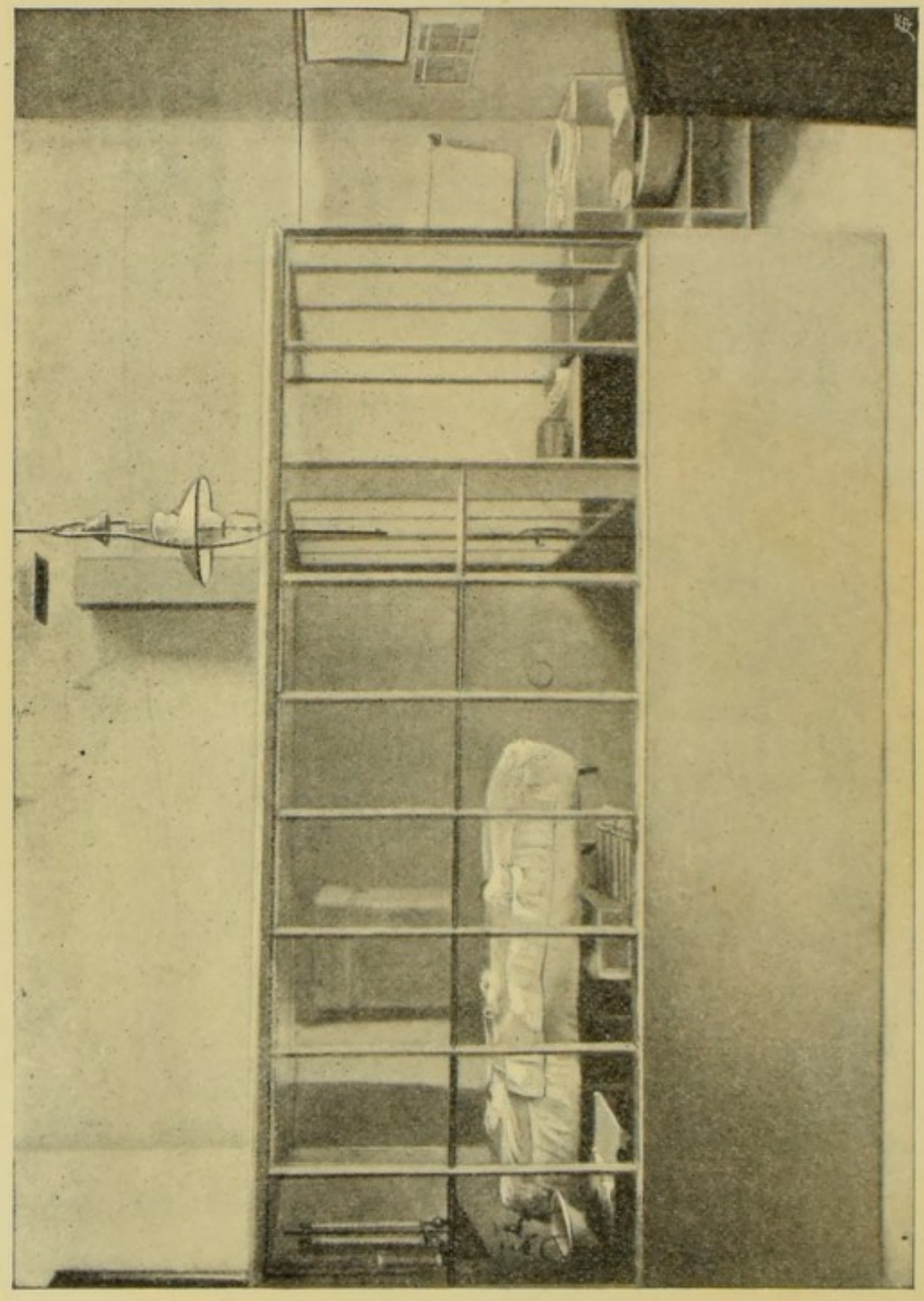
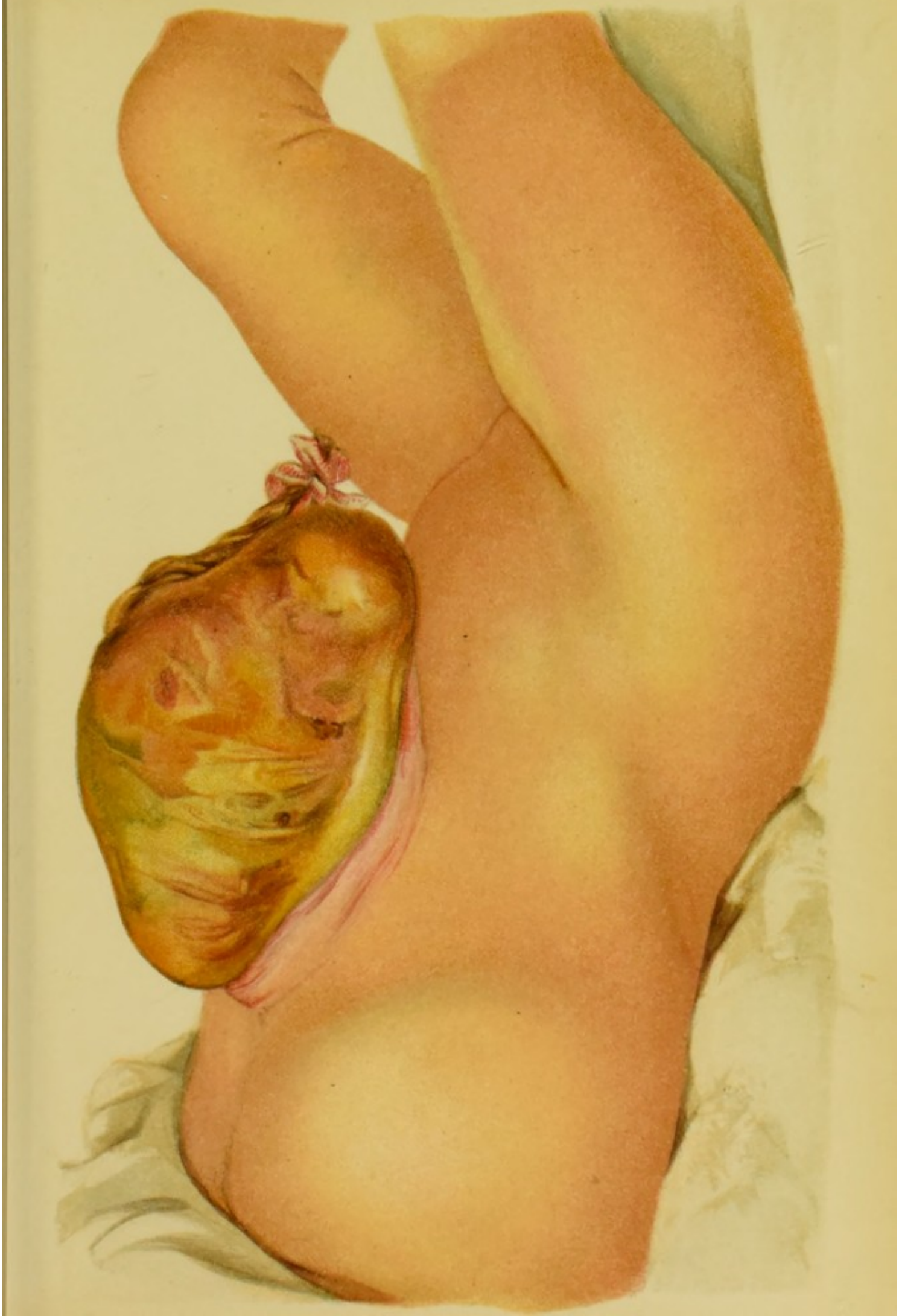


FIG. 21.—Incubator room. Constructed by Escherich and Pfaundler.



DISEASES OF THE UMBILICUS

TREATMENT OF THE NORMAL UMBILICUS

It is best to allow only a short portion ($1\frac{1}{2}$ to 2 cm. [.6–.8 in.]) of the umbilical cord to remain. The stump is covered with a dry sterile dressing, which is permeable to the air and which does not cause any tension ; apply no ointment. Authorities have not yet agreed as to whether the child should be bathed daily—excepting the first bath —or not until after the cord has fallen off. At any rate, the greatest cleanliness must be observed in bathing the infant. In the case of premature births and weak children, on account of their susceptibility to septic infection, it is wisest to postpone bathing until the umbilical cord has healed.

CONGENITAL UMBILICAL HERNIA

Funicular Umbilical Hernia, Omphalocele.—The abdominal wall around the umbilicus is the last to close. If any cause hinders this, the abdominal cavity does not become enclossd by the union of the walls, but only by closure of the peritoneum and the sheath of the umbilical cord, that is, the amnion. This thin covering is pressed forward by the viscera and forms an umbilical hernia. The umbilical cord is inserted at the apex of the hernial sac. Within the protruding mass, which varies in size from a walnut to a child's head, may be seen the intestines, and frequently also the liver and kidneys ; at the point where the abdominal walls pass into the amnion a swollen ring of tissue is formed. Small hernias may be cured with an ointment and bandage, which cause the amnion to become gangrenous. Larger hernias require surgical intervention.

ACQUIRED UMBILICAL HERNIA

This form of umbilical hernia does not occur until after the cord has dropped off and the wound healed. Favoring this condition are insufficiency of fat in the abdominal walls, too great intra-abdominal pressure when crying, or difficult micturition (phimosis) and meteorism.

The thin umbilical scar succumbs to the pressure, the umbilical ring stretches, and the hernia is produced. At first it is temporary and occurs only when the child cries and presses downward, later, however, it becomes permanent. The hernia, which usually contains a loop of small intestine, varies in size from that of a pea to an apple; larger hernias are elongated, pendulous, and possess a

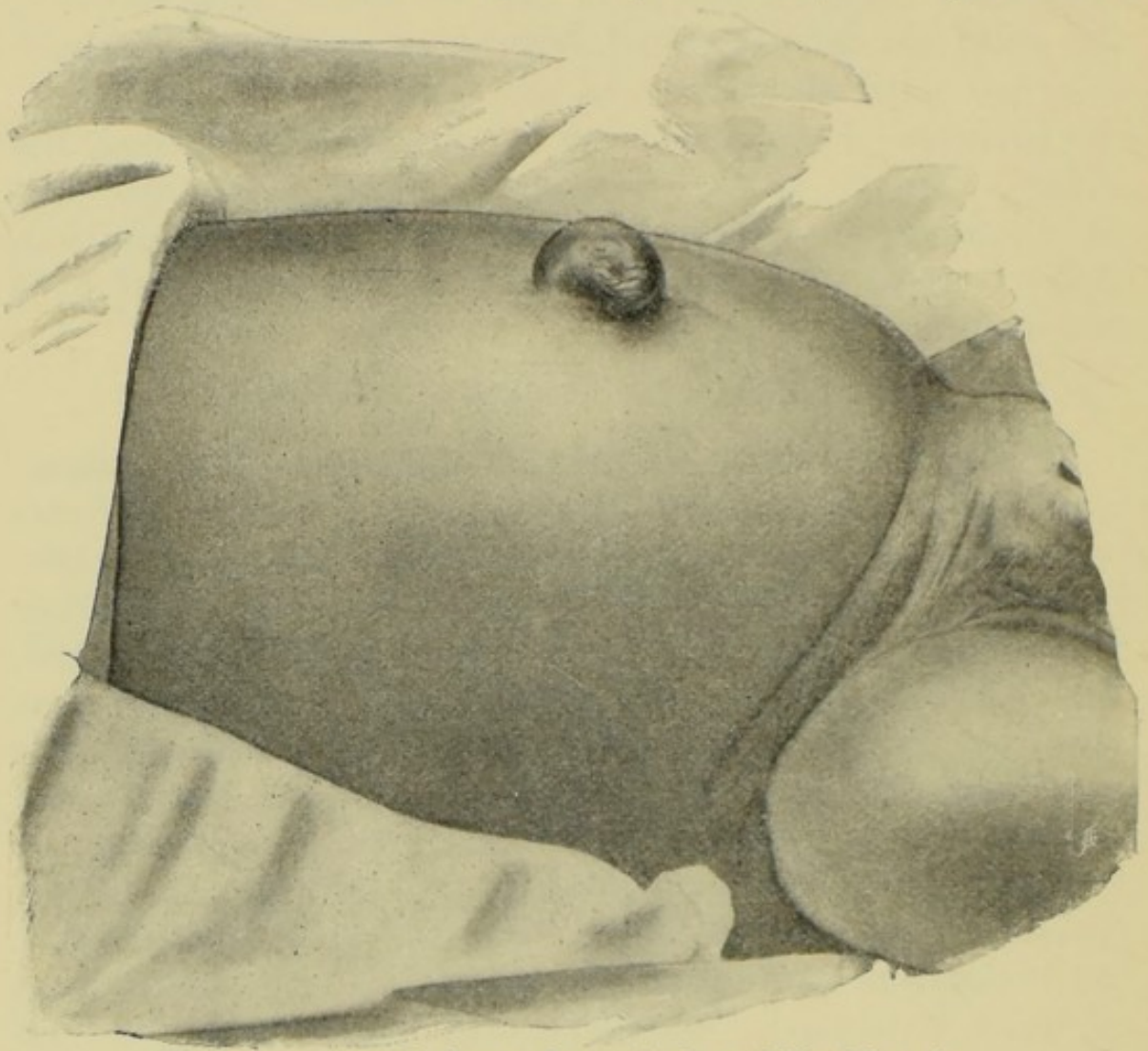


FIG. 22.—A mild form of acquired umbilical hernia.

dark pigmented tip. Umbilical hernias are usually easily replaced and only rarely become incarcerated.

Small hernias frequently heal spontaneously in the course of the first or second year of life by contraction of the umbilical ring.

Treatment.—Encourage spontaneous cure by relieving the pressure upon the umbilical ring by means of strips of adhesive plaster; the reduced hernia is pressed down-

ward by two folds of the skin, one from either side, which are kept in place with a wide strip of adhesive plaster (5 cm. [2 in.]) in such a manner that their surfaces touch each other. This strip reaches from one hypochondrium to the other. If the plaster, which remains in place when bathing, is protected during the first few days by a cloth binder, it should last for from one to three weeks. When changing binders it is advisable to clean the skin

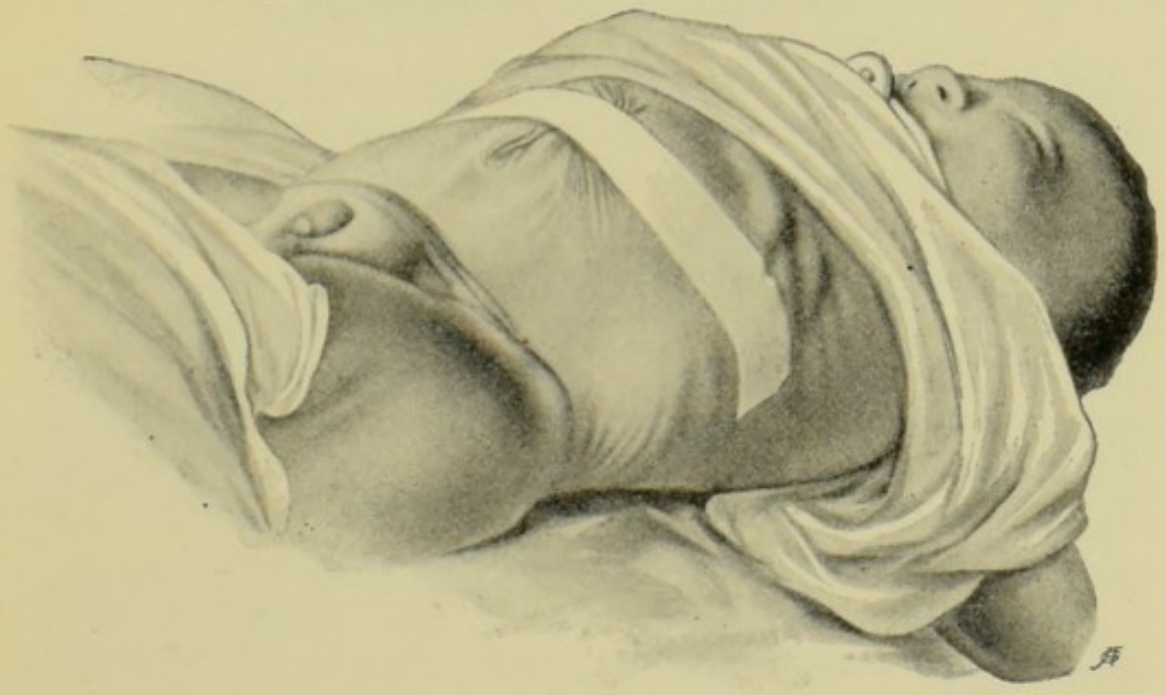


FIG. 23.—Band of adhesive plaster over an acquired umbilical hernia. The plaster is tensely drawn and applied and fastened over the ribs on both sides, so that a longitudinal fold of the abdominal wall is drawn over the hernia.

with ether. In place of this dressing a small plate made of folded adhesive plaster, or a cork plate, may be employed; it must be at least 1 cm. [$\frac{1}{2}$ in.] larger in diameter than the hernial orifice. It is held in place by means of two crossed strips of adhesive plaster. The treatment may usually be discontinued after several weeks or a month, yet healing may be hoped for after even several years. Hernia-bandages or circular rubber hernia-bands should be rejected. In case the hernia resists this treatment or when the opening is too large, an operation is necessary, that is, a radical operation or the more recent paraffin injection method (Escherich). The par-

affin (melting-point 39° C. [102.2° F.]) is injected into the hernial sac after the hernia has been reduced, after which the contents of the sac are again allowed to protrude, and covered for a short time with an ice compress. The latter causes the paraffin to harden, and it in turn pushes the hernia inward. A sterile dressing is applied for several days.

UMBILICAL HEMORRHAGE

We distinguish two forms of umbilical hemorrhage: One which occurs immediately after birth from a torn, poorly tied, and insufficiently thrombosed umbilical cord; and the other from the umbilical wound, which does not occur until after mummification or after the cord has fallen off. The latter form occurs in sepsis, syphilis, and acute fatty degeneration. The hemorrhage is of a parenchymatous character, and arises suddenly or gradually; it is not always continuous and leads, after a few hours or days, with symptoms of severe anemia, to death. The pale red blood shows no tendency to coagulate.

Treatment.—The first form of hemorrhage is usually controlled by early ligation, but the second form is usually fatal. The treatment consists in instituting the following procedures: Tampons of chlorid of iron; digital compression; suture of the umbilicus (Dubois); filling the umbilical groove with plaster of Paris (Hill); clamping of the umbilicus with forceps (Fischl); gelatin externally and subcutaneously; and adrenalin (1 : 1000) subcutaneously. [The local treatment by chlorid of iron and digital compression is unavailable in most cases. Suture and plaster of Paris have been recommended. Gelatin has been recently reported on favorably. Adrenalin has also given favorable results applied locally.—Ed.]

UMBILICAL FUNGUS. UMBILICAL GROWTHS

An umbilical fungus is due to an excessive development of the granulation tissue, which occurs normally in conjunction with the healing of the wound. These are

little red growths which reach the size of a cherry and situated in the depths of the umbilical groove, or they rise in a fungus-like manner above its sides, and always discharge a little secretion (see Plate 18, Fig. 2).

Treatment.—Apply caustic; in case of larger growths tear off or remove with a pair of scissors. [Treat by solid nitrate of silver or, if large, may be tied off with fine silk.—Ed.]

INFECTION OF THE UMBILICUS

The umbilical wound may become infected through unclean hands, dressings, instruments, etc. The virus infects either the umbilical wound itself and then leads to suppuration, ulceration, and gangrene, or it spreads to the surrounding tissue and there causes a phlegmonous inflammation (periomphalitis). Then again it may travel through the Whartonian jelly in the walls of the umbilical blood-vessels, preferably the arteries, where it sets up severe inflammation (periarteritis). The phenomena which arise are:

Pyorrhea of the Umbilicus.—Dried secretion collects at the reddened entrance of the umbilicus, an odorless pus flows forth from the umbilical wound, and a suppurative granulation occurs at the base of the wound. There are but slight constitutional symptoms.

Omphalitis and Periomphalitis.—These are marked by swelling and reddening of the umbilical fold, painful phlegmonous swelling and bulging forth of the area surrounding the umbilicus, and the appearance of enlarged lymph-vessels upon the abdominal wall. This condition terminates in spontaneous healing, abscess formation, gangrene, and fatal peritonitis; under certain circumstances a superficial ulceration of the umbilical wall results. It is always accompanied by fever.

Gangrene of the Umbilicus.—The swollen or phlegmonous area turns bluish black, the discoloration spreads rapidly, and the tissue softens and sloughs off, leaving a discolored and foul-smelling ulcer. As a rule the process spreads over the peritoneum and the symptoms of

peritonitis develop. The patient suffers from abdominal pain and distention of the abdomen. Death follows from sepsis or peritonitis.

Umbilical Arteries.—Local phenomena are frequently absent. The patient becomes restless and develops fever, intestinal disturbances, slight icteric coloring of the skin, temporary erythema or punctiform hemorrhages in the skin, or we notice, proceeding from within outward, the signs of a septic omphalitis. Occasionally it is possible, by squeezing from below upward, to force pus out of the umbilicus. Bacteria are found in the venous blood. The result is usually death after a few days from peritonitis and collapse.

Anatomically both arteries (the vein is rarely involved) are, as a rule, observed to be thickened and discolored grayish brown; the lumena are filled with pus or semi-solid thrombi, the intima is softened and ulcerated, and the surrounding tissue is infiltrated and discolored green.

Aside from these disturbances there occur in nearly all cases ecchymoses and degenerative changes in the organs of the chest and abdomen, pulmonary infarcts and consolidation, multiple abscesses, and enlarged spleen.

Treatment of Infection of the Umbilicus.—The prophylaxis consists in the careful observation of asepsis in treating the umbilicus, especially in premature births and weakly infants. Mild cases are cauterized and covered with a dry antiseptic powder. In severe cases make applications of aluminum acetate or lysol ($\frac{1}{4}$ to $\frac{1}{2}$ per cent.); pus should be removed with sterile swabs. In case of suppurative phlegmon make warm applications, and later incise. Feeding must receive careful attention and, if possible, the child should receive nourishment from its mother's breast.

SEPSIS OF THE NEWBORN

Etiology.—The newborn infant shows a special predisposition to septic infections because of the undeveloped state of its protective mechanism (skin, mucous membranes, lymph-nodes, spleen). The causal agents of infection are

the various pus, inflammatory, and putrefactive bacteria. The infection occurs before or, as a rule, after birth. It develops *in utero* through infected liquor amnii or by transmission through the placenta. After birth it is caused by micro-organisms, some of which were deposited in the body itself (auto-infection), while others are introduced from without (hetero-infection). The most important points of entrance for the infection are existing inflammations or loss of substance of the umbilicus, the skin, the oral cavity, the lungs (Fischl), the intestinal canal, the bladder (Escherich, Trumpp, Epstein), the ears, the eyes, and, finally, apparently uninjured skin and mucous membrane. Everything which comes in contact with the child's body in the course of its care may serve as the source of infection, including the hands, bath-water, clothes, sponges, thermometer, the air, incubator, etc.

Morbid Anatomy.—If the toxin has entered the circulation and caused blood intoxication, we find parenchymatous degeneration of the internal organs, ecchymoses in the mucous membranes, and thin sterile blood. In case of blood infection degenerations and ecchymoses are also found, together with multiple abscesses, pneumonia, inflammation of the serous membranes, ulceration of the mucous membranes, and a rapidly developing foul-smelling putrefaction.

Symptoms.—There is no regularity in the symptomatology, for, on the contrary, it assumes many forms and, indeed, in some cases is quite indefinite. The symptoms develop immediately or in a few days after birth, and consist in a loss of appetite, high fever, a choleraic diarrhea, great acceleration of pulse and respiration rate, somnolence, and a rapid loss of strength. Hemorrhages occur in the dirty icteric and, later, cyanotic skin. Restlessness, tremor, and convulsions are noted. The high temperature falls to or below normal, and in many cases symptoms of pneumonia, peritonitis, pleuritis, meningitis, multiple inflammation of joints, embolism, and suppurative processes of the skin develop. The primary focus may be apparently absent or it appears as a suppurative, phleg-

monous, ulcerated or gangrenous process of one of the previously mentioned points of entrance. The course is nearly always unfavorable; healing in very mild cases or an early limitation of the process is possible.

Diagnosis.—This is not easy when the external localization is absent. Designating the indefinite and unclear constitutional condition as a septic one is made easier by the ophthalmoscopic finding of a retinal hemorrhage or by discovering pus-germs in the aspirated venous blood or in the cerebrospinal fluid.

Treatment.—Prophylaxis consists in insisting upon thorough cleanliness in the care of the pregnant woman and of the child, especially as regards the linen, the treatment of the umbilicus, the bath, the thermometer, the feeding apparatus, the preparation of the milk, hygiene of the milk, hygiene of the room, etc. Rhagades or injuries occurring during birth must be treated antiseptically. The treatment of the condition itself consists in supporting the child's strength, the administration of mother's or hot cows' milk, stimulants, meat broths, wine, and infusions of normal salt. To combat the fever give baths, cold pack, and quinin (0.1 gm.). Abscesses should be opened.

BLENNORRHEA NEONATORUM, OPHTHALMIA NEONATORUM

Blennorrhea neonatorum is a directly transmissible inflammation of the conjunctiva of newborn infants caused by the gonococcus of Neisser. A large percentage of all cases of total blindness is due to this condition. The infection occurs either immediately after birth, during which the infected genital secretion enters the eyes, or later through infected fingers or toilet articles.

Symptoms.—The symptoms develop usually within the first week, but in later infection they do not appear until after that period. The eyelids are red and swollen and a bloody, serous secretion flows from the palpebral fissure. The swollen lids increase in size during the next one or two days and form, especially the upper lid, a

vaulted eminence. The secretion becomes converted into a yellowish-red pus. The conjunctiva of the lids is swollen and greatly reddened. When the lids are inverted the transitional folds bulge forward as tense, shiny red eminences. The conjunctiva of the eyeball is also swollen and congested. If the secretion is not constantly removed it may erode the cornea; in that case a minute speck develops in the middle of the cornea, which rapidly enlarges, turns yellow, and its superficial portion undergoes destruction. The resulting suppurating ulcer shows a great tendency to spread and to perforation. The complications of this infection of the cornea may be central macula, anterior capsular cataract, prolapse of the iris, staphyloma, or panophthalmia. After a number of days the secretion becomes thicker, yellowish green, and excreted in such profuse amounts that it oozes from the palpebral fissure as often as the lids are opened. In the course of several days the swelling of the eye diminishes, the palpebral conjunctiva becomes granular, and the secretion gradually lessens. In from two to three weeks the conjunctiva becomes pale, and finally nothing is noticed excepting a little mucopurulent secretion at the inner canthus.

Course and Prognosis.—The total duration is from three to five weeks or even longer. The later the disease develops the more favorable the prognosis. It is unfavorable in the newborn and in weakly and sickly children. Even in infants otherwise healthy the prognosis should always be guarded.

Treatment.—As a prophylactic measure drop a 2 per cent. solution of nitrate of silver in both eyes after birth (Credé). It is usually impracticable to protect the healthy eye in newborn infants, and furthermore both eyes are infected, as a rule, from the very beginning. In the acute stage the treatment should be as mild as possible, in order to avoid injuring the conjunctiva. The pus as it collects should be regularly and as frequently as possible removed from the conjunctival sac, preferably by rinsing with a mild solution (sodium chlorid, 0.6 per

PLATE 5

Ophthalmoblennorrhea of the Newborn.—(From Haab's *External Diseases of the Eye.*)

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cent ; boric acid, 4 per cent. ; dilute solution of potassium permanganate). It is allowed to flow every quarter or half hour from a low height into the eye, which is held open with the fingers. During the intervals apply cool compresses made from one of the above-mentioned solutions (employ no ice compresses, which may increase the damage already done to the cornea). Not until the swelling has lessened and pseudomembranes no longer develop should a caustic be employed. For this purpose use a 3 per cent. silver nitrate solution, which should be neutralized later with normal salt. The treatment is the same when the cornea is involved. When perforation is threatened the intra-ocular pressure may be relieved by pilocarpin. In order to encourage the child to open its eyes spontaneously the room should be darkened and the eyes slightly cocainized. In cases running a slow course a 20 per cent. protargol salve should be employed (Salzer).

TETANUS NEONATORUM

This condition consists of tonic spasms of the general musculature, which begin in the muscles of mastication and which increase in severity in paroxysms. These spasms are brought about by the toxin of the bacillus of tetanus.

Etiology.—The bacillus of tetanus, which is quite prevalent in garden earth and wood, is transmitted to the umbilical wound through carelessness in the care of the infant. In this location, from which oxygen is excluded, it finds conditions favorable to its development and travels from there into the body. The bacillus may be demonstrated in the blood or in the umbilical wound.

Symptoms.—In from five to six days after prodromal symptoms, consisting of restlessness, crying out in sleep, and tremor of the jaw, the child becomes unable to take food because of the tonic spasm of the muscles of masti-



cation. Infants fed from the breast show the first indication of the disease by biting the nipples, and when

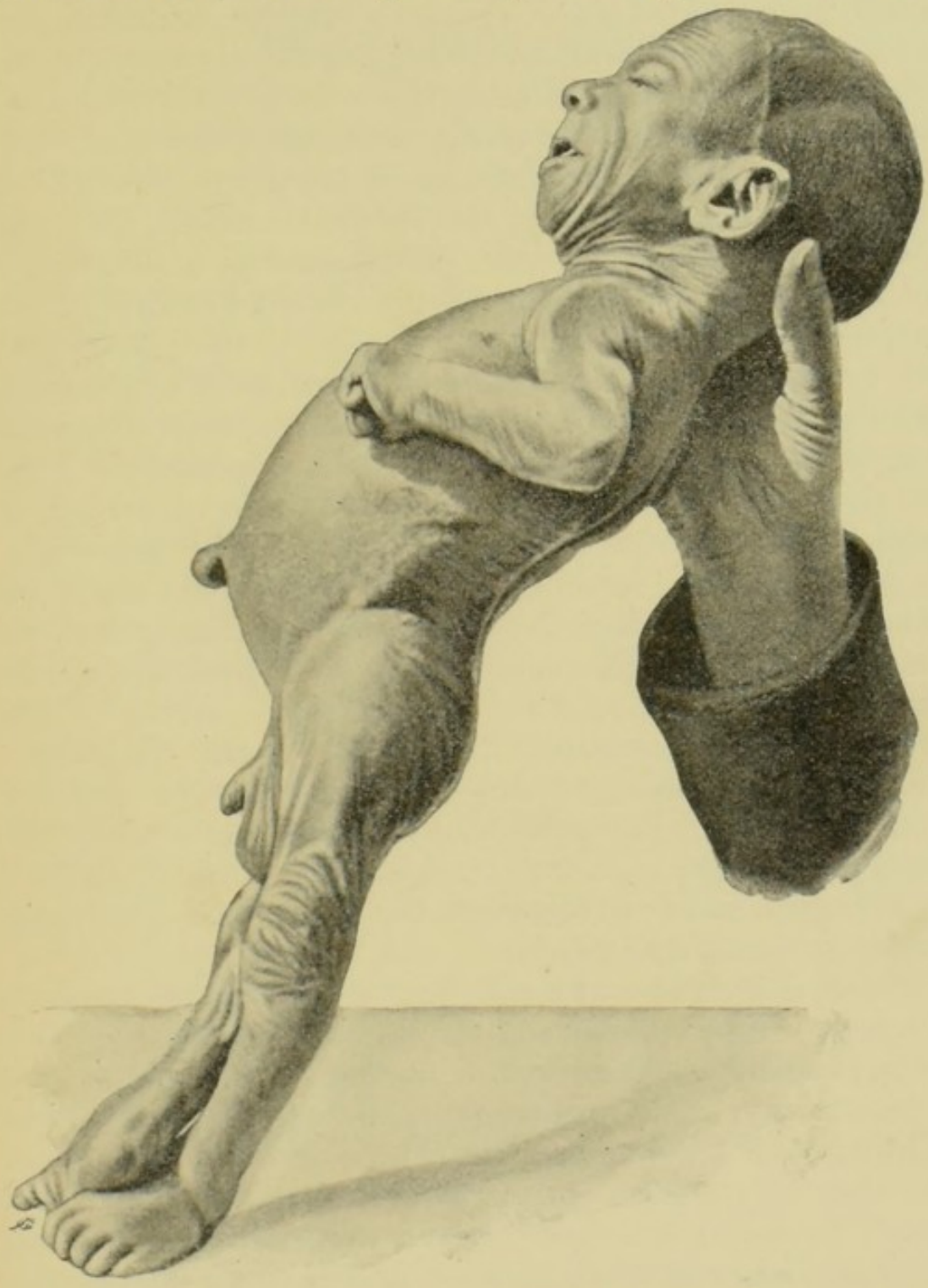


FIG. 24.—Tetanus neonatorum. The body is rigid; the expression of the face and the position of the arms and legs are characteristic. (Escherich's Clinic, Vienna.)

liquids are administered they flow out again. The cheeks become hard as a board. Extension of the spasm to the

remaining muscles of the face, the muscles of the neck, trunk, and extremities presents a typic picture. The mouth is puckered as if about to whistle or laugh, the forehead and eyebrows are wrinkled, the eyes are closed, and deep furrows extend from the nose to the lower jaw. The head is held drawn stiffly backward, the neck and back are in a state of opisthotonos, the arms flexed and pressed against the body, the hands clenched, the legs and feet extended, and the abdomen has a board-like hardness. The whole body of the child when lifted feels like a stiff wooden doll. Involvement of the pharyngeal and respiratory muscles causes difficulty in swallowing and breathing. The spasm is not continuous, but lessens in severity to a certain degree for a short period of time and then is followed by a convulsive tremor of the whole body. The intervals of quietude last at the beginning for a few minutes, but later only for a few seconds. The spasms are exacerbated by external mechanical irritation, moving the body, the administration of food, etc. The whimpering of the child indicates the severity of the pain. The temperature is high (40° to 42° C. [104°–107.6° F.]), especially toward the end, and the pulse is small and weak. The child dies in a few days or, at the most, in a week, from exhaustion or respiratory failure. In the rare cases of recovery the symptoms gradually disappear one after the other.

The diagnosis is easily made when the disease is pronounced. Whenever newborn infants refuse nourishment the musculature of mastication should be examined.

Treatment.—As a means of prophylaxis the mother and child should receive the most rigid aseptic treatment. The results from the use of Behring's tetanus antitoxin cannot be depended upon yet, and the treatment is therefore mainly symptomatic. Rest is absolutely necessary; feeding is carried on by means of a tube through the nose. Narcotics and repeated chloroform inhalations are necessary until sleep sets in (Heubner). Chloral should be administered by means of enemata twice daily in 0.5-gm. doses, and potassium bromid is given in the same manner

in from 1.0- to 1.5-gm. doses daily. Finally, an attempt should be made with the antitoxin as soon as possible.

MELENA NEONATORUM

Melena neonatorum is a rare disease which is characterized by hemorrhages into the gastro-intestinal tract, accompanied by bloody stools and the vomiting of blood. The real cause is unknown. It occurs in connection with septic processes, Buhl's disease, syphilis, and trauma.

Morbid Anatomy.—The stomach and intestines are filled with black blood, the source of which are minute erosions and round ulcers in the intestinal and gastric walls, or a diffuse hyperemia of the mucous membrane (diapedesis), thrombosis, or bacterial emboli. The remaining organs are anemic.

Symptoms.—This condition has a sudden onset during the first few days of life. The stools contain blackish blood-clots, which when deposited on the swaddling cloths are distinguished from the meconium by a dark red halo (see Plate 37, Fig. 1). The child also vomits bloody masses, but not in all cases. Collapse sets in early, the extremities become cold, the pallor increases, and the features have a death-like appearance. The child may develop the symptoms of hydrocephalus and die in from one to two days, or the hemorrhage ceases and recovery slowly sets in; in that case the stools continue to have a tea-like character for one or two days. Local symptoms, pain, etc., are absent. If sepsis develops the characteristic phenomena of that condition are also present. The mortality rate is 50 to 60 per cent.

Diagnosis.—The characteristic collapse and the anemia fail to appear in *melena spuria*, which consists in vomiting and passing per rectum blood that has been swallowed from the mother's nipple or from the nose and pharynx of the child itself.

Treatment.—Inject subcutaneously 15 ccm. of a 2 to 5 per cent. (Merck's) solution of gelatin (Zuppinger). Give 1 drop of liquor ferri sesquioxid in 1 spoonful of gruel. In every case an ice-bag should be applied to the abdomen

and heat supplied to the rest of the body. [The reports from gelatin injections are not uniformly favorable. The editor has observed symptoms of collapse in children who have received these gelatin injections. He believes the gelatin should be administered by mouth. Other medicinal treatment should consist of the administration of 1- or 2-drop doses of adrenalin solution.—Ed.]

ACUTE HEMOGLOBINURIA OF THE NEWBORN

(*Winckel's Disease*)

Acute hemoglobinuria of the newborn is, on the whole, a rare disease, occurring endemically and sporadically, and is associated with cyanosis, icterus, and hemorrhages from the various organs. Death occurs, as a rule, in thirty-two hours. Anatomically we find swelling of Pyer's patches and of the mesenteric glands, dark red discoloration of the kidneys (the pyramids of which show dark hemoglobin striation), and fatty degeneration of the liver and other organs. The urine contains hemoglobin, casts, bacteria, and detritus. Treatment is useless.

BLOOD-TUMOR OF THE HEAD. CEPHALHEMATOMA

A cephalhematoma is a collection of blood between the cranial bones and the periosteum, either external (between the periosteum and the skull) or internal (between the dura and the skull); as a rule it is a combination of both varieties. It is caused by laceration of blood-vessels and loosening of the periosteum during birth.

The external cephalhematoma appears as a fluctuating and elastic growth, varying in size from that of a hazelnut to that of an apple, which is usually situated over one of the parietal bones. It is joined to the edges of a bone and never extends over a suture or a fontanel. The skin is movable and somewhat bluish. After several days a wall is formed at the periphery of the growth, which is at first soft, but later as hard as bone. This wall is caused by ossification of the loosened periosteum. The growth

increases in size during the first four days, it then remains stationary, and after the second week grows smaller. On palpation, crepitation is felt because of the deposition of bony substance in the upper layer of the tumor. The swelling disappears in about twelve weeks and the prognosis is favorable.

Diagnosis.—A cephalhematoma is to be distinguished from the edema of the head occurring during labor, which is doughy in consistency and extends over the sutures, by its distinct limitation to one bone, growth after birth, fluctuation, and bony wall. The absence of inflammatory and constitutional symptoms differentiates it from abscesses. A cephalhematoma may be mistaken for cerebral hernia, but this error may be avoided by remembering that a cerebral hernia occurs between the bones, that it is reducible, pulsates, and is enlarged by crying and coughing.

Treatment.—This may be expectant and consists in applying a simple protecting bandage, followed in eight days by aseptic puncture and aspiration, or making an incision and applying a light pressure dressing (von Winckel). [The expectant plan of treatment is all that is required, as the condition tends to spontaneous recovery.—Ed.]

MASTITIS NEONATORUM

The physiologic swelling of the colostrum-secreting mammary glands of the newborn may lead, through infection from without, to inflammation. It is caused by unnecessary squeezing of the gland with unclean fingers. When the abscess, which is usually one sided, is not incised at the proper time, it discharges externally and causes permanent disturbance of individual portions of the gland.

Treatment.—Prophylaxis consists in forbidding the squeezing out of the milk in the breasts of the newborn. If inflammation is present apply aluminum acetate; in case of an abscess apply poultices. [Wet dressings of boric acid or hot boric-acid solutions may be used.—Ed.] Then make radial incision and dress with aluminum acetate.

MALFORMATIONS

Double monstrosities are of no practical interest. Of the single monsters only the most important will be considered here. Malformations arise, as a rule, from external or internal causes. The latter are chiefly typic forms, which are due to heredity or primary pathologic variations in the embryo. The former are due to any form of external injury which disturbs the embryonal rudiment in the course of its development; the atypic types are usually of this class.

MALFORMATION FROM ARRESTED DEVELOPMENT, MONSTRA PER DEFECTUM

(*Incomplete Closure of the Cerebrospinal Canal*)

Anomalous fontanels occur as osseous lacunæ in the mesial plane of the vault of the cranium. They are most frequently found in the region of the glabella and in the middle of the sagittal suture.

Dermoid spaces within the cranial bones themselves are most common in the parietal bones. The prognosis is good. If they persist until the walking period, the child must wear a cap with leather or metal plates.

The formation of clefts or fissures in the region of the cerebrospinal canal is due, according to von Recklinghausen, to primary agenesis and hypoplasia of the embryonal medullary ridge. If the arrest of the development occurs in the cranial portion of the canal it results in fissure formation of the cranium.

Cranioschisis or **acrania** is a congenital defect of the skull, which is accompanied either by total absence of the brain—total anencephalus—or by partial absence—partial anencephalus. The contents of the skull frequently protrude through the fissure, which acts as the orifice of a hernial sac. If only the cerebral membranes and fluids enter the sac the condition is called a meningocele, but if it also holds cerebral substance, an encephalocele is formed.

An encephalocele occurs in various degrees, that is, small and large, and can be detected when it is small only by a very careful examination. The hernial sac is composed of either the dura and arachnoid, or both of these together with the soft cranial plates, or the latter alone.



FIG. 25.—Acrania, partial anencephalus (insufficient brain), encephalocele.

The swelling is elastic, it communicates with the interior of the skull and is situated between the bones (a cephalhematoma lies directly upon the bones); its presence may finally be decided by a cautious puncture. Cerebral hernias are most frequently found in the occiput, less often

in the frontal region and at the base of the skull. Partial hernias of moderate degree are accompanied by pain and also convulsions when the swelling is pressed upon;

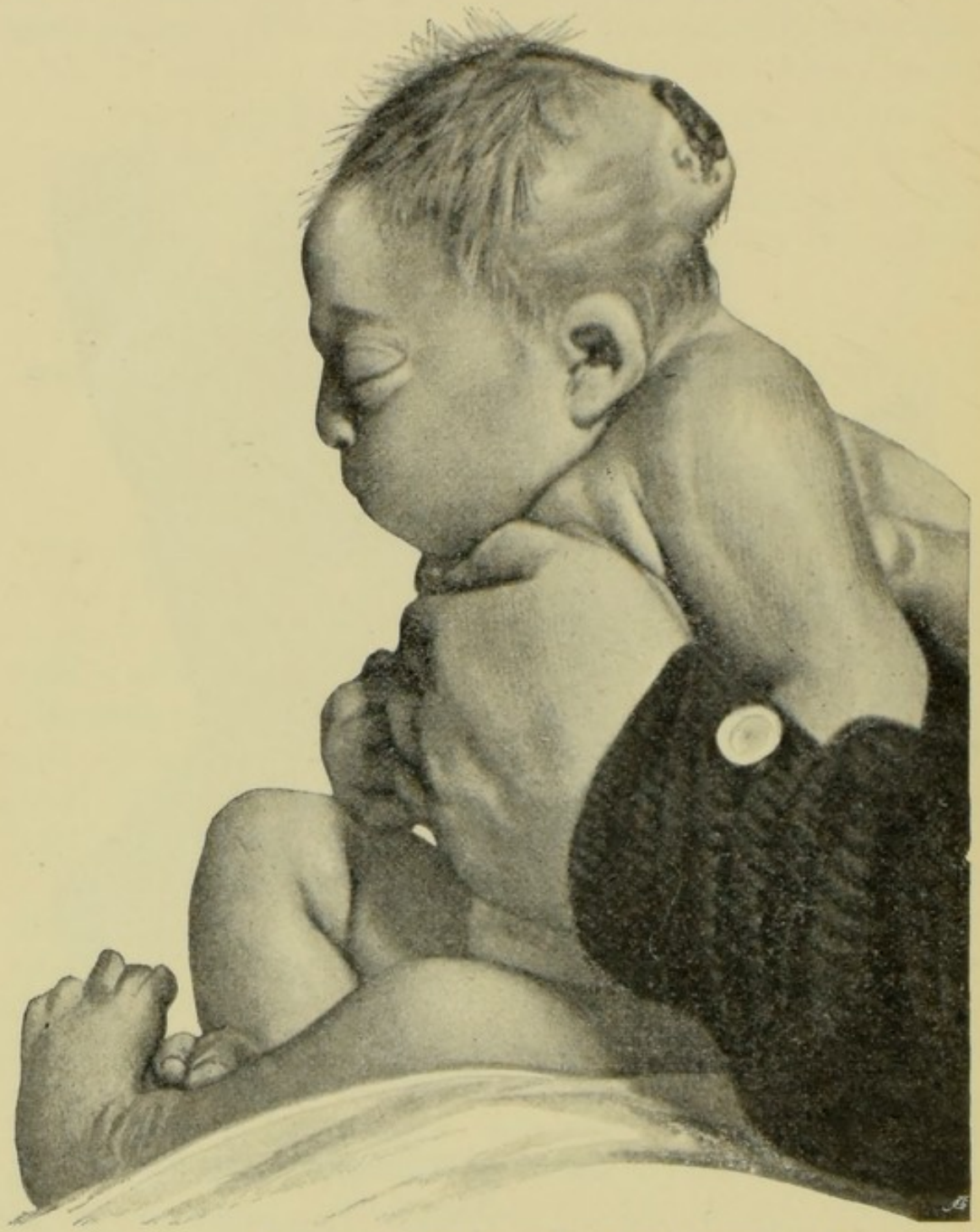


FIG. 26.—Cranioschisis and encephalocele of mild grade; double club-foot.

various nervous symptoms also occur. Characteristic of the severe cases are the bulging eyes and the whole expression of the face—toad's head. Only the very mild

cases continue to live and may heal spontaneously. The treatment consists in wearing a protecting cap or an operation. In most cases this condition is associated with other malformations, such as hare-lip, club-foot, etc.



FIG. 27.—Sacral spina bifida. (von Ranke's Clinic, Munich.)

Rachischisis is partial or complete failure in closure of the spinal canal. When the cranial cavity is also exposed the condition is called **craniorrachischisis.** Of practical importance are the partial fissure formations, which lead to a hernia of the spinal cord—**spina bifida.** Three varieties are distinguished :

Spinal Meningocele.—This hernia consists of a protruded portion of the pia, which is filled with cerebrospinal fluid.

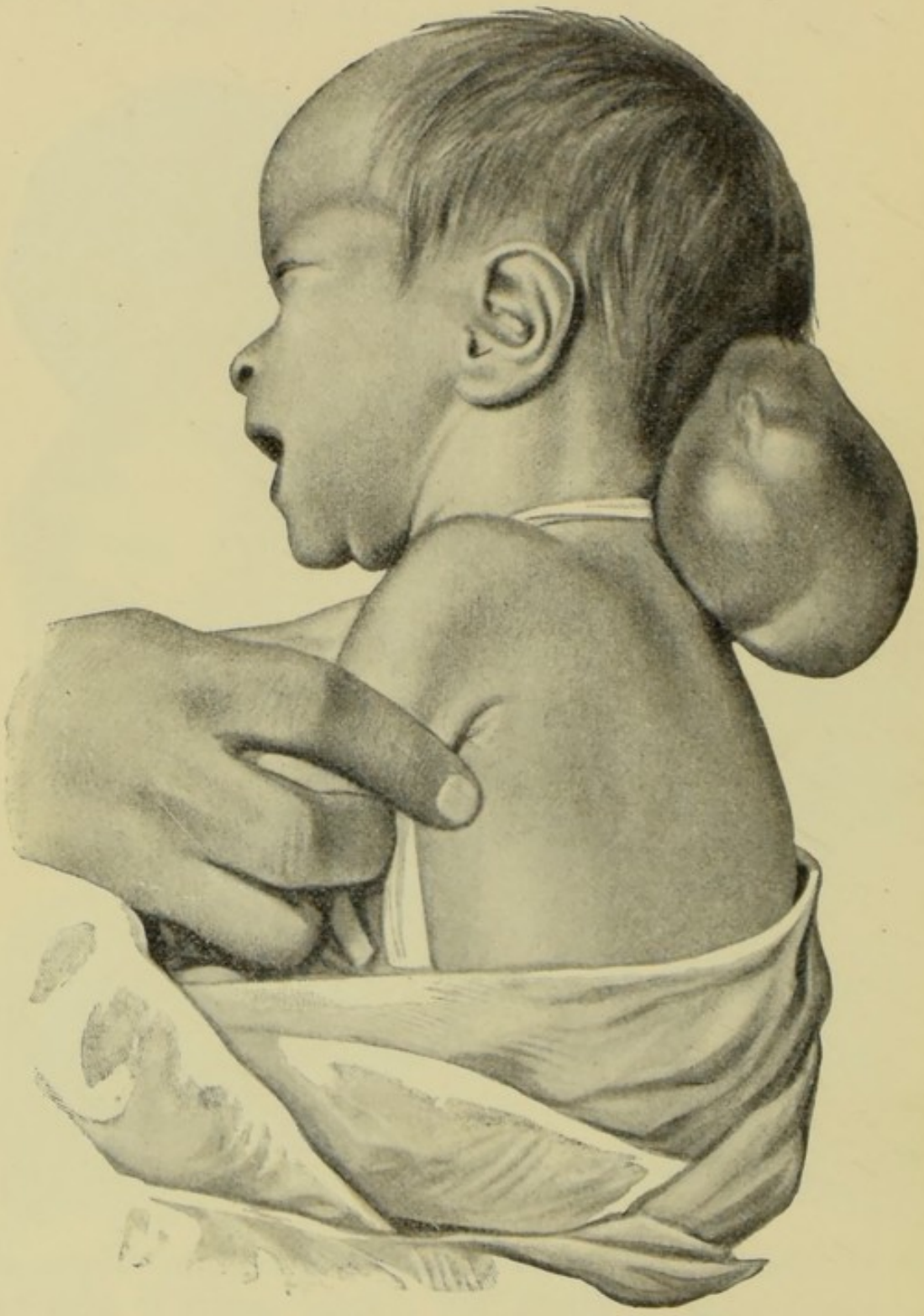


FIG. 28.—Cervical spina bifida. (Escherich's Clinic, Vienna.)

Myelomeningocele, in which the hernial sac contains also the nerve roots or a portion of the spinal cord.



FIG. 29.—Hare-lip, associated with encephalocele, left perodactylus, and club-foot. Infant five days old, which died in eight days.

Myelocystocele, which is a protrusion of the pia due to a cystic enlargement of the central canal.

Spina bifida is an elastic, fluctuating, usually elongated tumor in the lumbar, sacral, and rarely in the cervical portion of the spine. It reaches the size of a child's head and may be made smaller by pressure (stretching of the fontanels and eventually convulsions).

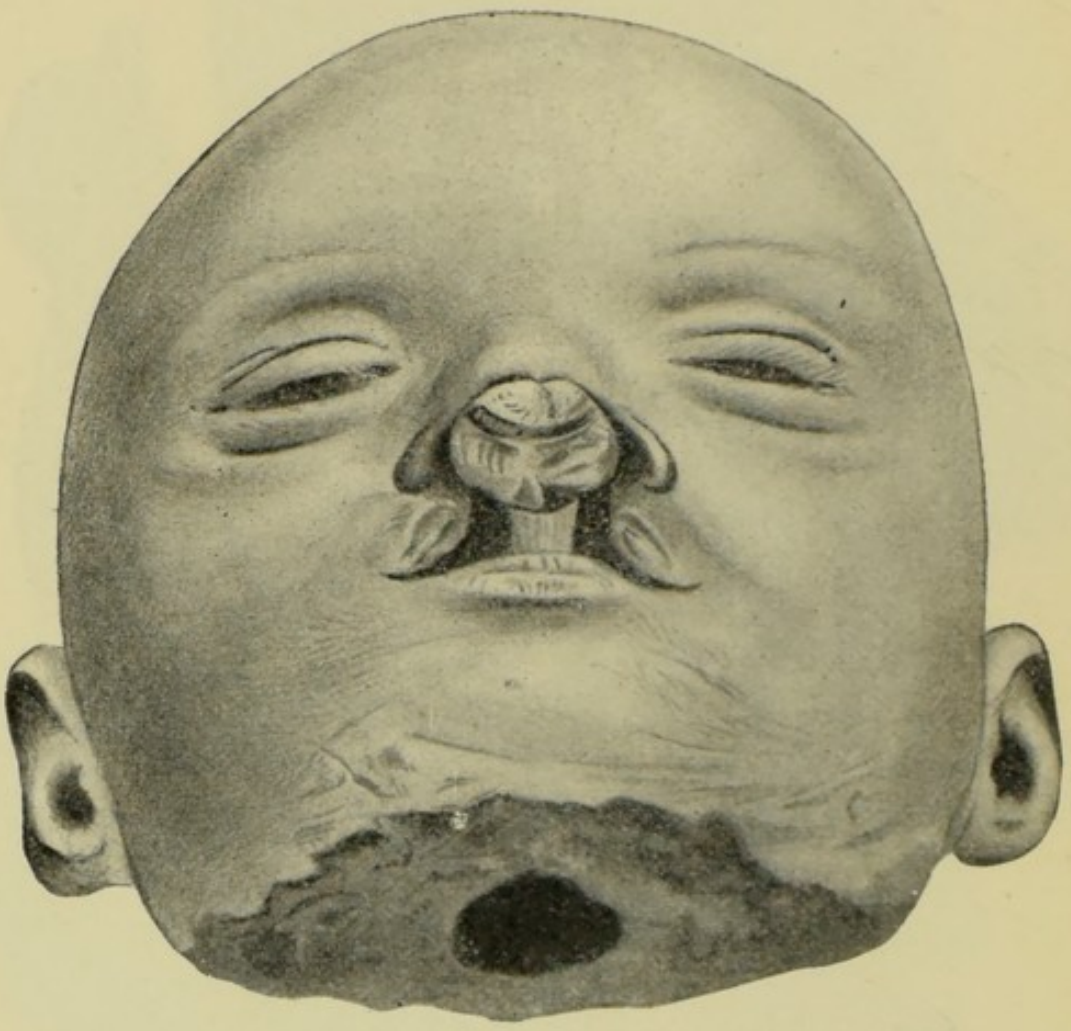


FIG. 30.—Double cleft of the lip, jaw, and palate, with a rudimentary intermaxillary bone, which is a continuation of the frontal process. Preparation of the Munich Pathologic Institute.)

The skin over the swelling is normal or thin and livid. The second and third forms are usually associated with paralytic phenomena in the region supplied by the involved spinal nerves. The small spinal bifidas may undergo spontaneous cure, but the prognosis for the larger ones, especially the second and third forms, is hopeless.

The treatment involves the use of protective appliances, puncture, or operation.

Facial Defects.—The face is formed by the union of the paired processes from the visceral and branchial arches with the frontal process, which is single. Disturbances in this union lead to a more or less marked deformity of the face.

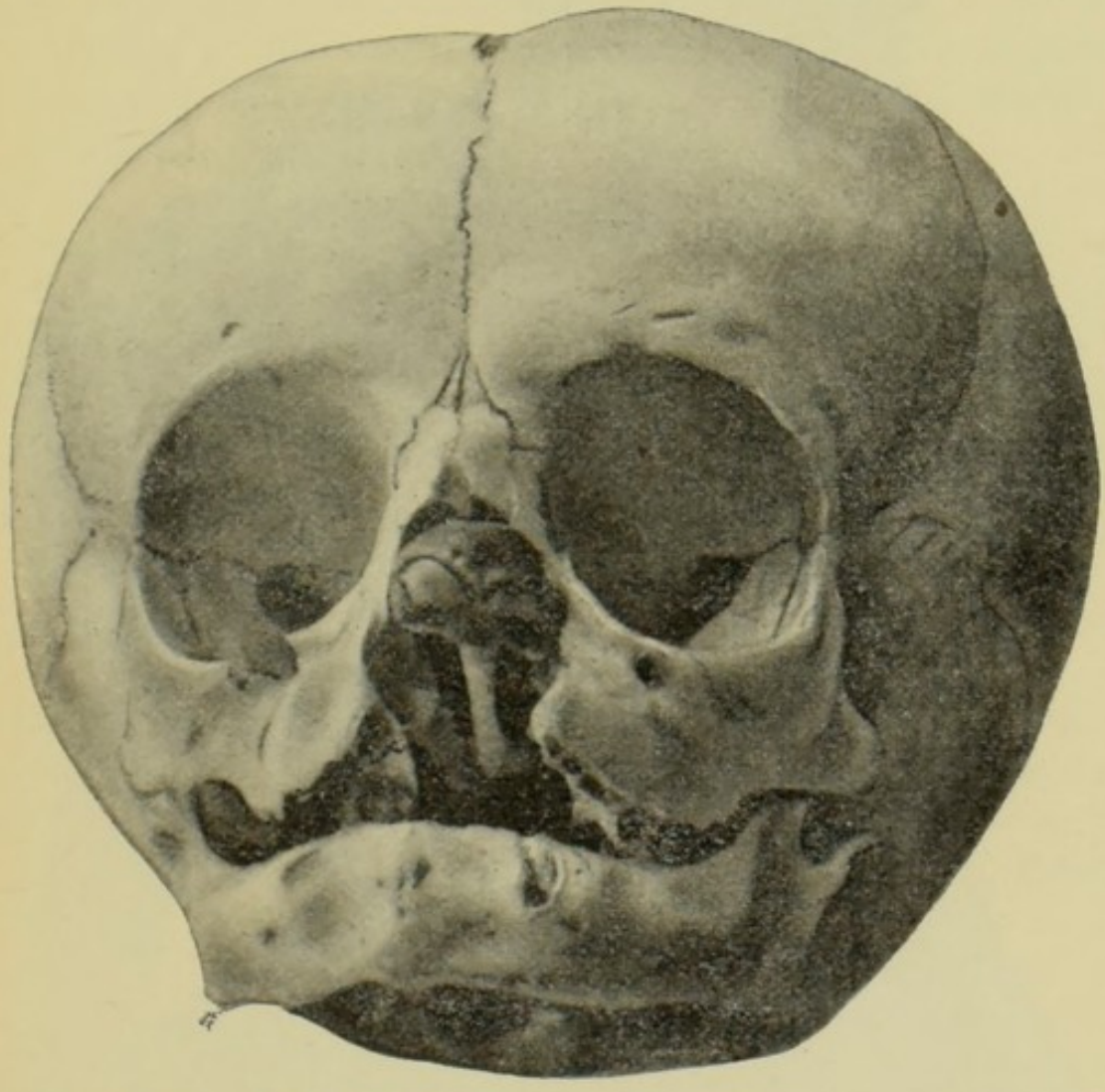


FIG. 31.—Double fissure of the jaw and the palate, with a rudimentary intermaxillary bone projecting from the frontal process. (Preparation of the Munich Pathologic Institute.)

The lowest degree of deformity is represented by an indentation or scar-like line in the upper lip or a forked division of the uvula. A stage further is the *hare-lip* (labium leporinum), a lateral fissure of the upper lip, which is frequently combined with a cleft alveolar proc-



FIG. 32.—Microcephalus, involving mainly the skull. (Escherich's Clinic, Vienna.)

ess. Next comes the *cleft palate* (palatum fissum), which consists of a fissure of the hard palate, and is usually

associated with hare-lip and occurs either on one or on both sides. At times the intermaxillary bone rests as a nodular process in the median line. These deformities result in difficulty in sucking, varying according to the depth of the cleft; in case of defects of the alveolar process and the hard palate, sucking is impossible, and feeding must be performed with a spoon while the head is held up. The treatment is exclusively operative. [Owing to the fact that infants bear operations poorly, they should be deferred until at least the third month of life.—Ed.]

Microcephalus is a condition in which a skull is abnormally small in all dimensions or only in the cranial portion. It follows premature synostosis of the cranial bones or arrest in the growth of the brain because of encephalitic and meningitic processes. It is accompanied by a flat receding forehead, pointed head, a low cranium, and protrusion of the jaws (*prognathism*). As a rule the patient is an imbecile, varying in degree from the lowest to the highest grade.

Congenital Cervical Fistula.—This follows failure in union of the second branchial arch (Strübing). The external orifice of this fistula lies between the two sternomastoid muscles near the clavicle, and ends either blindly or opens in the pharynx. It secretes a mucoid fluid. The treatment should consist in an attempt at total extirpation.

Congenital Hygroma of the Neck.—This is a serous cyst, possessing multiple compartments, which lies beneath the inferior maxilla or over the clavicle. It penetrates deeply into the connective tissue of the neck and mediastinum and may grow to a considerable size. The treatment involves extirpation or incision, followed by iodoform tampons.

Hypertrophy of the Tongue, or Macroglossia.—The tongue may be congenitally enlarged because of an overgrowth of interstitial tissue or of the muscular tissue itself. This enlargement causes the tongue to protrude (prolapsus linguæ) and to interfere with speech and the ingestion of

food. In mild cases the tongue is treated by applying alum or by painting it with a weak solution of iodin; in severe cases excise a wedge-shaped section or cauterize.

Ranula.—This is a tumor on the floor of the mouth, due to cystic degeneration of the sublingual gland or its excretory ducts. Its walls are thin and transparent; it contains liquid and grows as large as a pea or a walnut. It interferes to a certain extent with swallowing by distention, and thus elevating the tongue. The treatment consists in excision of the anterior wall and in cauterizing the stump.

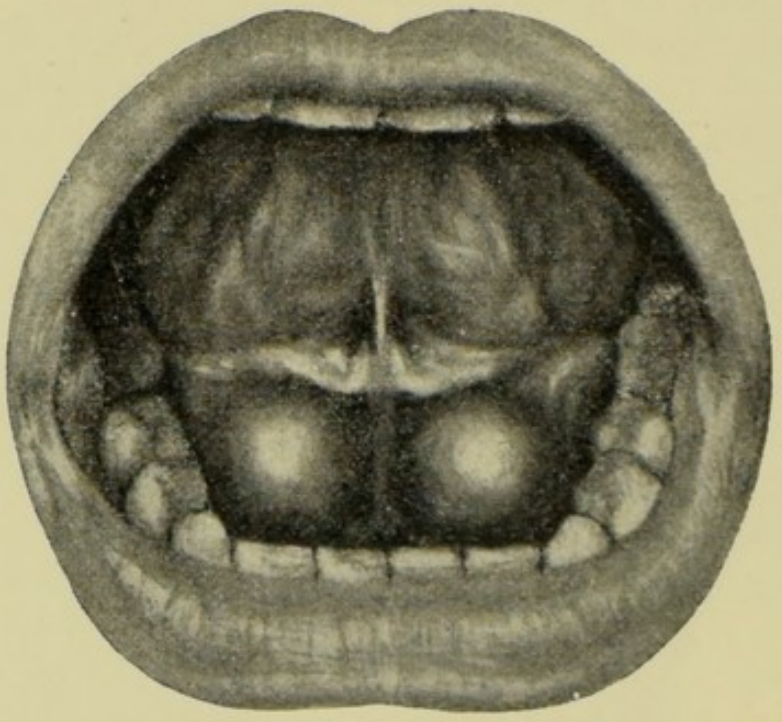


FIG. 33.—Ranula. The growth lies in the middle of the mouth, and seems to be divided into two parts by the constriction of the frenum. (From Grünwald, *Diseases of the Oral Cavity.*)

Abnormal Attachment of the Tongue.—The lingual bands may be too short or inserted too far forward (ankyloglossia), on account of which there is difficulty in sucking and speaking.

Treatment is less often indicated than is expected by the mothers and midwives. The frenum is severed while the tongue is held upward by the thumb and index-finger of the left hand, or by means of a tongue-tie. When the lower surface of the tongue is congenitally grown to

the floor of the mouth, it must be loosened either by blunt dissection or by incising, and followed by cauterization. Fissures of the sternum as well as defects of the ribs and clavicle are practically of little importance.

Hernia of the Umbilical Cord.—(See p. 81.)

Ectopia of the Bladder, Prolapsus Vesicæ or Inversio Vesicæ.—This is a defect of the anterior wall of the abdomen and bladder through which the posterior wall is seen. Soon after birth the abdominal pressure causes the posterior vesical wall to protrude as a red, shiny tumor the size of a walnut. The flow of the urine from the orifices of the ureters may be plainly seen. The urine, which has a strong odor and has undergone ammoniacal changes, causes excoriations and eczema of the surrounding skin. These children, who usually possess other malformations, as a rule, soon die, yet if the defect is a mild one the subject may continue to live. The treatment is surgical.

Meckel's Diverticulum.—The omphalomesenteric duct, which in the embryo forms a communication between the mid-gut and the umbilical vesicle, may persist as a blind pouch of the intestine lying at right angles to the wall of the lower portion of the ileum. This glove-finger-like sac possesses the same structure as the ileum and either hangs freely in the abdominal cavity (genuine diverticulum) or is fastened at its apex to the umbilicus. It forms within the umbilical ring a small prominent growth (open diverticulum, see Fig. 34), which at the time of, or after the umbilical cord has dropped off, becomes patulous, and thus leads to a fistula between the umbilicus and the intestine.

Atresia of the Anus.—Simple atresia of the anus is due to persistence of the anal membrane, which separates the blind end of the rectum (proctodeum) and the invagination of the skin from without. If the blind end of the rectum opens in another direction, into the bladder, the urethra, or vagina, there results either atresia ani vesicalis, urethralis, or vaginalis. Excepting the last form, in which under certain circumstances defecation may be per-

FIGURE 34

Meckel's Diverticulum.—The omphalomesenteric duct protrudes from the umbilical ring as a round growth (open diverticulum), and shows a small opening which developed when the umbilical cord dropped off. This opening communicates with the intestines (fistula intestini umbilicalis). There is also malformation of the right hand. (Escherich's Clinic, Vienna.)

formed, the patient can only live after the malformation has been corrected by an operation.

DEFORMITIES OF THE EXTREMITIES

The many different types of deformity or complete absence of the extremities are of more theoretic than practical interest. (For chondrodystrophia and osteogenesis imperfecta as a cause of micromyelia, see p. 132.)

The most important defect of individual bones is the congenital malformation of the radius. This may be completely absent or be defective only at one end. If the lower end is absent, it may be substituted by a vicarious enlargement of the end of the ulna. The results of this defect are: Strong radial flexion of the hand; the long axis of the hand forms with that of the forearm an acute angle; the radial border of the hand is easily placed against the radial surface of the forearm, and the hand is rotated on its long axis (club-hand). The treatment consists in an attempt to secure a useful position of the arm and hand by means of splints and fixation bandages.

Polydactylism, or Abnormal Number of Fingers.—Supernumerary fingers are attached either to one side of the hand or placed between the other fingers. If the supernumerary finger is well developed it shares either a metacarpal bone with a neighboring finger or it possesses its own, and, indeed, such a finger may have a separate carpal bone. The latter type is to be regarded as retrogression to the heptadactyle prototype of the mammalian hand. The treatment consists in removal by operation.

Syndactylism.—This consists of union between two or more fingers by means of a membranous growth. It may be total or partial; the bones are usually separated. The



FIG. 34.

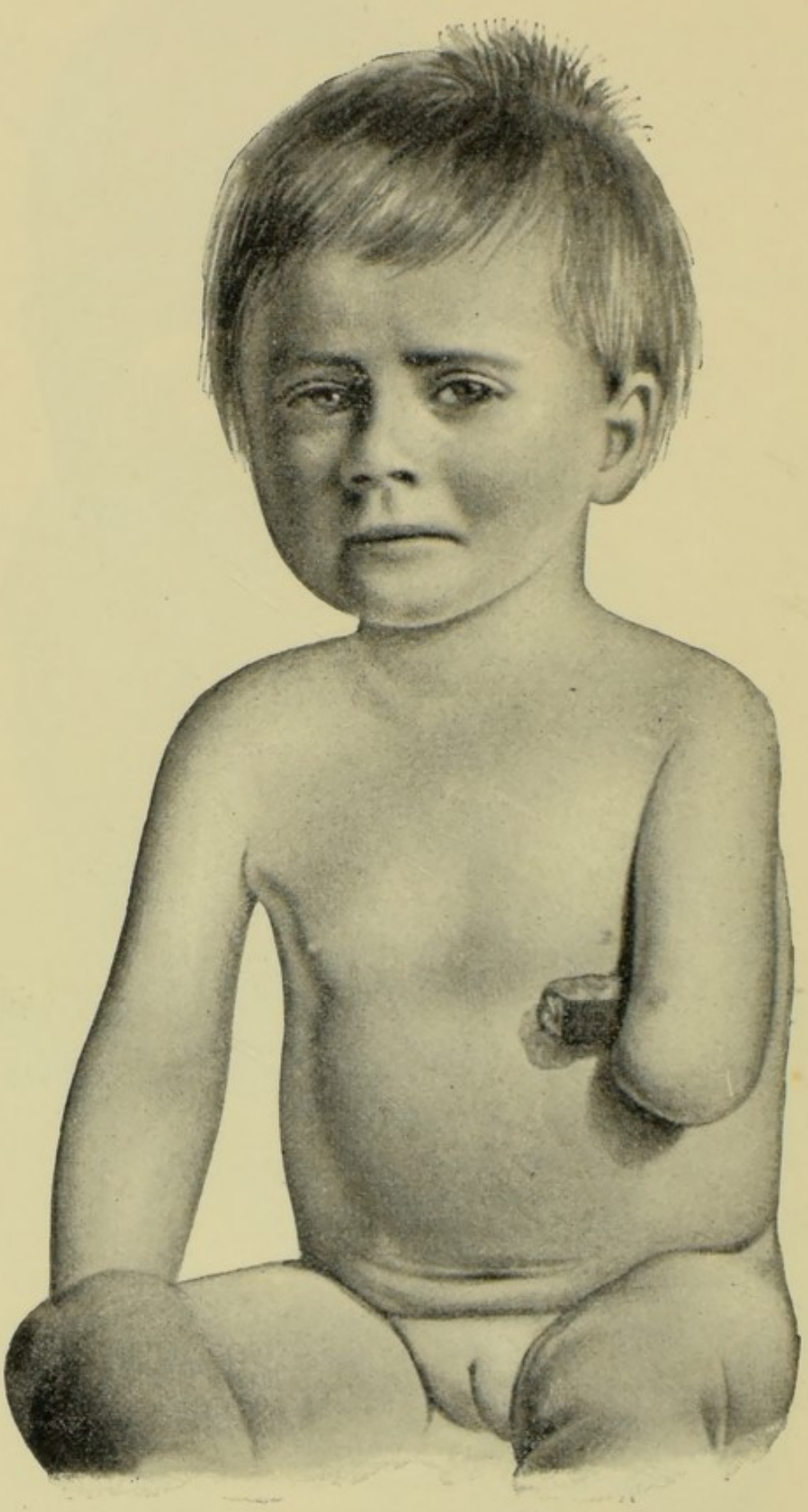


FIG. 35.—Intra-uterine amputation of the left forearm by an amniotic band. (Escherich's Clinic, Vienna.)

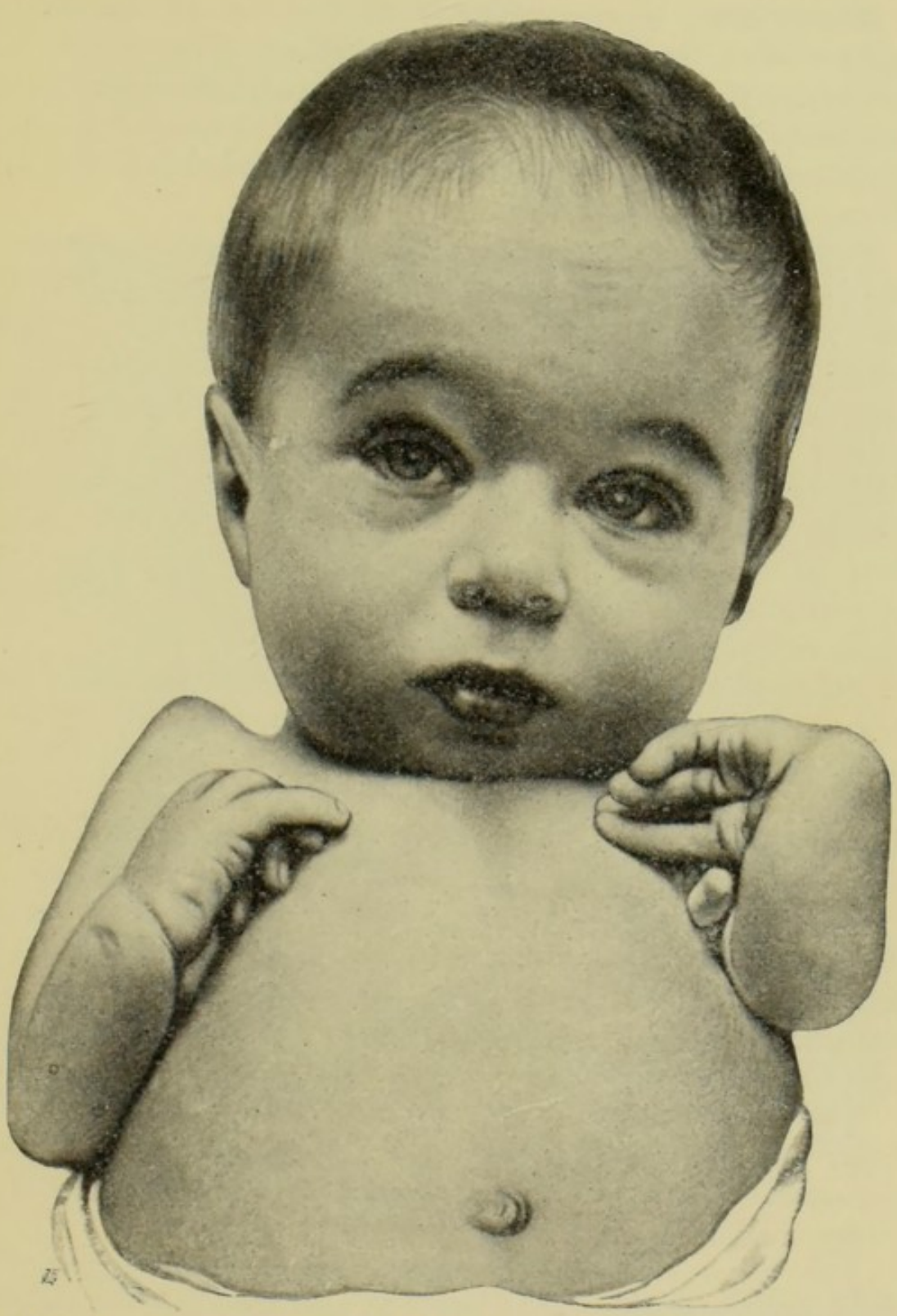


FIG. 36.—Congenital defect of the left radius. Strong radial flexion of the hand and rotation of the same on its long axis—club-hand. (Escherich's Clinic, Vienna.)

treatment is surgical and should take place during the first years of life.

Manus vara is a congenital contraction of the wrist-joint. The hand is held in a position of strong volar and ulnar flexion; when the wrist is extended the fingers are bent. It, like club-foot, is due to intra-uterine disturbance, and is distinguished from club-hand by the absence of a radial defect.

Pes varus, or congenital club-foot, depends upon an incorrect position of the foot, which is held supinated under abnormal circumstances (Bessel-Hagen). The cause is partly due to the germinal layer and partly to intra-uterine pressure and disturbance. The outer edge of the foot is depressed and the inner edge is elevated. The sole is drawn inward; and the condition is nearly always combined with excessive plantar flexion and adduction of the toes (pes equinovarus). Proper orthopedic procedures may correct this deformity and restore complete function to the part.

Pes equinus, pes valgus (flat-foot), and **pes calcaneus,** like genu valgum and varum (knock-knee and bow-leg), are, as a rule, acquired during life.

Congenital Luxation of the Hip-joint.—According to Lorenz this is the most frequent congenital deformity. Three grades of luxation are distinguished (F. Lange):

Supracotyloid luxation, in which the head rests above the socket, and can be felt anteriorly in the groin on extending and flexing the leg. It cannot be felt in the external iliac fossa by the Malgaigne method (adduction and flexion).

The *supracotyloid and iliac luxation,* in which the head of the femur, when the leg is extended, is felt anteriorly in the groin, and when flexed, posteriorly in the external iliac fossa.

The *iliac luxation,* in which the malposition of the head is permanent. The latter is palpable in the external iliac fossa, both on extension and flexion of the leg. Distinct symptoms usually show themselves first when the child begins to walk. A waddling gait is caused by the shortening of the leg, the loose capsular ligaments, and the insufficient stretching of the gluteal muscles.

The pelvis sinks downward and scoliosis develops. In case of double luxation the child develops a duck-like, waddling gait, pronounced lumbar lordosis, and protrusion of the abdomen. Since these phenomena are also possible in rachitic deformity of the neck of the femur (coxa vara),

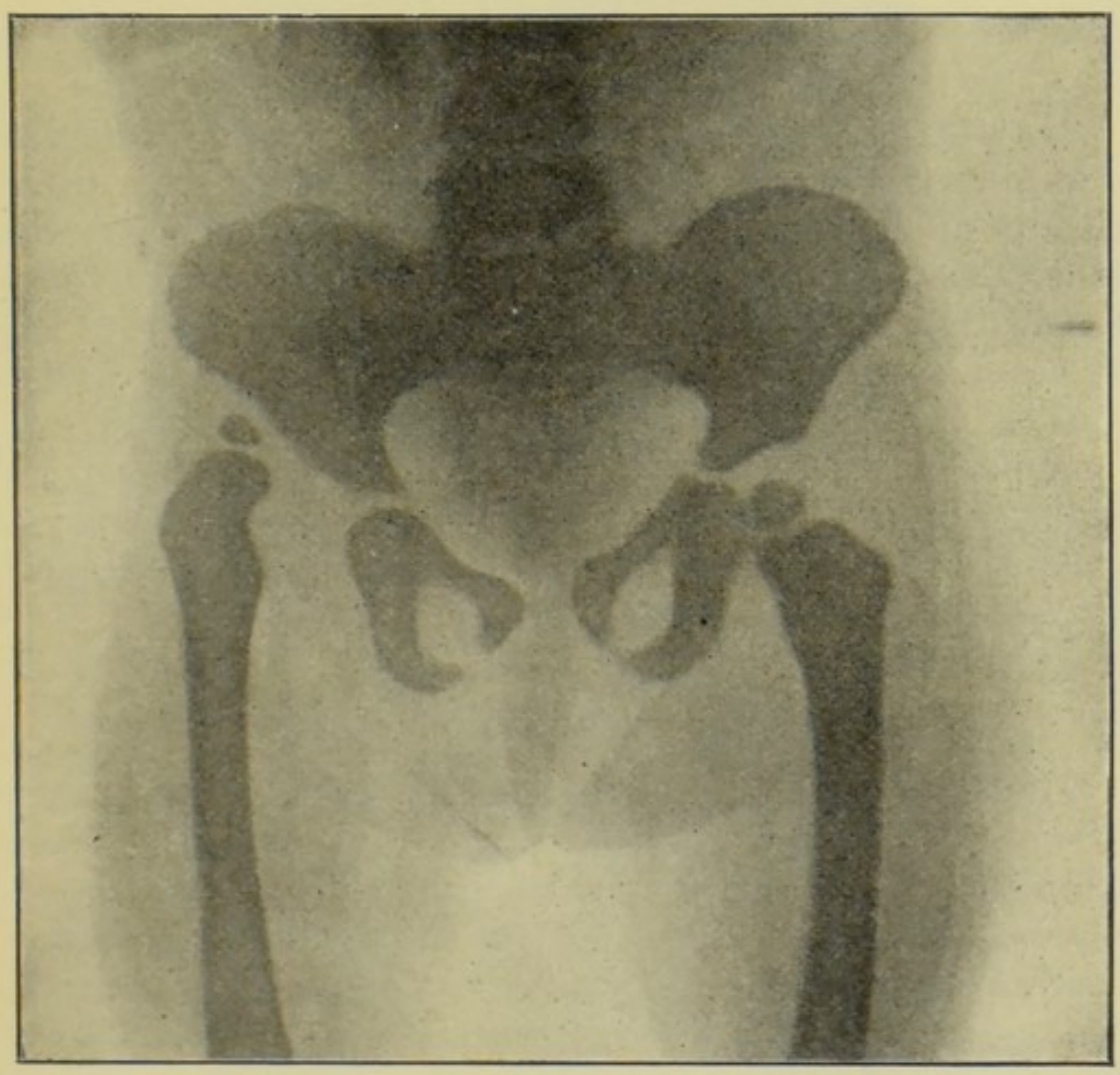


FIG. 37.—Congenital supracotyloid luxation of the left femur. Radiogram, taken in dorsal posture, of girl two and a half years old. The head of the femur lies at the upper border of the socket. (From Lüning and Schulthess, *Atlas of Orthopedic Surgery.*)

the diagnosis should be made in every case by means of a Röntgen photograph. Orthopedic treatment (girdle, bloodless reduction) offers permanent cure, provided it is instituted before the fifth year of life.

CONSTITUTIONAL DISEASES

RACHITIS

RACHITIS is a condition characterized by an insufficient deposit of calcium salts in the bony tissue. As direct results of this insufficiency of lime are an abnormal degree of softness, abnormal hypertrophy, and a lack of longitudinal growth of the bones, while the weight of the skeleton as well as the tension of the muscles cause a large variety of deformities of the soft and flexible bones.

Rachitis occurs usually between the ages of one and two years, yet it may develop in very young infants or even at birth. As a rule rachitis of the skull appears during the earlier periods of life.

SYMPTOMS

The Skull.—The large fontanel is more patulous than it should be at that age, and remains open until the second or third year (normally it closes in from the twelfth to the fourteenth month). Its edges are soft, and being thickened they rise above the surface of the scalp. The small fontanel, which should be closed at birth, is still open and its edges are likewise soft. The portions of the temporal and occipital bones adjacent to it show isolated and softened thin areas called *craniotabes*,[1] which give a parchment-like crepitus on pressure. The sutures gape. Swelling of the frontal and occipital tuberosities give the skull a square appearance—*caput quadratum*. As a result the whole head seems enlarged and may sometimes be mistaken for hydrocephalus.

[1] The examination for craniotabes is performed with the third and fourth fingers of both hands, which are placed flat upon the sides of the skull.

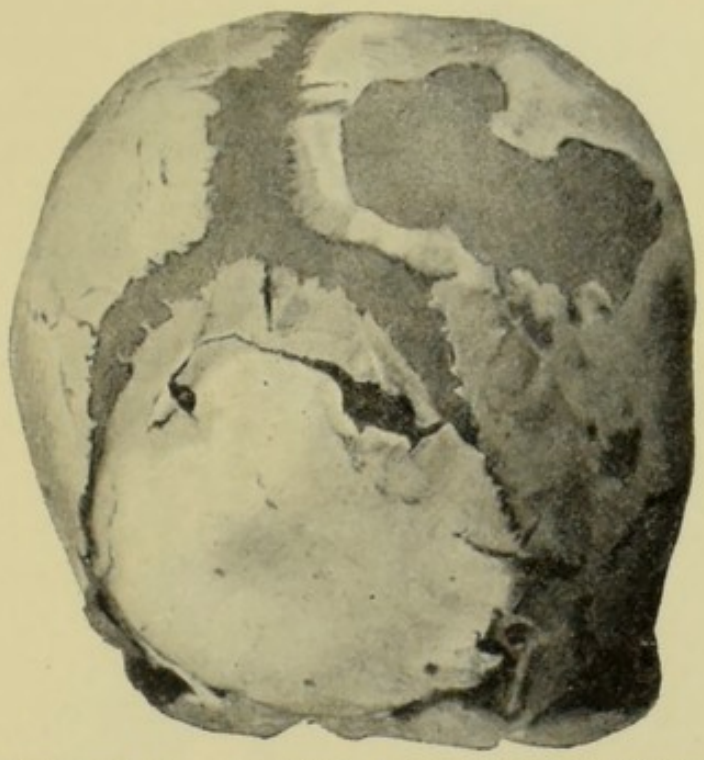


FIG. 38.—Craniotabes. Rachitic decalcification of the right paretal bone; gaping sagittal and lambdoidal sutures. One-year-old child. (Preparation in the Pathologic Institute of Munich.)

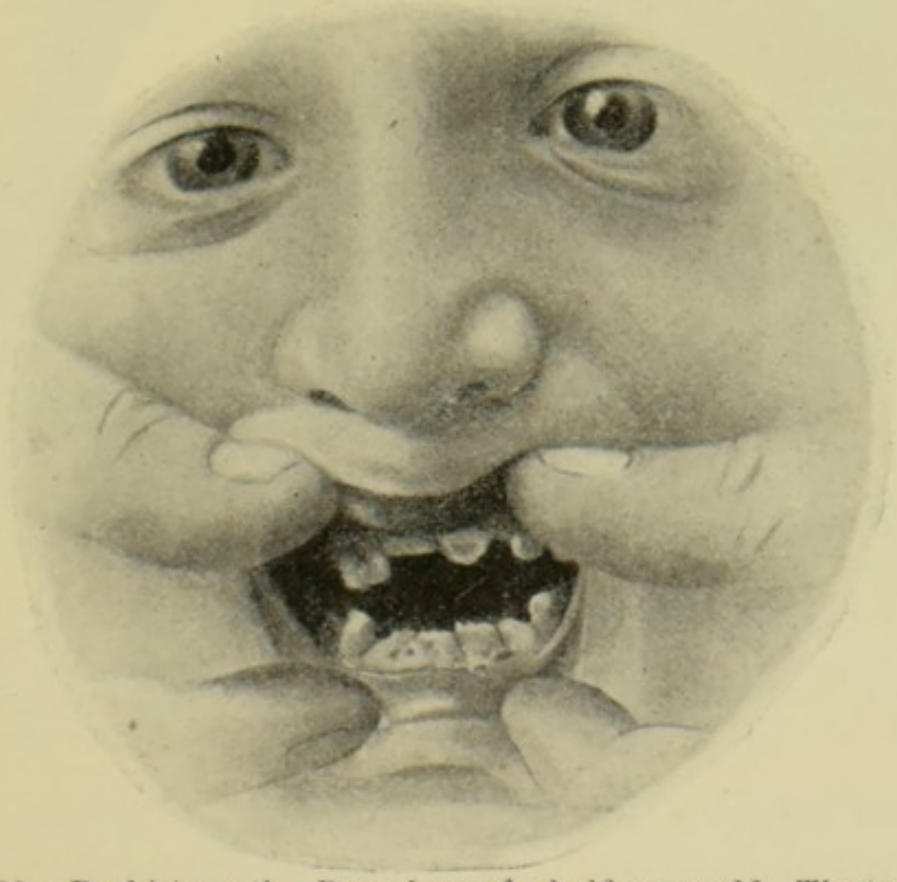


FIG. 39.—Rachitic teeth. Boy nine and a half years old. The teeth are poorly developed, considerably eroded and grooved. Their position is very irregular; the lower incisors occupy a frontal position (not in the arch of the jaw) and the inferior maxilla makes an angular turn at the canine teeth.

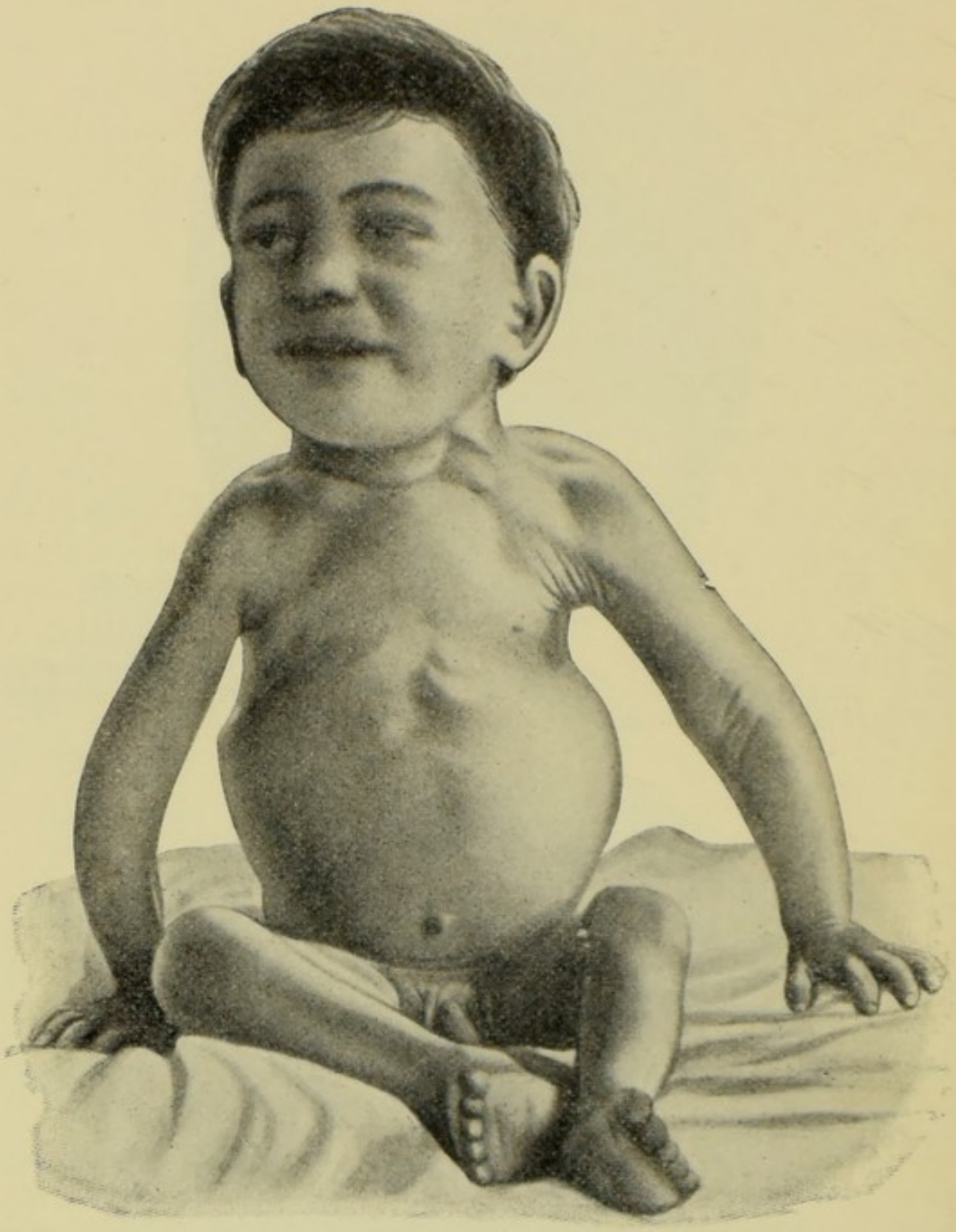


FIG. 40.—Rachitic boy of three years. A large and somewhat angular head. The typic posture of a rachitic child, with the arms supported at his side. Curvature of the clavicles and the spine (see Fig. 41) causes the neck to appear short. Contraction of the lateral diameter of the thorax; abdomen protrudes; curvature of the bones of the forearm.

The superior maxilla is lengthened in the sagittal diameter, on account of which its middle portion is more prominent. The inferior maxilla makes an angular turn in the neighborhood of the second incisor teeth, and

therefore appears to be flattened in front. The teeth, whose eruption is delayed and occurs at irregular intervals, are considerably displaced on account of deformity of the alveoli in which they are inserted. Their occluding surfaces do not coincide, the canine teeth meeting in a sagittal line and the lower incisors in a frontal line.

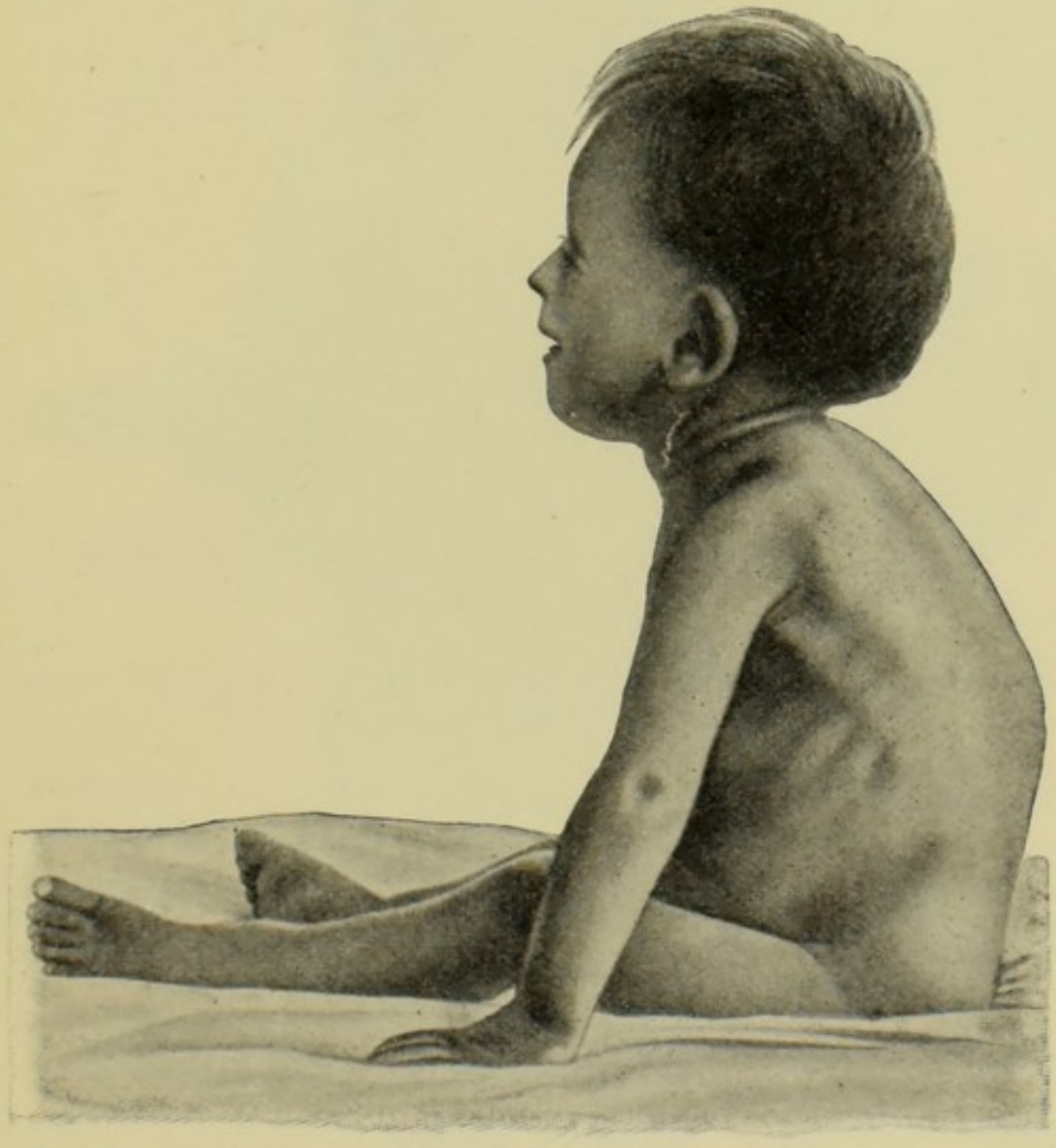


FIG. 41.—Rachitic boy (lateral view of Fig. 40). Rachitic rounded kyphosis (perfectly compensated when lying upon the abdomen); swelling of the epiphyses of the bones of the forearms and at the junction of the cartilaginous with the bony portions of the ribs.

The surface of the teeth is usually prematurely discolored a dirty yellow, and presents fossæ in close proximity to the gums. Later they are striped, grooved, notched, undergo decay, and crumble away.

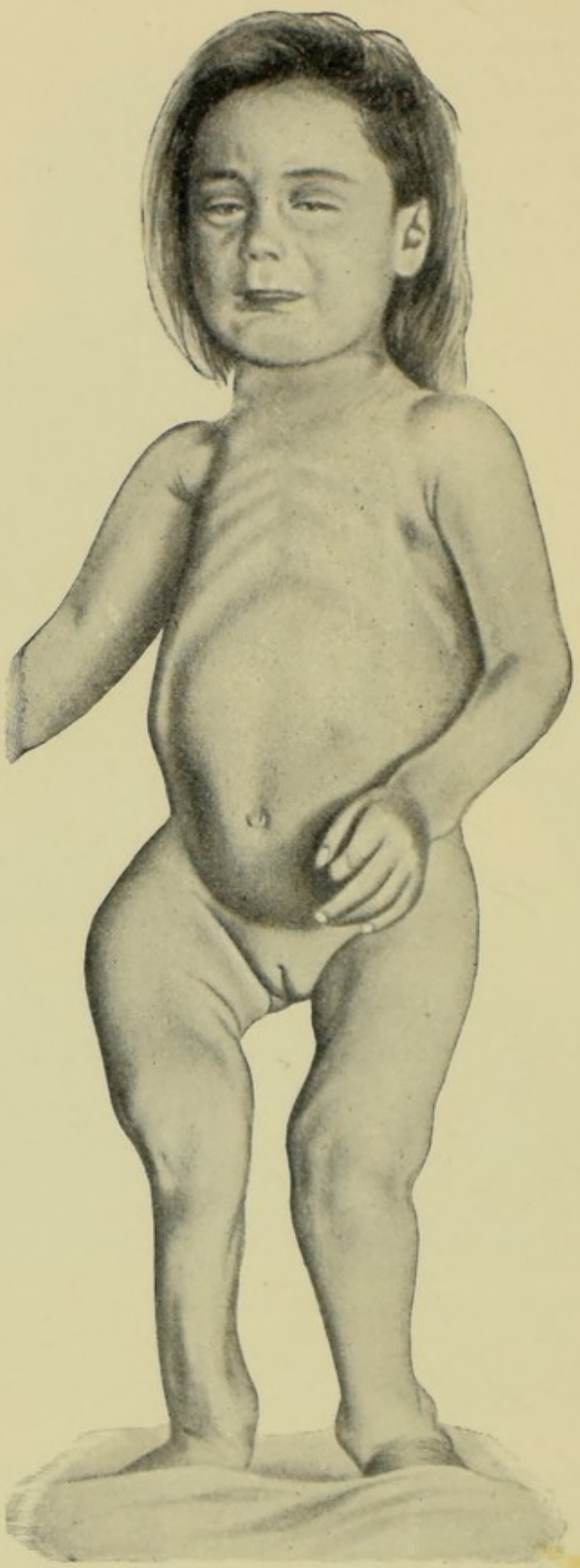


FIG. 42.—Extinct rachitis. A six-year-old girl, showing decided curvatures of the bones, which have now become hard. (Escherich's Clinic, Vienna.)

Thorax.—Either all or only individual bones are enlarged at their cartilaginous extremities, and in some cases plainly knobbed—"rachitic rosary."

The weight of the arms, the traction of the diaphragm, and the atmospheric pressure cause flattening and retraction of the lateral thoracic walls, a diminution of the transverse diameter, and an increase of the sagittal diameter (rachitic chicken-breast). The lower portion of the thorax over the liver, spleen, and stomach bulges outward.

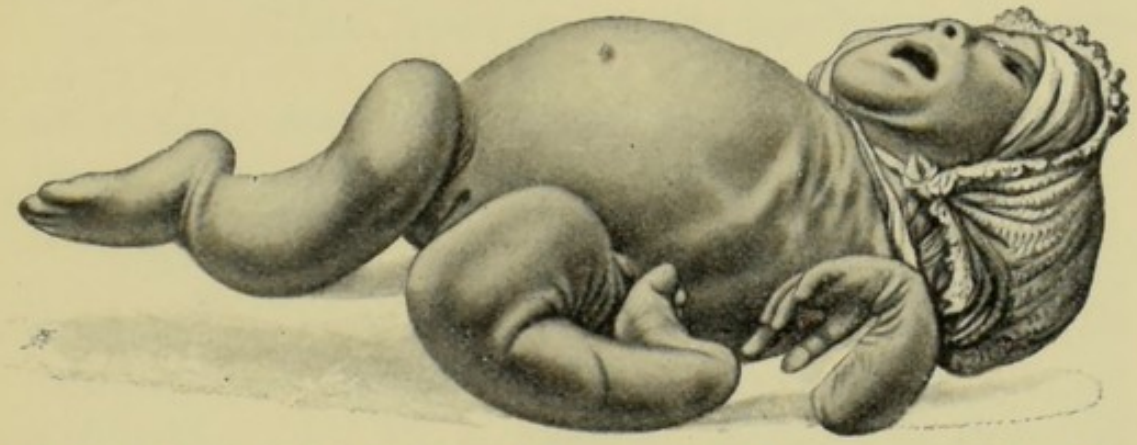


FIG. 43.—Severe rachitis; osteomalacic form with enormous deformities of all extremities. (Escherich's Clinic, Vienna.)

Vertebræ.—Posterior curvature of the lower portion of the thoracic spine is one of the most frequent phenomena. This rachitic form of kyphosis, contrary to the type met with in spondylitis (Pott's disease), disappears when the subject lies on its stomach (provided ossification has not set in) and is not painful. A vicarious lumbar lordosis is frequently present. The other forms of scolioses do not develop, as a rule, until later in life, and from other causes.

Pelvis.—The weight of the body presses the iliac bones outward, the sacrum and promontory forward, and elevates the os pubis. As a consequence the true conjugate diameter is shortened and the transverse diameter is increased.

Extremities.—The changes in the extremities, as a rule, occur later in the disease than those of the skull. They consist of a nodular swelling of the lower epiphyses of

the radius, ulna, tibia and fibula, and, more rarely, the humerus and femur. Furthermore, traction of the muscles and their weight cause curvatures or even greenstick fractures of the shafts of these bones ; as a rule the radius and ulna are bent convexly on their extensor surfaces, the humerus forward and the tibia and fibula outward. The frequent deformities of the joints, like genu valgum and genu varum, have their origin in the rachitic thickening of the epiphyses, the abnormal relaxation of the ligaments, the traction of the muscles, and the weight of the body.

These various phenomena are not well marked in every case and may frequently occur singly.

DIRECT RESULTS OF DISEASE OF THE SKELETON

In craniotabes, rubbing of the head upon the pillow causes profuse sweating of the head ; the cranium is extremely sensitive to palpation. Disease of the teeth interferes with mastication and salivation, thus at times contributing to the gastro-intestinal disturbances so frequently met with in rachitis. Disease of the ribs is manifested by pain when the child is lifted and by a decrease in the thoracic volume. The walls of the thorax being yielding permit the muscles of the chest to expand it sufficiently, excepting in certain parts where the intrathoracic space is diminished. An inspiratory retraction occurs in the neighborhood of the insertion of the diaphragm. Disease of the vertebral column and of the extremities make it impossible for the child to sit up, to stand, or to walk at the proper period of life. Frequently the subjects forget what they have already learned as regards walking or standing. The continuous insufficient ventilation of the lungs results in a decided predisposition to bronchial catarrh and bronchopneumonia.

PHENOMENA WHICH ARE NOT DIRECTLY DUE TO DISEASE OF THE SKELETON

Characteristic of rachitis is its pronounced tendency to disturbances of digestion. The dyspepsia is manifested

either in constipation or by the discharge of foul-smelling alcoholic stools. Meteorism, which is nearly always present, causes the characteristic enlargement of the abdomen, forces the diaphragm upward, and helps to diminish the intrathoracic space. Anemia is only rarely absent. In some cases the number of red blood-corpuscles is reduced to two or three millions and the leukocytes are increased. The skin as well as the subcutaneous tissue and the musculature undergo atrophy, and become relaxed and flabby in the course of time. Enlargement of the spleen is a frequent symptom, but not constantly present; enlargement of the liver occurs even less frequently. In a large number of cases there is a certain excitability of the nervous system which shows itself, as a rule, by restlessness, peevishness, and crying, and in some cases by more serious phenomena, such as laryngospasm, spasm of the glottis, tetany with its latent and manifest symptoms, and attacks of eclampsia. (Concerning these symptoms, refer to their respective sections.)

The course of rachitis is prolonged and extends, with intervals of improvement and exacerbation, over months and even years. Although the various phenomena frequently arise within a few weeks, their disappearance is gradual, inasmuch as a normal growth of bone must first set in. The large amount of osteoid tissue which is present becomes impregnated with lime salts, thus developing unusually hard and compact bones—"ossa eburnea." The primarily soft state of the bones is thus followed, after a certain period of time, by an abnormal firmness. A large number of the deformities, even the pronounced ones, are corrected without artificial aid by the traction of the muscles, provided they have not previously undergone ossification. The well-established deformities, however, such as chicken-breast, pronounced bow-legs, thickenings of the cranium, and changes in the pelvis, remain unaffected throughout life. If craniotabes only is present it may heal without the appearance of other symptoms.

The prognosis of the disease itself is favorable. The association, however, of rachitis with any other disease is

a serious complication, as is especially true of pneumonia and intestinal diseases, which are frequently followed by death.

ETIOLOGY

We are still in the dark as to the real cause of rachitis. Although the infectiousness of this disease has been frequently referred to, yet it has never been proved; hereditary influence, however, seems to play a very important part. In favor of this claim is the existence of rachitic and non-rachitic families living under similar unfavorable hygienic conditions (Siegert). Etiologically, two factors which influence the general health are of prime importance, namely: 1. Long-continued living in foul air (small, dark rooms, overcrowded rooms, damp floors, or cellars). 2. Improper feeding, in which the special form of nourishment is of less importance than the individual digestive ability of the child. Artificially fed children are more frequently diseased than breast-fed infants; the latter are, however, by no means immune to rachitis.

In order to understand the morbid anatomy of rachitis it is of importance to understand the processes concerned in

NORMAL OSSIFICATION

We distinguish between bones preformed in cartilage (primary) and bones developed from a connective-tissue (secondary) foundation. Each type presents a different form of development:

Bones Preformed in Cartilage.—This type presents three main processes of bony development:

Absorption of the cartilage and its substitution by bony tissue—*endochondral ossification.*

Deposition of newly formed bony tissue at the periphery—*periosteal ossification.*

The reabsorption of fully developed bone, which is of importance in the building and nourishing of bone.

This constant bony growth from without and destruction from within occurs alike in all bones, and causes a

decidedly active local metabolic process during the period of development. Endochondral ossification is only seen at the epiphyses of the long bones where longitudinal growth occurs, whereas the diaphysis, which is likewise originally preformed in cartilage, is already ossified (endochondral), and continues to grow only in width through periosteal ossification.

The transit of cartilage into bone is performed within two narrow zones: 1. An upper pale blue and slightly swollen layer, the zone of cartilage proliferation. 2. Below it a thinner whitish layer, the zone of provisional calcification. Endochondral ossification occurs as follows: In the upper zone the cartilage cells become swollen and arrange themselves in rows and columns. Next the soft cartilaginous ground substance begins to harden by impregnation with calcium salts, the cartilage cells become enclosed, and their further growth is arrested. Blood-vessels accompanied by a large number of cells (osteoblasts) now extend from the medullary space of the diaphysis[1] into the cartilage prepared as above, which absorb the calcified cartilaginous ground substance, and causing the disappearance of the cartilage cells, build primary medullary spaces. Each column of cartilage cells represents a medullary space; the latter are separated from each other by undisturbed processes of calcified cartilaginous ground substance. The osteoblasts now settle everywhere on the walls of these trabeculæ and begin the development of the bony ground substance, which is free of cells—the osteoid tissue. The osteoblasts become gradually surrounded by this tissue, cease to build bone, and become permanent bone-cells. Simultaneously the osteoid tissue takes up the lime salts and is converted into completed bone-tissue. While the primary medullary spaces become filled in this manner with bone-tissue, a simultaneous absorption of the newly built bone again is brought about by the activity of a certain group of large cells—the osteoclasts. Thus are formed the definite medullary spaces of the spongiosa. Isolated remnants

[1] Or from the tissue lying beneath the perichondrium.

PLATE 6

FIG. 1. **Bony Development.**—Portion of a longitudinal section through the metacarpal bone of a five-months'-old embryo. Magnified 50 times. The figure shows the border of the endochondral ossification zone and the changes the cartilage passes through before it is absorbed. Externally at the perichondrium is a layer of perichondral bone. 1. Endochondral bone. 2. Cartilage. 3. Zone in which cartilage cells form columns. 4. Zone of enlarged cartilage cavities with temporary calcification of the ground substance. 5. Rudiments of calcified ground substance. 6. Marrow. 7. Periosteal bone. 8. Giantcells (osteoclasts). (From Sobotta's *Atlas of Histology.*)

FIG. 2. **Normal border between bone and cartilage** of the upper epiphysis of the femur of a seven-months'-old fetus (premature birth due to trauma to mother). Magnified 6 times. The border between the bone and cartilage runs as a slightly curved but regular and continuous line; the individual layers are easily distinguished from one another. 1. Unchanged cartilage. 2. Zone of growing cartilage. 3. Zone of temporary excretion of lime. 4. Zone in which medullary spaces are constructed. 5. Endochondral bone. 6. Periosteal bone. 7. Marrow.

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of calcified cartilaginous ground substance are always found in endochondral bone.

Periosteal ossification (apposition of bone), which occurs primarily only at the diaphysis, but later at the epiphysis also, is formed as follows: The osteoid tissue situated beneath the perichondrium is converted into bone substance or osteoid tissue. The penetration of blood-vessels and osteoblasts into the bone substance leads to the formation of Haversian canals, trabeculæ, and lamellæ; here also, extending toward the spongiosa, an absorption of the bone occurs through osteoclasts.

Connective-tissue Bone.—Single bundles of connective tissue become calcified; osteoblasts which are derived from embryonal cells are deposited upon these calcified bundles, and form bone in the same manner as above.

PATHOLOGIC ANATOMY OF RACHITIS

Macroscopic Changes.—A fresh rachitic bone is flexible, frequently cylindric in shape, and of lessened consistency; its periosteum is thickened and hyperemic. Such a bone presents the thickening, swelling, softening, and partial fractures which were described above. The amount of calcium is reduced from 30 to 50 per cent. On section

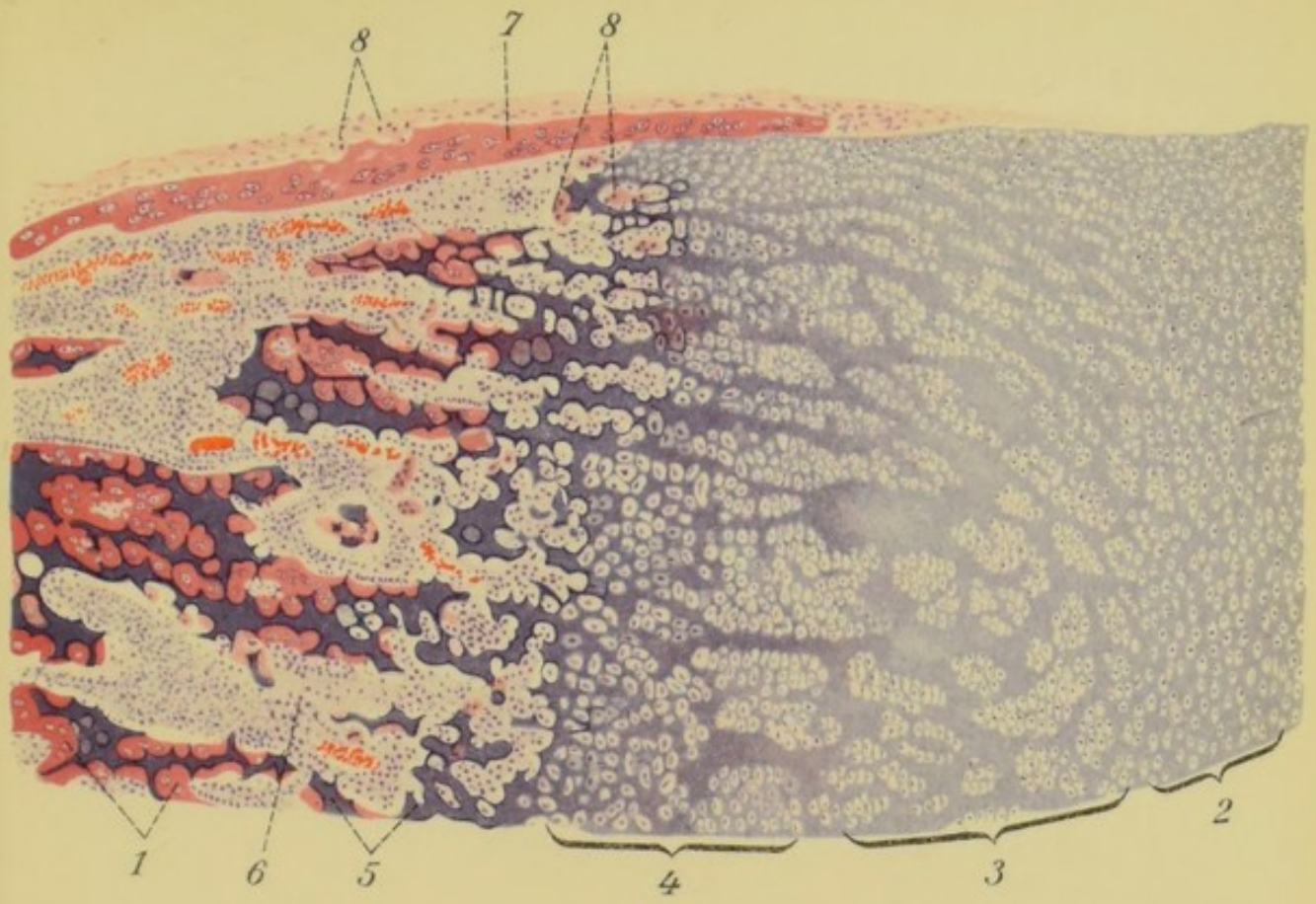


Fig. 1.



Fig. 2.

the epiphysis of a long bone is found to be enlarged in all directions, the walls of the diaphysis thickened, the wall of the medullary canal narrowed, and the spongiosa and marrow red and congested. At the junction between the bone and cartilage the following conditions are observed:

The cartilage seems to be thickened externally and driven forward.

The zone of cartilaginous growth is darker, swollen, and plainly increased in width.

The limiting zone between the bone and cartilage, which is normally straight, is irregular or indented.

Microscopic Changes.—*Endochondral Bone.*—Normally the cartilage, the zone of calcification, and spongiosa are arranged in rows one after the other, but in rachitis this order is disturbed. The border-line of the process of calcification is interrupted and the tissues are irregularly placed and are intermingled with each other. Thus we find in growing—and even in the quiescent—cartilage areas of calcified ground substance, and, indeed, osteoid tissue foci in which medullary spaces are being formed. On the other hand, separated foci of calcified and decalcified cartilage are seen in the spongiosa. There is temporary insufficient excretion of the lime salts. In the spongiosa the osteoid tissue—that is, the bony substance which is still decalcified—is thickened; an insufficient calcification of bony tissue occurs in this area.

Periosteal and Connective-tissue Bone (see Plate 8, Fig. 2).—Growth of the inner periosteal layer; broadening of the osteoid substance and deficient excretion of calcium salts in the same; in consequence there is but a small amount of real bony tissue. The bony trabeculæ become calcified within, but remain soft at the periphery, which accounts for the flexibility of the bones. The reddened bone-marrow shows an increase of red blood-cells.

The explanation of the rachitic process which is most generally accepted at the present time is that of Pauer: The process of ossification pauses as it reaches the stage when normally lime salts are being excreted. On the

PLATE 7

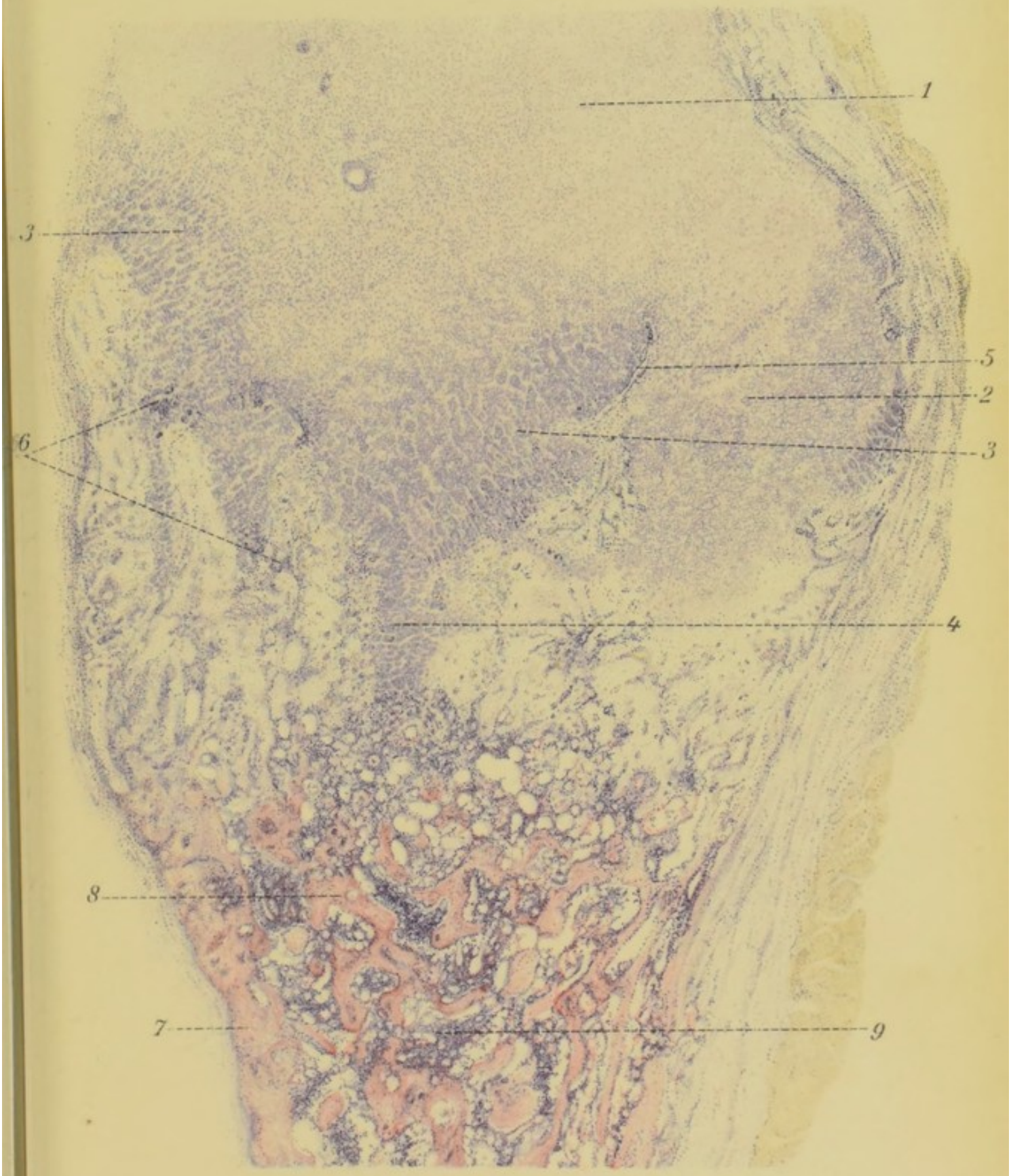
Rachitis at the Junction of the Bony with the Cartilaginous portion of a Rib. Enlarged 8 times. The bony cartilaginous junction is irregularly formed; the zone of provisional calcification is absent; the cartilage has undergone excessive proliferation; the formation of medullary spaces and cartilaginous proliferation occurs in the same plane side by side. 1. Quiescent cartilage. 2. Proliferated cartilage which has led to lateral protrusion. 3. Columns of cartilage cells. 4. Columns of cartilage cells which have penetrated the zone of medullary spaces. 5. Primary marrow which has penetrated from the medullary spaces deeply into the cartilage. 6. Indication of provisional excretion of lime (dark color). 7. Osteoid tissue of periosteal origin, which is thickened and poorly supplied with lime. 8. Thickened osteoid tissue which is endochondral in origin. 9. Marrow.

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one hand, the temporary calcification of the cartilaginous ground substance is insufficient, while on the other hand, there is imperfect transformation of the osteoid substance (containing no lime salts) into bony tissue, which, although similar in structure, contains calcium salts. The results, therefore, are of two kinds:

If the boundary line between cartilage and bone is absent, together with the zone of calcification, the regular and necessary arrangement, in which the column of cartilage cells and a primary medullary space face each other, is not preserved, so the absorption of the cartilage cells does not proceed at equal stages. Without a limiting line the two tissues grow past each other without any order. Thus are explained the serrated edges and intermingling of the layers, the presence of primary medullary spaces in the cartilage, and of cartilaginous foci in the bone, etc. Such a process naturally arrests the growth of the bone in its longitudinal axis.

If in spite of the insufficient further growth of these two tissues, the growth of the cartilage, the formation of large vesicular properly arranged cartilage cells, as well as the deposition of osteoid tissue, proceeds uninterrupted, excessive tissue forms on both sides, which hinders the transformation into the next stage. This excessive amount of unfinished tissue spreads out, and thus leads to the characteristic cartilage and bony swellings. The latter are not, therefore, due to an active proliferation, but to arrested development of the bone-building tissues.


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In the case of periosteal and connective-tissue bone practically the same processes are concerned, that is, unlimited deposition of osteogenetic layers upon the surface of the bone and deposition of osteoid tissue within, with faulty or imperfect conversion into fully developed bone. Such is the origin of the enlarged cranial protuberances; the craniotabes is explained by either an excessive resorption of the newly formed bone or by a lack of bony apposition with undisturbed resorption. Insufficient excretion of calcium salts is undoubtedly accompanied (in certain cases of rachitis) by an osteoporosis, that is, excessive resorption of fully developed osseous tissue, which, especially in the severe osteomalacic form, is accompanied by extreme deformities; hence the permanent abnormally soft consistency of such bones. The innumerable theories advanced as to the pathogenesis of rachitis are still too uncertain to permit of discussion here.

DIAGNOSIS

This is not difficult in typic cases. The beginning of rachitis may be recognized by increasing restlessness, sweating of the head, increasing pallor, pain on being lifted, and dyspepsia without any apparent reason.

DIFFERENTIAL DIAGNOSIS

HYDROCEPHALUS	THE RACHITIC HEAD
Rounded, with protruding frontal and temporal bones; fontanels are arched outward and widely open. Lack of proportion between the small facial and the large cranial portion of the skull. Convergent strabismus. Signs of cerebral pressure; mental disturbances.	Angular; fontanels often covered by the overlapping edges of the bones; craniotabes. No symptoms of cerebral pressure.
KYPHOSIS OF SPONDYLITIS	RACHITIC KYPHOSIS
Forming a sharp angle; lying upon abdomen does not cause it to disappear.	Round; disappears in time when the abdominal posture is assumed; painless.

PLATE 8

FIG. 1. **Fetal Chondrodystrophia.**—The distal epiphyseal border of the tibia of an almost full-term infant which was dead when born. Magnified 41 times. The bony cartilaginous border shows gross irregularities. The cartilage protrudes somewhat in a lateral direction. Endochondral bone growth is hindered by the fact that the quiescent cartilage is not transformed into large vesicular cells with their arrangement into columns, because of the entrance of connective-tissue processes from the perichondrium between the cartilage and diaphysis. In place of the columnar cartilage there is found a spongy degenerated cartilage tissue. Temporary calcification is only suggested here and there; primary medullary spaces are absent; the cartilage rests in certain areas directly upon the marrow and bone tissue. The bony trabeculæ of the spongiosa are few and thickened and the medullary spaces widened. The periosteal bony growth—that is, the growth in width—is only slightly disturbed. A plate of periosteal bone grows over the cartilage for some distance. 1. Quiescent cartilage. 2. Hyaline degenerated cartilage. 3. Connective-tissue process growing inward from the perichondrium. 4. Temporary calcification. 5. Periosteal bony lamellæ. 6. Enlarged medullary space. 7. Periosteal bony plate. (Preparation of Prof. Stoeltzner.)

FIG. 2. **Rachitis of a Flat Cranial Bone. Craniotabes.**—Enlarged 52 times. The osteogenetic layer of the periosteum is richly supplied with cells and is increased in size by proliferation. Between its fasciculi are embedded narrow indentations and trabeculæ of young bone tissue which are periosteal in origin. Toward the center the trabeculæ lie closer together and, finally, form a connected cortex. The excretion of lime salts is insufficient in amount. Although the outermost (youngest) bony trabeculæ still plainly show calcification in their centers, yet the cortex is composed almost entirely of decalcified osteoid tissue. The inner surface of the bone is serrated on account of the active absorption of bone by the osteoclasts. 1. Fibrous layer (periosteum). 2. Osteogenetic layer (periosteum). 3. Calcified bony tissue. 4. Osteoid tissue, possessing no calcium salts. 5. Cortex poorly supplied with lime. 6. Osteoclasts. (Preparation of Prof. Heubner.)

MOLLER-BARLOW'S DISEASE	RACHITIS
Clinical	
Sensitive to movement of the lower extremities. Painful swelling of the epiphyses, especially of the lower extremities. Specific disease of the gums. No response to the rachitic treatment; follows use of uncooked food.	Pain on moving thorax. Almost painless swelling of the epiphyseal cartilages of the upper and lower extremities. Gums intact.
Anatomic	
Hemorrhages into bone-marrow and periosteum; characteristic degeneration of the bone-marrow.	Insufficient bone formation. Absence of order in the process of ossification.

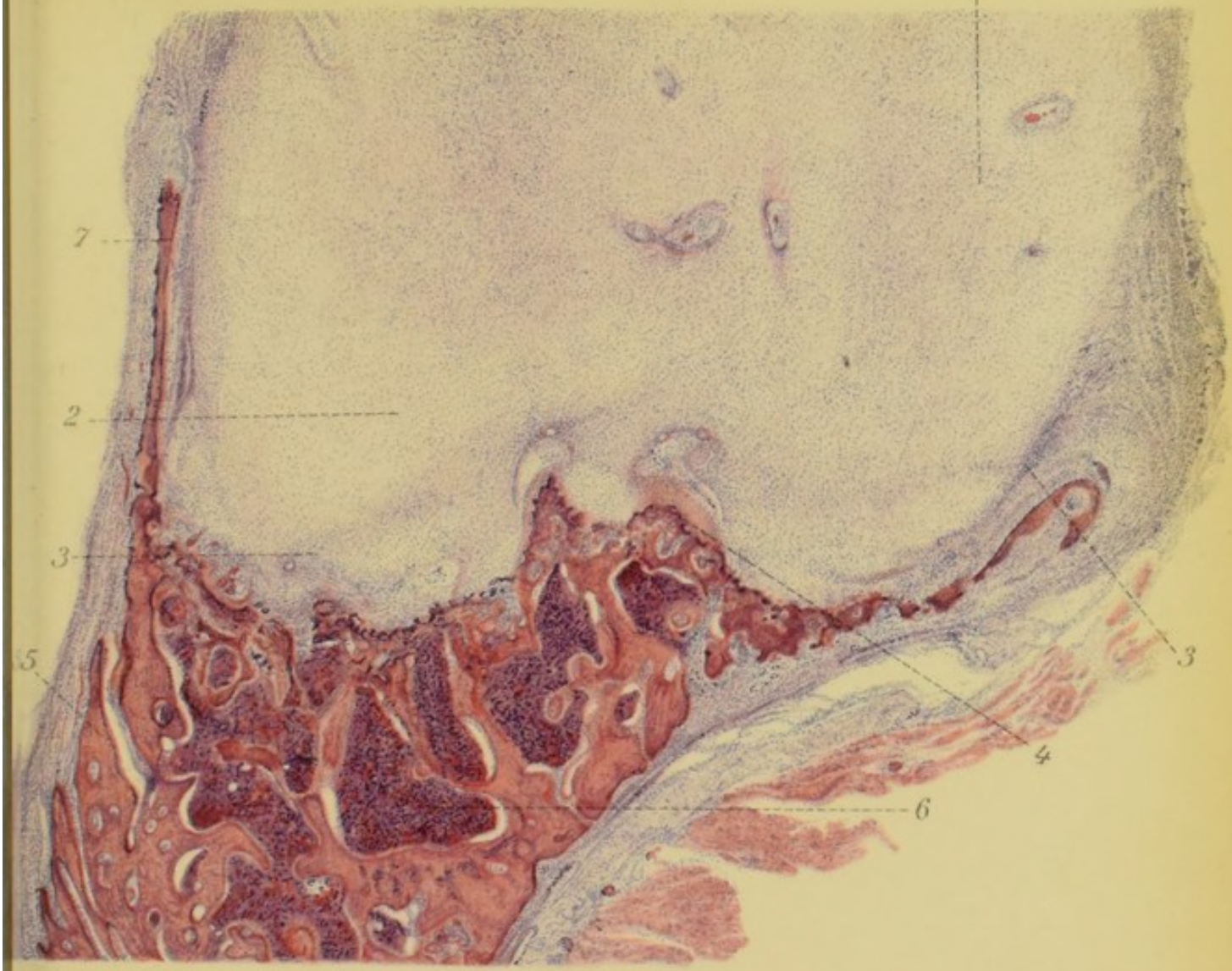


Fig. 1.

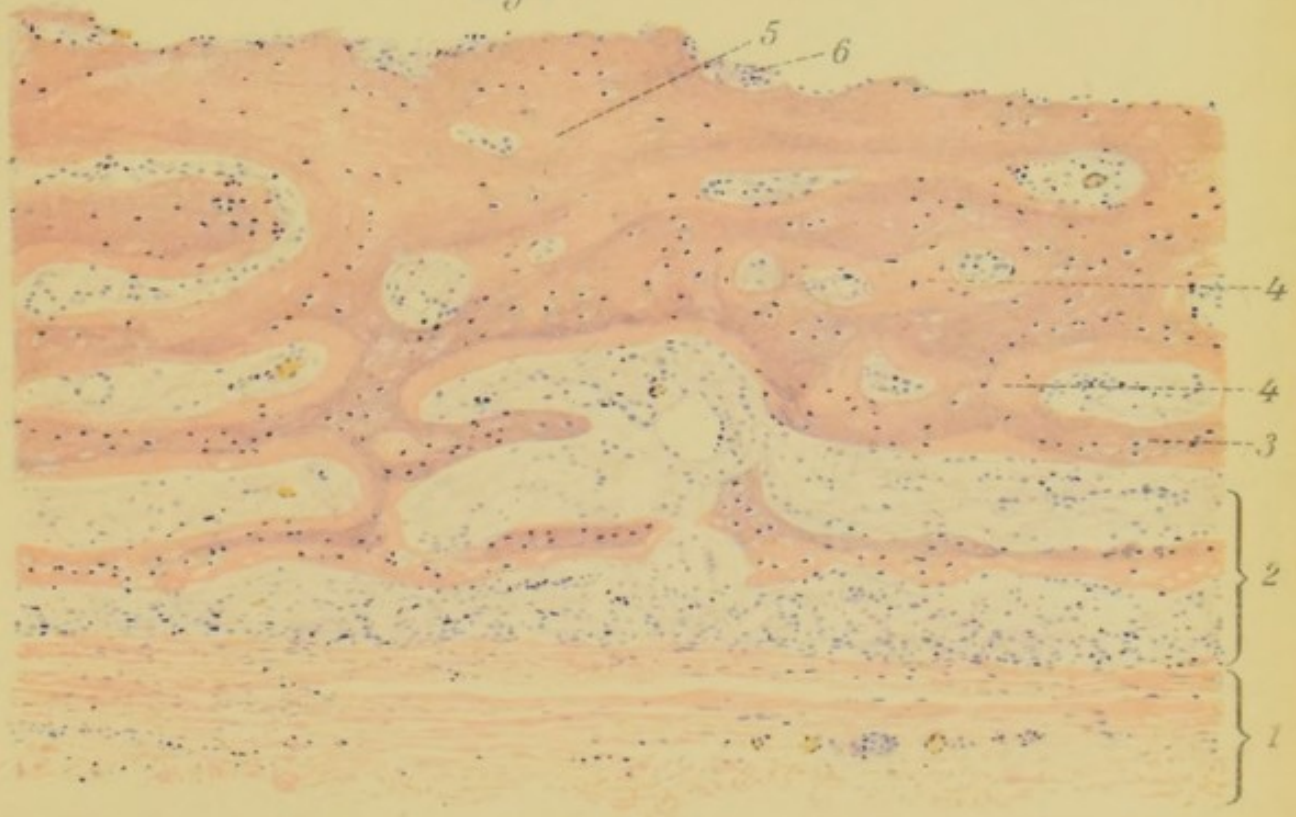


Fig. 2.

Syphilitic Osteochondritis	Rachitic Swelling of the Epiphyses
Clinical	
Occurring during first weeks of life.	Occurring usually during the last half of first year of life.
Painful.	Almost painless.
Swelling usually of only one lower epiphysis of the humerus or femur. In severe cases separation of the epiphysis and pseudo-paralysis.	Multiple epiphyseal swellings in all extremities.
Anatomic	
Irregular borders between cartilage and bone, which are pointed, rough, and serrated.	Irregular border between cartilage and bone, which is rounded off, and presents serrated notches which feel soft to the palpating hand.
Narrowed zone of cartilage proliferation.	Thickened zone of cartilage proliferation.
Enlarged zone of temporary calcification.	Partial or complete absence of zone of temporary calcification.
Interference with construction of organic (osteoid) tissue, associated with undisturbed deposition of the inorganic (calcium) substance.	Insufficient deposition of the inorganic (calcium) substance, associated with undisturbed formation of organic (young bone) substance.

Chondrodystrophia	Rachitis
Interference with longitudinal growth of the tubular bones, associated with undisturbed growth in width, in consequence of a cessation in cartilaginous proliferation due to the intergrowth of connective tissue.	Disturbance of all bony growth, on account of insufficient deposition of calcium, and the effects which arise thereform.
Appears to be congenital.	Occurs during early life of infant.
Incurable.	Curable.

THE TREATMENT OF RACHITIS

Both as a preventive and as a cure it is of prime importance to combat the injurious effects of a bad atmosphere. Provide fresh air in the room or insist upon sojourn in the open air (even when the weather is bad); much sunshine. [Many of the leading clinicians abroad

send their little patients to the seacoast, believing they are benefited.—Ed.]

Diet.—As a prophylactic measure, the child should be fed on mother's milk or that of a wet-nurse; or, as a substitute, raw milk properly modified and animal broths made from fresh bones. Limit starchy food. Fruit juices are considered antirachitic. At the end of the first half year allow a mixed diet, with the addition of fresh vegetables, vegetable broths, potatoes and chopped meat, scraped or crushed fruit.

Salt or brine baths (32° to 35° C. [89.6°–95° F.], see p. 70) two or three times a week, for a period not longer than four weeks. Exercise judgment in case of weak children. Mild massage; rub off with brandy or bathe with eau de Cologne; in midsummer, short sun-baths. The soft bones should be spared in carrying, standing, or walking. Corrective movements indicated for the curvatures. Osteoclasis and other orthopedic procedures. Osteotomy. [Since many of the rachitic deformities of the extremities tend toward spontaneous cure, no surgical intervention should be employed until at least the fifth or sixth year.—Ed.]

Medication.—Phosphorus, 0.01 : 100 gm. ($\frac{1}{100}$–$\frac{1}{200}$ gr.); cod-liver oil, 1 coffeespoonful once or twice a day. Gärtner's phosphoretted chocolate plates, of each $\frac{1}{2}$ mg. In this manner a favorable influence is produced upon the general condition, especially upon the nervous and spasmodic phenomena. To combat the anemia give iron or the iodid of iron, and later give the compound tincture of cinchona in 1-drop doses. Somatose. For the sweats give vinegar-water, 10 per cent. vinegar spirits.

CONGENITAL DISTURBANCES IN BONE DEVELOPMENT

(*Fetal Rachitis; Imperfect Osteogenesis; Congenital Achondroplasia; Fetal Myxedema*)

Purely congenital rachitis is rarely encountered. The conditions which have been described under this name



FIG. 44.—Imperfect osteogenesis (fetal rachitis). Girl of eight months. The extremities, which in comparison with the length of the body are abnormally short, show deformities and healed fractures. Mild macroglossia; head slightly hydrocephalic. Death following bronchitis. (Escherich's Clinic, Vienna.)

FIG. 45.—*Fetal Achondroplasia.* Skeleton of a micromelic dwarf. Bones of the extremities short and thick, with wide—especially at the knee—and considerably thickened epiphyses. No fractures. (Graz Pathologic Institute.)

FIG. 46.—*Fetal Achondroplasia.* Pure type. The extremities are shortened, but not deformed or fractured. (Clinic of Pfaundler, Graz.)

FIG. 47.—*Fetal Achondroplasia.* Skiagram of foregoing case, showing the shortened long bones of the extremities, without deformities or fractures, and their enlarged epiphyses. It may be seen from the intensity of the shadows that a certain amount of sclerosis exists. (Clinic of Pfaundler, Graz.)

should be classified either under imperfect osteogenesis, achondroplasia, or fetal myxedema.

Imperfect Osteogenesis.—The child's extremities at birth are abnormally short and plump, and show considerable distortion and fractures. Crepitation is plainly felt in the flat cranial bones, the jaws, the pelvis, etc. The rest of the body, as a rule, shows nothing abnormal. The child may continue to live. The cause is unknown.

Morbid Anatomy.—The short and thick tubular bones show a thin cortex, a brittle and sparse spongiosa, and enlarged medullary spaces; multiple fractures. The zones of cartilaginous proliferation, the progress of calcification, and the formation of primary medullary spaces, in the main, occur normally; on the other hand, however, there is great irregularity in the formation of real bone. The bony trabeculæ are lacking in size and number and are not arranged in lamellæ; the development of the bone-forming cells—the osteoblasts—is faulty, and they fail to functionate properly; there is also excessive absorption of bone tissue.

The endochondral ossification is usually disturbed to a greater extent than the periosteal (see also Normal Ossification, p. 122); the marrow is poor in cells, gelatinous, and "inactive." Ossification is undisturbed and callus forms at the site of the fractures. The thyroid gland is normal (Harbitz).

Congenital Achondroplasia.—The extremities are notably short, usually straight, but at times somewhat curved. No fractures. A pure form of dwarf growth. The bones are hard, sclerosed, and the epiphyses thickened.

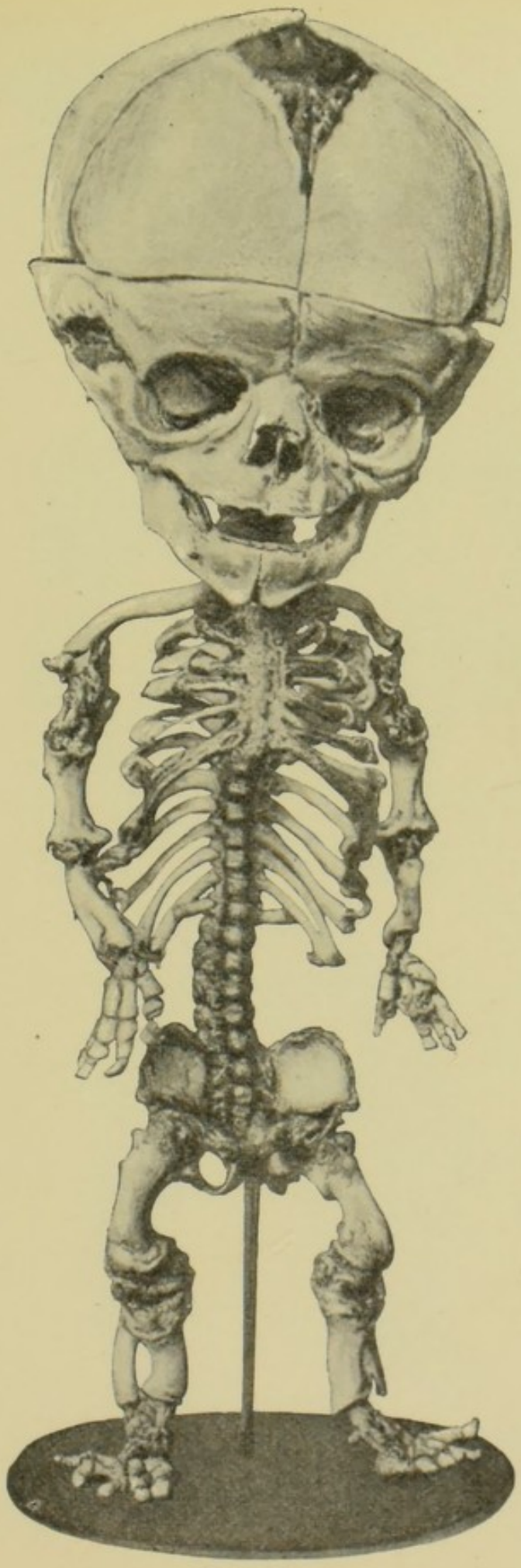


FIG. 45.

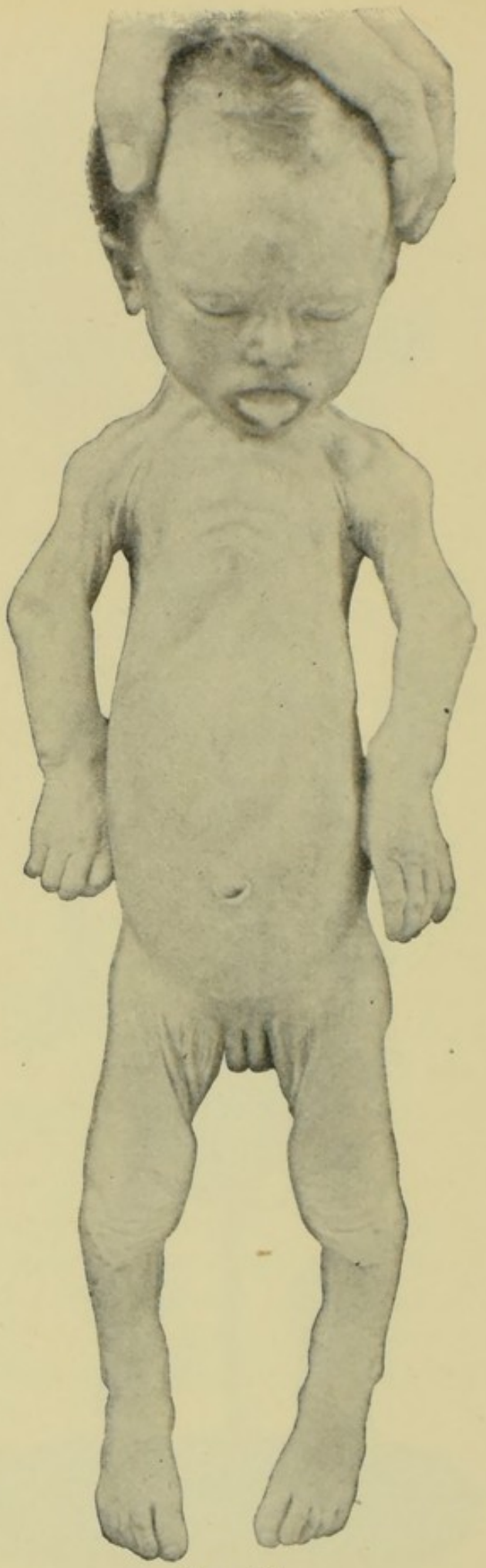


FIG. 46.

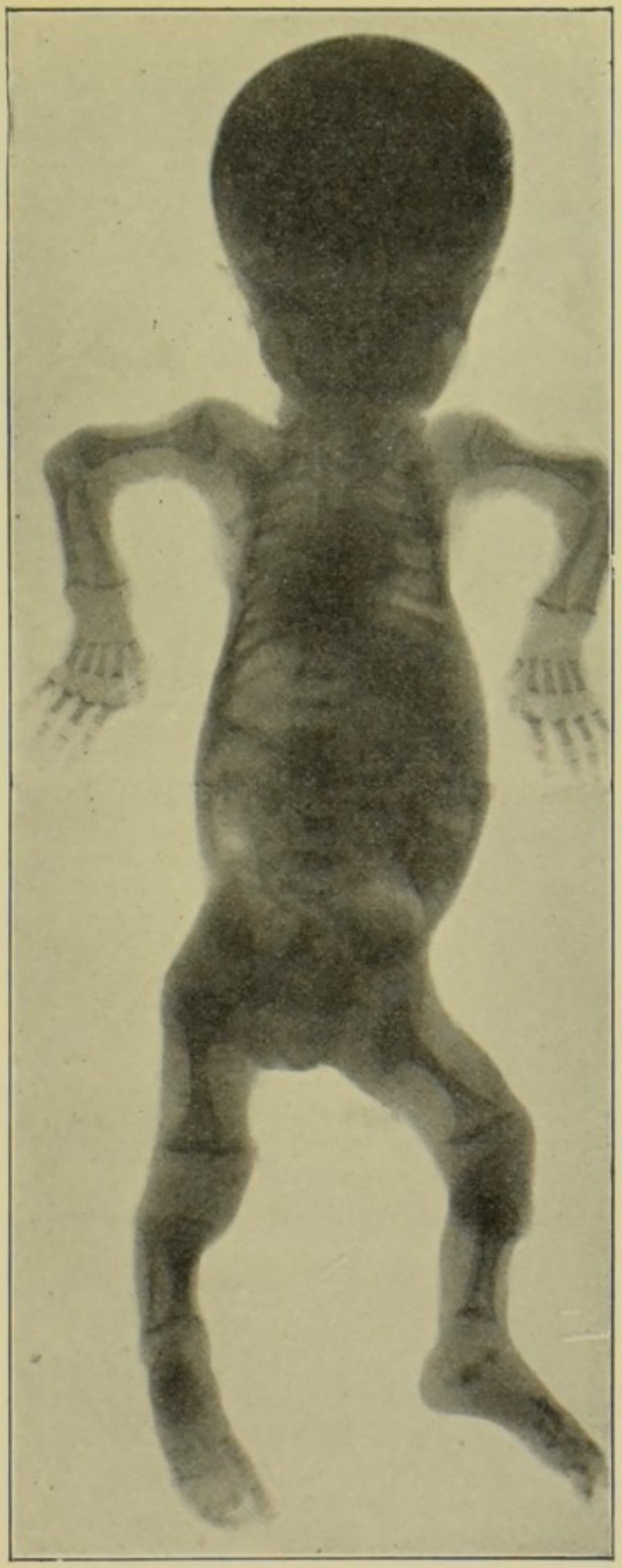


FIG. 47.

PLATE 9

Barlow's Disease.—A portion of Fig. 49, which represents the area marked by a rectangle. Enlarged 150 times. 1. Large vesicular cartilage cells. 2. Temporary calcification of cartilaginous ground substance. 3. Youngest bony trabeculæ. 4. Endosteal hemorrhage. 5. Subperiosteal hemorrhage. 6. Bony trabeculæ disappearing on account of bony absorption. 7. Fibroid degeneration; marrow poor in cells.

FIGURE 49

Barlow's Disease.—Longitudinal section of a distal epiphysis of the femur. Enlarged 5 times. (As the artist unfortunately broke the preparation, it had to be repaired and somewhat reconstructed at the line of fracture; it is therefore undecided whether a fracture occurred during the disease or whether one existed before that period.) Hemorrhages (red) are noted spreading underneath the periosteum, as well as within the spongiosa, where they are especially well marked in the vicinity of the line between the cartilage and bone (loosening of the epiphysis usually occurs at this point). The zone of proliferation, especially of the columnar arranged cells, is broadened. Underneath this is a narrow irregular zone of temporary calcification; connecting with it are young bony trabeculæ (blue), which are well developed, but few in number; near the diaphysis they become converted (through excessive bony absorption and crowding in of marrow tissue) into small closely crowded bony trabeculæ. This gives the spongiosa a checkered appearance. The cortex cannot be distinguished, for it has disappeared as the result of a pathologic increase of bony absorption. (From a preparation of Prof. Schmorl, in the possession of Geh.-Rat Heubner.)

Microscopically are seen: Disturbance of the endochondral ossification by limitation of cartilaginous proliferation (no column formation of cells), and the penetration between cartilage and bone of perichondral connective tissue. These disturbances interfere with the longitudinal growth. The asymmetric growth of periosteal strips leads to disproportionate development and the formation of deformities. (Histology of Achondroplasia, see Plate 8, Fig. 1.)

Fetal Myxedema.—The infant presents the phenomena of a pronounced case of myxedema, including thick, dry skin, peculiar expression of face, large head, and plump body. The extremities are short and thick. The thyroid gland is imperfectly or excessively developed. Anatomically the bones are the seat of achondroplasia or imperfect osteogenesis. Such children are either still born or live but a short time as micromelic dwarfs.


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Fig. 49.

BARLOW'S DISEASE

Infantile Scorbutus: Acute Hemorrhagic Rachitis.—This is a peculiar disturbance of nutrition, which can be classified neither with scorbutus nor with rachitis. It is a disease process of the osseous system, accompanied by distinct changes in the bones and a tendency to hemorrhage.

Occurrence.—It develops in the first or second half year of life and almost exclusively in artificially fed children.[1] It seems to affect especially the well-to-do middle class, and is frequently combined with rachitis.

Symptoms.—Severe pain upon movement, especially of the lower extremities; adduction of the legs; livid color of the face with increasing pallor of the skin; extreme weakness. Painful swelling of the epiphyses of the knee or of the diaphyses of one or both femurs. At a later stage in the disease there is also epiphyseal swelling of the tibia and the arms. The skin over the swellings is tense, shiny, whitish, and (rarely) bluish red. There is extravasation of blood in the gums, which may be recognized by the presence of little bluish-red masses between the teeth.

Occasional Phenomena.—Hemorrhagic swellings of the ribs, scapula, and in the orbits, where the eyeballs are pushed forward; hemorrhages of the skin and mucous membranes; hemorrhagic nephritis. (For Differential Diagnosis between it and Rachitis, see p. 128.)

Course.—This disease lasts for weeks and months, and if the proper treatment is instituted the prognosis is always favorable.

Morbid Anatomy.—The swellings are due to hemorrhages in and under the periosteum, as well as between the spongiosa and bone. As a result of the latter we have epiphyseal separation and greenstick fractures. Another phenomenon is a peculiar degeneration of the highly cel-

[1] Whose food—no matter in what form—is constantly heated to the boiling-point. Definite food preparations or sterilization need not necessarily cause this condition (Heubner).

lular bone-marrow, consisting of conversion into loose connective tissue, which is but poorly supplied with blood-vessels and cells. The osteoblasts disappear, the spongiosa undergoes softening and destruction. The general growth of the bone is interfered with.

Treatment.—Barlow's disease is highly responsive to treatment. Administer raw milk or, if not feasible, give milk which has been boiled as little as possible. When necessary dilute the milk with uncooked solutions of infant meal, nutritive sugar, etc. Also give the juice of raw meat and uncooked fruits, green vegetables, and mashed potato.

DISEASES OF THE THYMUS GLAND

STRUMA

Enlargement of the thymus gland involves one or more of its lobes. Anatomically struma is usually of the parenchymatous form (soft), rarely of the colloidal, cystic, or fibrous variety.

Etiology.—It is frequently hereditary and is influenced by peculiar geographic conditions (endemic goiter). Other causes are tight collars, development of puberty, and not infrequently this condition is caused by continued lying on the abdomen. Dorsal flexion of the head during infancy, incorrect position when writing (school goiter), and pertussis are also of etiologic interest.

Complications.—Compression of the trachea; noisy, dyspneic respiration; compression of the jugular veins; mental disturbances; vertigo; headache.

Course.—Usually favorable.

Treatment.—When possible, the cause must be eliminated. The neck should be free from clothing, and if the subject is an infant it should be allowed to assume none but the dorsal position. Inunctions of potassium iodid ointment should be resorted to as well as the tincture of iodin and of nutgalls; thyroid extract tablets (see p. 145). [Favorable results are being constantly obtained

by the administration of thyroid extract in doses of from 1 to 5 gr. *t. i. d.* These patients should be carefully watched for the physiologic symptoms, such as rapid pulse, loss of weight, pyrexia, and diarrhea. Drugs should be stopped when either of these symptoms occur. When there is mechanical interference with the trachea or great vessels, surgical intervention is indicated.—Ed.]

BASEDOW'S DISEASE

This condition depends upon excessive function of the thyroid gland. The cardinal symptoms include: Pulsating goiter, exophthalmos, palpitation with hypertrophy of the heart, which is often incompletely developed, sweats, dyspnea, tremor, excitability, and a feeling of fear.

Course.—Basedow's disease runs a chronic course. The result is either complete recovery with enlargement of the heart, or death due to cardiac failure or some intercurrent affection.

Treatment.—The treatment consists in rest, an easily digestible diet, especially milk; fresh air and mild hydrotherapeutic procedures. A trial should be made with myxedema serum, "Rodagen" (Merck-Moebius).

HYPOTHYROIDISM. DYSTHYROIDISM

These terms are applied to all diseases due to faulty or to complete absence of the function of the thyroid gland. In such cases only a rudimentary gland exists and sometimes cannot be felt (athyroidia). Clinically the following groups of symptoms may be distinguished:

Chronic Benign Hypothyroidism (Hertoghe).—This is usually not recognizable until the second or third decade of life. Sexual instinct undeveloped; beardless face; childish voice; narrow thorax; dryness of hair. During childhood it is often preceded by wetting of the bed, adenoid vegetations, headache, chronic constipation, and meteorism.

FIG. 48.—Mild type of myxedema with a moderate degree of imbecility. Five-year-old girl. Stupid, anxious expression of face, a heavy appearance about the lower jaw; double chin; short neck; broad chest. The skin felt hard, dry, and was thickened. The thyroid gland was not palpable; all movements were somewhat stiff.

FIG. 50.—Pronounced infantile myxedema. Girl of six and a half years. A uniform tense swelling of the skin of the whole body; dull expression of face. Eyes small and deeply set; nose but slightly elevated; lips swollen; a thick, short neck; double chin; protruding breasts; thick hands and arms; mild genu valgum. Excerpts from patient's history: Increase in weight since the second year of life; cessation in longitudinal growth during the fourth year. At that time the weight was 20.8 kg. [44 lbs.] (normal, 19.5 kg. [42.9 lbs.]); length, 92.7 cm. [37.4 in.] (normal, 105 cm. [42 in.]). (Clinic of Escherich, Vienna.)

FIG. 51.—The same girl after three months' treatment with a fresh preparation of the thyroid gland. Length, 95.5 cm. [38.2 in.]; weight, 16.7 kg. [36.7 lbs.]. (Note the change in expression in the eyes, the nose, mouth, neck, chest, waist, and upper and lower extremities.)

FIG. 52.—Myxidiotie. Symptoms of myxedema and idiocy. (Clinic of Escherich, Vienna.)

FIG. 53.—Myxidiotie. A pronounced case of myxedema with fully developed idiocy. Six-year-old girl. Length, 78 cm. [31.4 in.] (normal, 102 cm. [40.8 in.]). The thyroid gland is absent. Specific treatment was refused by the mother.

Infantile myxedema represents the true myxedema of adult life. The skin is pale, thickened, dry, stretched, and usually cool; the eyelids are swollen and the face is muddy. The lips and tongue are thickened; chronic constipation; distended abdomen; dry, brittle hair, which has a tendency to fall out; a chilly feeling is pronounced. The disposition is apathetic and peevish. The facial expression is immobile and slightly stupid; slowness of speech and movement; diminished longitudinal growth; increased body weight.

Myxidiotie, Sporadic Cretinism, Infantilism.—The symptoms of myxedema together with an enlarged protruding tongue; flowing of the saliva from the mouth; a silly and dull facial expression; plump, short extremities; varying grade of idiocy; interference with longitudinal growth resulting in dwarfism. In the majority of cases of hypothyroidism the Röntgen-ray photograph demonstrates a retardation of ossification in the skeleton of the hand.

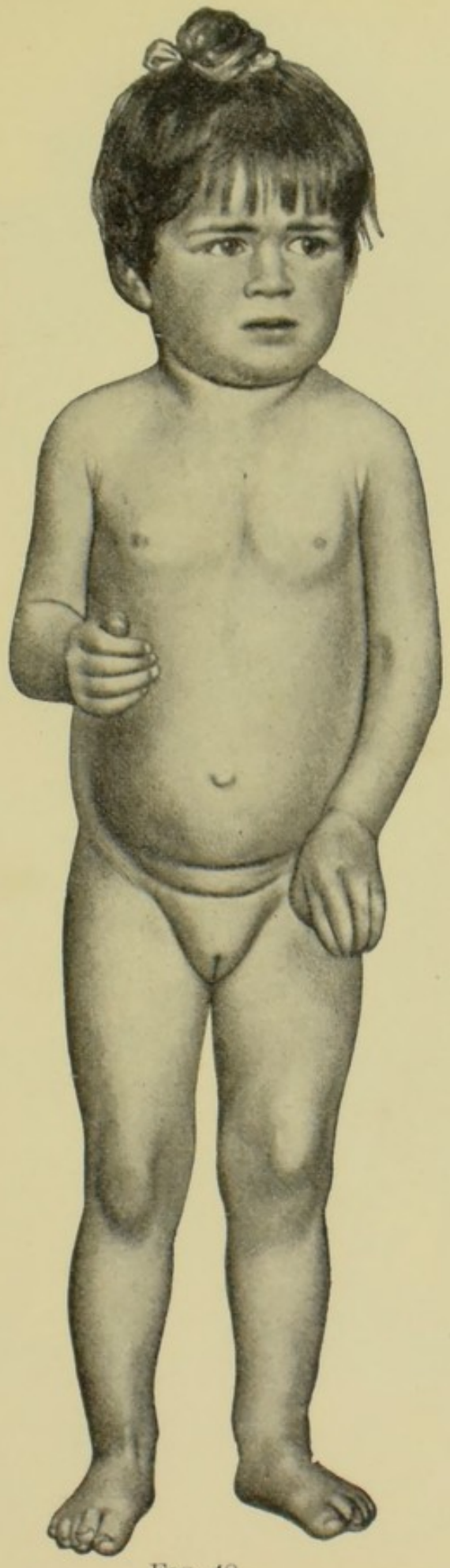


FIG. 48.

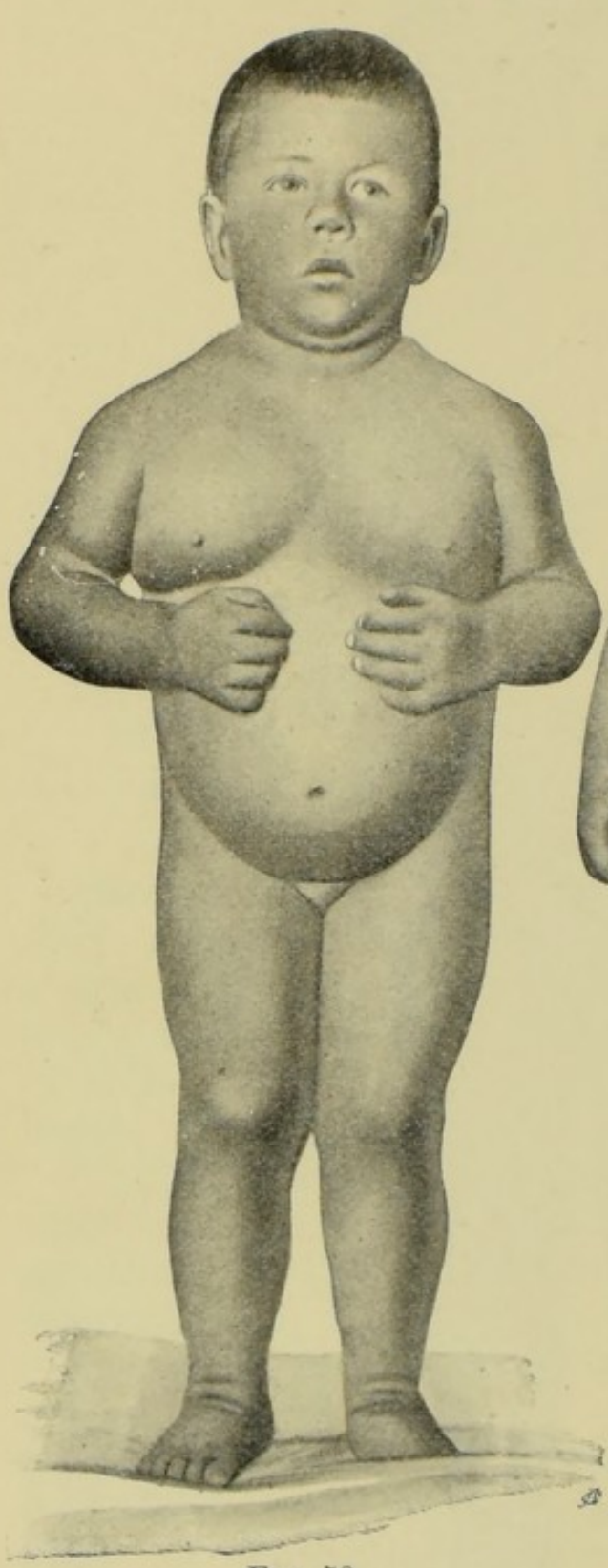


FIG. 50.

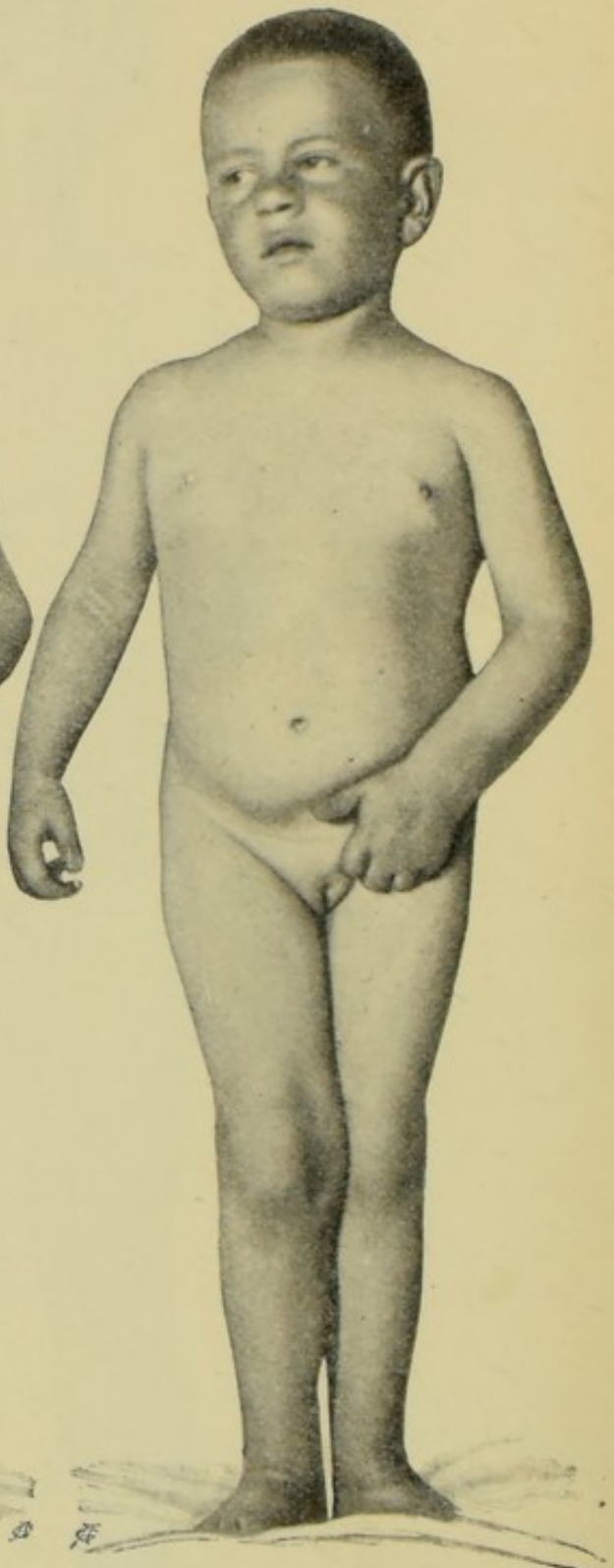


FIG. 51.



FIG. 52.



Fig. 53.

The course is chronic in all forms of hypothyroidism; which with the cessation of treatment may disappear or even continue for a long time. If the skiagram demonstrates the presence of the intermediary cartilaginous plate on the epiphyses of the hand, it may be considered a favorable sign.

Fetal Myxedema.—(See p. 136.)

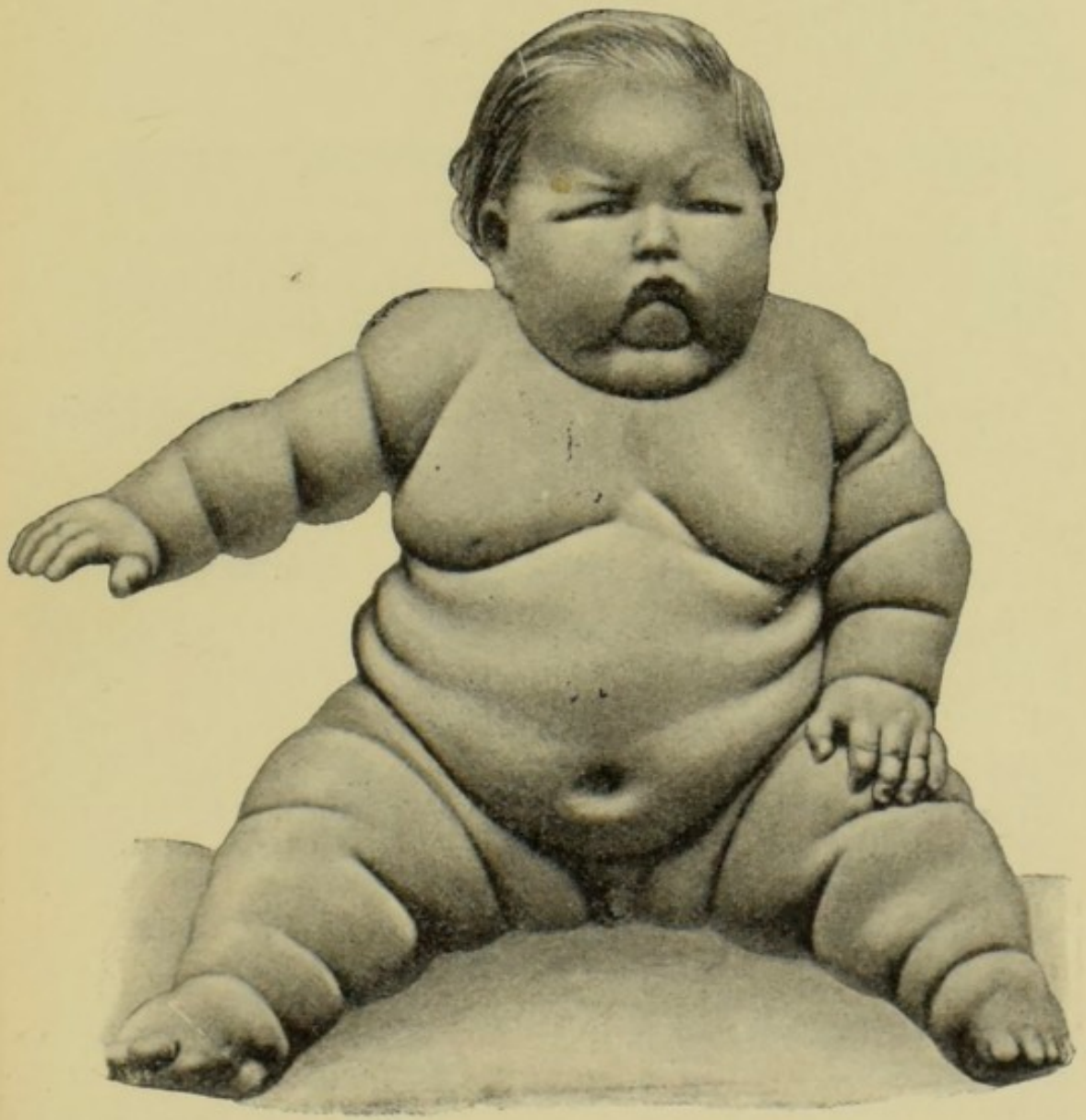


FIG. 54.—Infantile obesity. (Clinic of Escherich, Vienna.)

Treatment of Hypothyroidism.—This disease responds most satisfactorily to the specific treatment, which consists in the administration of from 0.05 to 0.2 gm. or 0.3 gm. of thyroidin (Merck; Burroughs and Welcome).

Begin cautiously with small doses, and stop its use when cardiac palpitation, restlessness, fever, and albuminuria arise. This treatment should continue throughout life. Warm baths and hot packs should be frequently resorted to.

OBESITY

This rarely occurs in children; it is either congenital or due to improper feeding. In nursing infants it is frequently rapid in development. If of the anemic type there is pallor of the face and of the mucous membranes; the pulse is small and there is a tendency to become easily fatigued. In the plethoric type the face is fresh and red in appearance, the pulse is full, the musculature strong, and the temperament phlegmatic. In nursing children the course is chronic and there is danger from the lack of resistance against intercurrent infections.

Treatment.—Reduction of fats and carbohydrates; active and passive motion; massage.

HEMORRHAGIC DIATHESIS. PURPURA

These two conditions are associated because of the following symptoms which they possess in common: They develop as independent diseases accompanied by hemorrhages in the skin and mucous and serous membranes. Their etiology is unknown. Each individual form represents but slight differences of the same disease process.

SIMPLE PURPURA

Hemorrhages occur only in the skin as isolated groups, about the size of a pin's head or, at the most, the size of a lentil. Then eruption begins in the legs and travels upward in groups. The general health is but slightly disturbed. There is no fever, but weakness and depression exist. The eruption disappears, but frequently tends to recur. Complete recovery may be expected in a few weeks.

RHEUMATIC PURPURA (PELIOSIS)

This is accompanied by an eruption of dark red spots on the legs and feet, abdomen, arms, and genitalia, and particularly also on the knee-, ankle-, and elbow-joints. The affected joints are swollen and painful; the tibia, small bones, and other bones are also frequently sensitive. There is weakness and loss of appetite. Fever is often absent or, if present, is but slight. The course is prolonged throughout a number of weeks on account of the tendency to frequent reappearance of the eruption.

HEMORRHAGIC PURPURA (MORBUS MACULOSUS WERLHOFII)

In this disease hemorrhages occur not only in the skin but also in the mucous membranes and in the internal organs. Dark red spots, varying in size from that of a lentil to that of a pigeon's egg, which do not disappear on pressure, are found on the skin of the extremities and of the trunk ; pea-sized hemorrhages occur on the mucous membrane of the mouth, nose, and in the conjunctiva. Occasionally there is also hematuria, bloody stools, vomiting of blood, rarely bloody expectoration, and retinal or meningeal hemorrhages. The disease begins with a vague feeling of ill health, followed, as a rule, by a sudden eruption and extension of spots over the whole body within twenty-four hours. These become confluent and lead to large extravasations, gradually changing to a brownish-red or dark blue color. There is considerable disturbance in the general health, weakness, headache, and articular pains ; decided anemia ; slight fever and slow pulse.

Course.—The symptoms recede within two or three weeks and the patient gradually passes into convalescence. Relapses are common. There is danger of continued hemorrhages, leading to severe anemia and weakness. The prognosis is, therefore, not absolutely favorable.

ABDOMINAL (HENOCH'S) PURPURA

To the symptoms of rheumatic purpura are added complex abdominal phenomena : vomiting, colic, and intes-

PLATE 10

Purpura Hemorrhagica.—The disease in a nine-year-old boy ran the course of Henoch's purpura, accompanied by severe gastro-intestinal symptoms. It began with fever and painful swelling of the left knee-joint. On the following day an eruption appeared in the neighborhood of both knees, consisting of a large number of red spots, varying in size from that of a lentil to that of a cherry, which failed to disappear on pressure. A similar eruption gradually appeared on the elbows, nates, scrotum, and penis, while all visible mucous membranes remained free. The fifth day of the disease was followed by almost constant vomiting, severe colicky pains, bloody stools, and rapid decline. Ice, opium, and ergotin remained ineffectual. Injections of 0.0003 gm. of atropin (three in all) led to immediate improvement and to a permanent relief of the intestinal symptoms. The hemorrhages of the skin recurred but twice during the course of the next year.

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tinal hemorrhage, the intensity of which causes them to predominate. The attacks occur gradually in spells at intervals of several days, weeks, or even a year. The vomiting is very obstinate and controlled with difficulty, the colic is extremely painful and deprives the patient of sleep (children lie in bed groaning and twisting about). The stools contain either fresh blood or they are discolored black and sometimes orange colored. The general health is decidedly affected and there is loss of strength on account of the hemorrhages and the inability to take food.

The **course** is protracted, but usually favorable.

FULMINATING PURPURA

Hemorrhages of the mucous membranes are absent. Extensive ecchymoses develop, which lead with enormous rapidity in a few hours to hemorrhagic infiltration and dark blue discoloration of the whole extremities. Death follows in from one to four days.

TREATMENT OF PURPURA

Rest in bed is necessary in all forms (including the mild types), as is also a non-irritating diet—milk, infant foods, meat-soups, eggs, chopped meat, tender vegetables, raw fruit juices, and fresh fruit. Acid drinks, citric or phosphoric acid, are of value. If hemorrhages occur, a 1 per cent. solution of ergotin or fluidextract of ergot



may be used. Also the powdered extract of the suprarenal gland or adrenalin.

In case of rheumatic purpura, immobilize the joints and apply ichthyol dressings ; internally give sodium salicylate or aspirin. In abdominal purpura a strict dietary must be followed : Ice, ice-water, iced milk, milk of almonds, ice-bag to the abdomen ; injections of atropin sulphate in from $\frac{1}{4}$ to $\frac{1}{3}$ mg. doses [1 to 5 per cent. solutions may be given internally with advantage] ; gelatin injections ; less certain than these in action is 0.05 gm. of the extract of opium in 120 gm. of the emulsion of amygdala.

ANEMIA

Condition of the Blood.—Diminished amount of hemoglobin and a decrease in the number of red blood-cells (oligochromemia and oligocythemia). Poikilocytosis ; leukocytes unaltered.

Etiology.—Simple anemias are most often secondary, following chronic and severe acute diseases, especially tuberculosis, syphilis, rachitis, gastro-intestinal diseases, intestinal worms, chronic kidney and heart disease, pleuritis, pneumonia, etc. Other powerful factors are unsuitable food, damp, overcrowded dwellings, attending school (school disease), and premature and excessive hardening processes. Anemia occurs at any age, but it is especially frequent in the first two years of life and the years preceding puberty.

Symptoms.—Pale and somewhat dry skin and pale mucous membranes ; certain constitutional phenomena ; weakness, easily fatigued, irritability and rapidly changeable disposition, headache, and constipation. Blowing anemic murmurs, chiefly over the pulmonic area, venous hums (apply stethoscope lightly, as tight pressure alone may create murmurs).

Pernicious anemia is very rare in childhood. The blood shows the changes incident to anemia together with megalocytes and megaloblasts. Symptoms and course as in adults.

CHLOROSIS

Condition of Blood.—Diminution in the amount of hemoglobin (oligochromemia), without a decrease in the number of red blood-cells. There is a noticeable difference in the size of the latter, there being many macro- and microcytes. The white blood-corpuscles show no characteristic changes.

Etiology.—The blood-building organs are less active. Predisposing factors are: Unfavorable conditions of dwellings and feeding, wearing of corsets, premature difficult physical or mental work, and insufficient sleep. The disease chiefly attacks girls before and after the period of puberty, yet it is sometimes found in boys.

Symptoms.—A pale to pale-green color of the skin; pale mucous membranes. Adipose tissue well preserved. Slight edema of the knuckles. An unusual degree of weariness and desire to sleep; dyspnea; loss of appetite, with a special dislike for meat. Irritability, headache, vertigo, ringing in the ears, and painful sensations in the region of the stomach and the ribs. Enlargement of the cardiac dulness to the right. Blowing murmurs of varying intensity at the apex of the heart and in the pulmonic area; venous murmurs in the right side of the neck. In the diagnosis we must exclude secondary anemia, especially tuberculosis, gastric ulcer, and intestinal worms.

TREATMENT OF ANEMIA AND CHLOROSIS

The cause must be eliminated. At the beginning the patient should receive much rest and sleep or indulge in a rest-cure lasting several weeks. Exercise in the open air is to be postponed until later. Protect against heat dissipation by careful selection of clothing; no cold-water procedures without supervision.

Feeding.—For nurslings give raw or slightly boiled milk, and adopt a mixed diet early in life. In case of older children exclude coarse, indigestible food and give pressed meat juice, chopped meat, iron-containing vegetables, green lettuce with oil and lemon, raw or cooked fruits, and good cows' or goats' milk. Vary the diet

as much as possible. Stimulate the appetite with purée of meat juice, caviar, sardines, etc. (all in small quantities). Medication includes the compound tincture of cinchona or wine of iron and quinin (6 to 10 drops of the former and a coffeespoonful of the latter before or after meals). [The albuminates of iron—although for the most part proprietary remedies—are best adapted for administration to children. They do not injure the teeth and are well tolerated by the stomach. This class is represented by the peptomanganates, hematogen, etc.—Ed.]

Hydrotherapy.—Older children are treated with heat by the application of hot packs to the whole body; hot baths (37° to 38° C. [98.6°–100.4° F.]) or vapor baths, followed by a short cold-water bath (be cautious in case of delicate children). Follow by rest in bed, rub down with brandy or eau de Cologne; massage. It is best to resort to these various procedures in regular rotation with days of rest intervening. All procedures should cease in from four to six weeks. Short air-baths in the room with exercise; sun-baths in midsummer. In severe cases give arsenic with iron (4.0 gm. of Fowler's solution added to liquor ferri albuminatus 20.0 gm.; give of this from 2 to 8 or 15 drops three times a day in slowly increasing and likewise decreasing doses). Sojourn in the country at sunny resorts; high altitudes; bathing resorts.

SPLENIC ANEMIA: INFANTILE PSEUDO-LEUKEMIA

Condition of the Blood.—Diminution of hemoglobin and of the number of red blood-cells; nucleated erythrocytes; slight leukocytosis; occasionally megaloblasts.

Symptoms.—The various phenomena of anemia accompanied by a protruding abdomen, enlarged spleen (which is palpable as a hard, non-painful movable tumor), and enlarged liver; the cervical glands are swollen. It runs a varied course with gradually increasing cachexia; cure is possible, but rare.

Treatment.—Arsenic with iron or injections of arsenic. (For other details of treatment, see Anemia.)

CHRONIC INFECTIOUS DISEASES

HEREDITARY OR CONGENITAL SYPHILIS: HEREDOSYPHILIS

Transmission.—The infection occurs in all cases before birth, and is usually transmitted from the father by the introduction of the virus into the ovum simultaneously with its union with the spermatozoön; in this case the mother remains healthy. More rarely the infection originates in the mother if she is a syphilitic before she becomes pregnant, when the poison travels through the placenta; a purely ovular infection has not been demonstrated.

The transmission of the virus from infection during pregnancy is exceptional, and is only possible by involvement of the maternal and fetal placenta.

If both parents are syphilitic, the disease can nevertheless be transmitted from only one parent, for the previously infected germinal cell is immune against a second infection.

The inheritance is facultative and depends upon the fact that either the spermatozoön or the ovum contain the syphilitic poison. This explains the possibility of parents who recently had syphilis giving birth to healthy children (Finger). The more recent the infection of the parents, the greater and the prompter in appearance is the infection in the child (Finger).

The poison tends gradually to lose its virulence in the parental organism, so that in an almost regular order of rotation the mother gives birth to miscarriages, premature births, full-term dead infants, children which at first live for but a short time, and later such as live a longer time, and finally, healthy children. This order may be interrupted by the birth of perfectly healthy children.

A syphilitic child which has been infected by its father cannot transmit the virus to the mother, for she became immune during pregnancy (Colles' law). (Exceptions to this rule are, however, met with.)

Clinical Symptoms.—The child is either born with the signs of syphilis, or they do not appear until after a latent period of from several days to months. The earliest phenomena are:

Snuffles.—Swelling of the nasal mucous membrane, which is accompanied at first by a dry, and later by a dirty pus-like and bloody secretion, causing a peculiar sniffing noise called the "snuffles"; not until later are râles also heard. Desiccation and maceration of the secretion causes the formation of brownish crusts or excoriations at the nasal orifices. Extension of the ulcerative process to the cartilaginous and bony portion leads to development of the saddle-nose. The coryza is the most constant, earliest, and most obstinate sign of hereditary syphilis; it may exist at birth, and outlasts, as a rule, all other signs.

Pemphigus, or Bullous Syphilid (see Plate 42).—These are soft vesicles, varying in size from a lentil to a cherry, which occur on all parts of the body, and especially on the palms of the hands and the soles of the feet. They may be present at birth or appear during the first three or four days. (For Differentiation from Pemphigus Vulgaris, see p. 166.)

Enlargement of the spleen and liver may occur as prominent signs of fetal visceral syphilis during the nursing period. An abnormal lack of weight and longitudinal growth are usually noticeable at birth. Atrophy and anemia are frequent, but not always present. The following are some of the symptoms, which do not, as a rule, appear until several weeks have elapsed:

The Skin.—*The Macular and Papular Squamous Syphilid.*—These are brownish-red, lentil- or mustard-seed-sized specks, which are especially likely to occur on the eyebrows, chin, nasolabial folds, soles, and the palms. The spots are considerably elevated and show a tendency to exfoliate and form crusts. A papular efflorescence is especially likely to occur in the anal and genital regions. Maceration of the secretion causes the development of multiple fissured and weeping excoriations at the angles

of the mouth and at the anus. In addition to these specific exanthems we also meet with eczematous and psoriasis-like varieties.

Diffuse Syphilitic Infiltration of the Skin (Hochsinger). —The skin of the palms, the soles, nates, genitalia, and folds of the groin are decidedly red, dense, thickened, and shiny, as if varnished. That of the face is tense and gives a mask-like appearance. Splitting of the stretched skin causes the formation of fatty rhagades covered with crusts, which radiate from the edge of the lips to the nose and chin; later they become converted into scar-tissue, which remains visible for many years. Paronychia follows infiltration of the matrix of the nails. The characteristic loss of hair from the scalp and eyebrows depends likewise upon a diffuse infiltration of the involved areas of the skin. The skin usually has a livid and at times dirty yellow and slightly shiny color [Trousseau's color]. The associated hemorrhages in the skin and mucous membranes (see Hemorrhagic Syphilis) are caused by a septic infection, which usually extends from the umbilicus.

Liver.—Children in whom the syphilitic symptoms are not very pronounced develop icterus in the course of two or three months, accompanied by bile in the urine and acholic stools; there is also an enlarged and resistant liver, splenic tumor, and ascites.

Kidneys.—Involvement of the kidneys is manifested (chiefly at the close of life) in the form of an ordinary acute nephritis with albumin and granular casts; or as a true hemorrhagic inflammation.

Lymph-nodes.—Swelling of the lymph-nodes, especially in the cervical, axillary, cubital, and inguinal regions, is rarely present. [Swelling of the cervical lymph-nodes occurs if there be an ulcerating lesion in the mouth, nose, etc.—Ed.]

Bones.—Painful, pale, ring-shaped swelling of one of the lower epiphyses of the humerus or femur. This is accompanied by a slight "voluntary" paralysis of the affected side, without the signs of degeneration and without involvement of the joint—Parrot's pseudoparalysis.

Syphilitic Phalangitis (Hochsinger).—This consists of a painless swelling, primarily of the first phalanges, secondarily of the middle and distal phalanges of the fingers and toes, which causes the former to assume the shape of a bottle, the latter the shape of a tenpin. There is no tendency for the swelling to rupture externally or to involve the soft parts. Both affections of the bones are

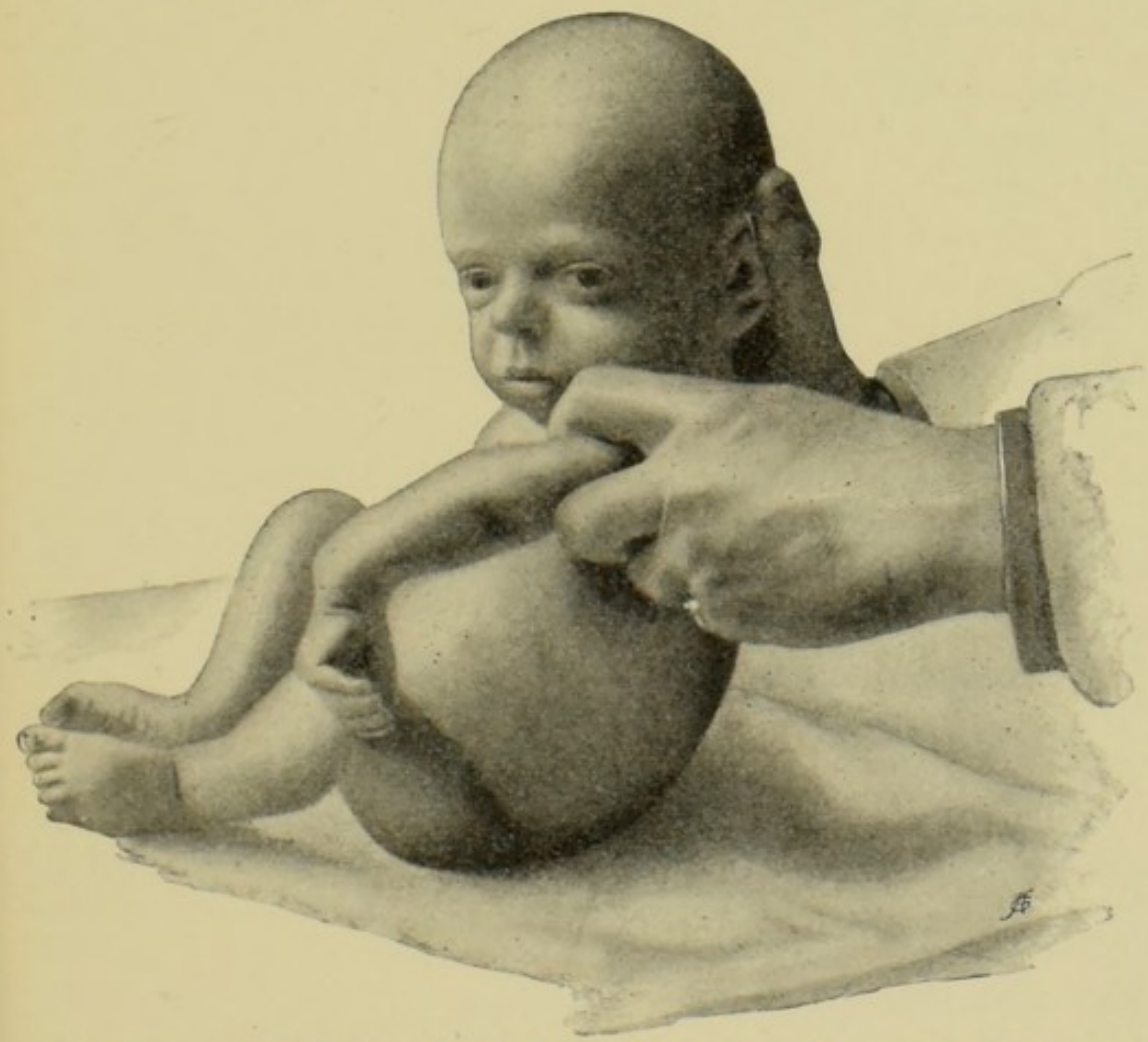


FIG. 55.—Parrot's pseudoparalysis of the left forearm and the hand in hereditary syphilis. Child four months old. Specific loss of hair from the scalp, eyebrows, and eyelashes. (A maculopapular exanthem had disappeared, but the splenic tumor and coryza still persisted.) (Clinic of von Ranke, Munich.)

caused by an osseous inflammation which has spread from the zone between the cartilage and bone (see Pathologic Anatomy). Syphilitic paralyses without involvement of the bones are due to a gumma or arteritis of the brain.

These processes present the symptoms of paralysis of

PLATE II

Papular Rash of the Nates and Labia Majora in Hereditary Syphilis.—This girl (seven and a half months old) presents on the skin of the labia majora and in the region of the anus a large number of efflorescent papules. These are twice the size of a lentil, pale blue in color, umbilicated, and excrete a slight discharge. This rash is accompanied by a non-specific eczema with dark red, elevated, and vesicular papules, which are also efflorescent (Eczema erythematosum, papulosum, and vesiculosum). Other symptoms manifested in this case were a pale-yellow, slightly shiny skin, splenic tumor, and saddle-nose. Cure in six weeks by means of iodid of mercury.

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the upper and lower plexuses of nerves, and also probably play an important part in many cases of Little's disease, polio-encephalitis, idiocy, congenital hydrocephalus, etc. Occasionally specific infiltrations and ulcers are found in the larynx, intestines, testes, and various other organs.

Syphilis Tarda.—Children whose parents have undoubtedly had syphilis occasionally develop, after the fifth year of life, certain phenomena which conform exactly with the tertiary manifestations of acquired syphilis. It has not yet been determined whether we have to do in that case with delayed manifestations of a case of true hereditary syphilis, with the continuation of an overlooked early syphilis, or with the tertiary stage of syphilis acquired in early life.

Chief Symptoms.—Periostitis of the hyperplastic, gummatous, and ulcerative type. As a result of this process we have chiefly a painful scabbard-like swelling of the tibia and the formation of the saddle-nose. A torpid, usually symmetric swelling of the knee-joints, which causes ankylosis. Gummata occur in the skin of the face and on the legs, arranged in groups, which heal slowly; also in the mucous membranes, especially in the mouth, where they undergo radiating cicatrization; perforation of the palate; cicatricial stenoses in the larynx.

Indolent swellings of the lymph-nodes of the cervical, axillary, cubital, and inguinal regions. There is frequently considerable swelling of the liver and spleen, and, notwithstanding the absence of other pronounced symp-



toms, the patient frequently suffers from a contracted kidney.

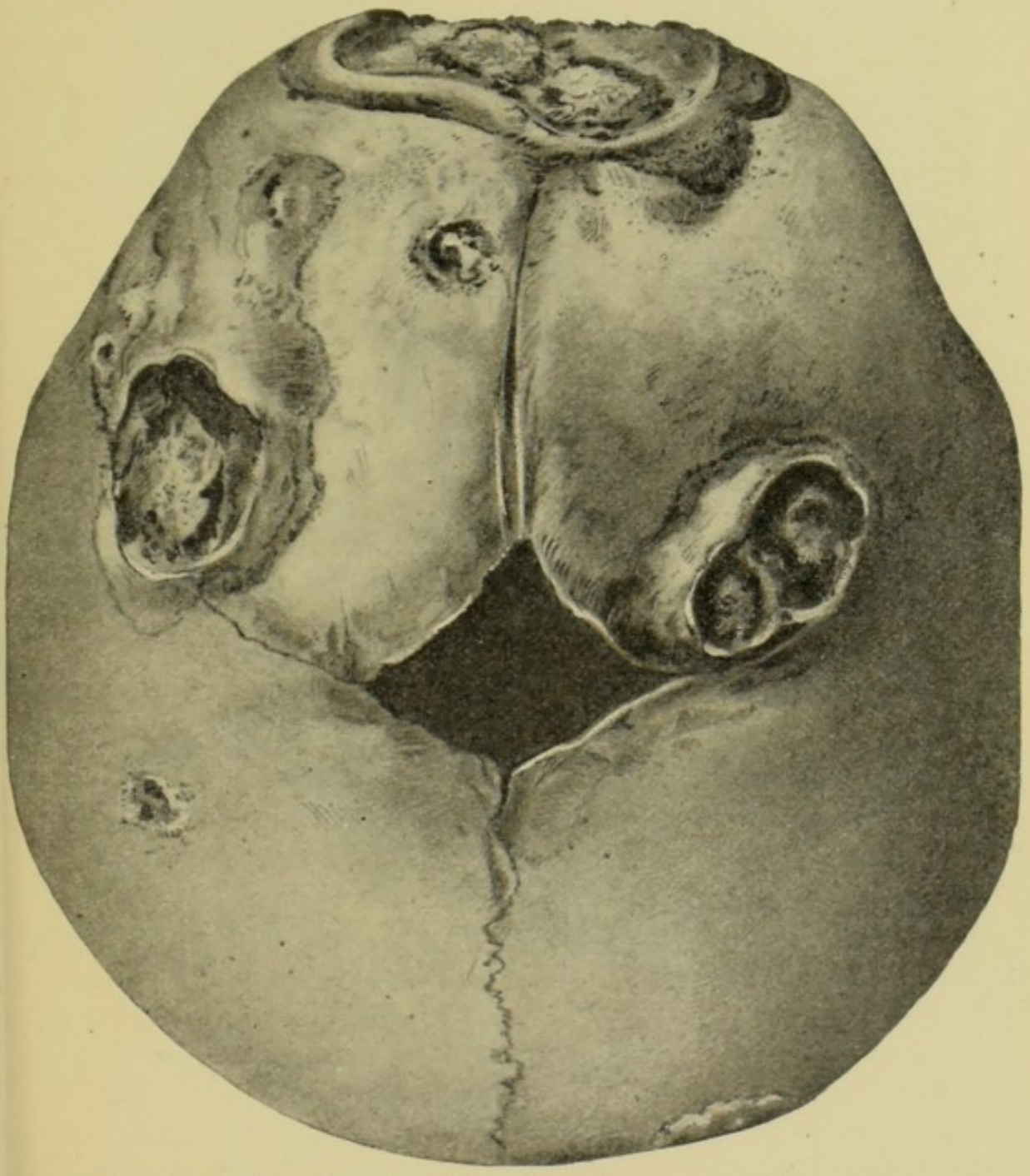


FIG. 56.—Crater-like ulceration of the bone brought on by gummatous and ulcerating periostitis and osteitis; congenital syphilis (relapse). Child one and a half years old. (Preparation from the Munich Pathologic Institute.)

Involvement of the Nervous System.—Headache, various forms of paralysis, infantile tabes, progressive paralysis, idiocy, etc., are caused by chronic endarteritis with local

PLATE 12

FIG. 1. **Diphtheria of the Uvula.**—Enlarged 300 times. 1. Swollen epithelium. 2. Vesicular spaces in epithelium. 3. Leukocytes. 4. Fibrin. 5. Nuclei of the destroyed epithelium in fibrin. (From Dürck, *Atlas of General Pathologic Histology.*)

FIG. 2. **Syphilitic Infiltration of the Liver in a Congenitally Syphilitic Eight-months' Old Fetus, which was Dead when Born.**—Enlarged 50 times. Beginning maceration. 1. Foci of small-cell infiltration, which are beginning to form a gumma. 2. Proliferation of hepatic parenchyma cells. 3. Thickening of the capsule of Glisson. 4. Enlarged bile-ducts.

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areas of softening, chronic meningitis, and cerebral gumma.

Of the so-called "Hutchinson's triad"—interstitial keratitis, central deafness, and peculiar deformity of the teeth—only the first is of pathognomonic value. The second is rare and occurs also in other affections; likewise the third, the median excavation of the upper inner permanent incisor teeth.

Recurrence.—In about one-third of the cases apparent cure is followed within the first four years by one or several relapses of this disease. The recurrence represents either a mild form of the first attack with maculopapular exanthem, coryza, rhagades, etc., or multiple weeping mucous papules, broad condylomata at the anus, genitalia, and oral cavity—the condylomatous stage of hereditary syphilis. The latter is accompanied by deep gummatous disease of the bones, the skin of the extremities, and of the skull; the liver, spleen, kidneys, pancreas, and testes; and endarteritic processes in the brain and spinal cord, together with polio-encephalitic, hemiplegic, and epileptic manifestations; iritis and localized chorioretinitis. (See Haab, *Atlas of Ophthalmoscopy*, Plates 39–41.)

Morbid Anatomy.—Macroscopic changes do not set in until after the fourth fetal month, and from the fourth to the sixth month the chief changes consist in an osteochondritis and an enlarged spleen. The whole chain of the remaining manifestations do not appear until later. The post-mortem examination is not infrequently nega-

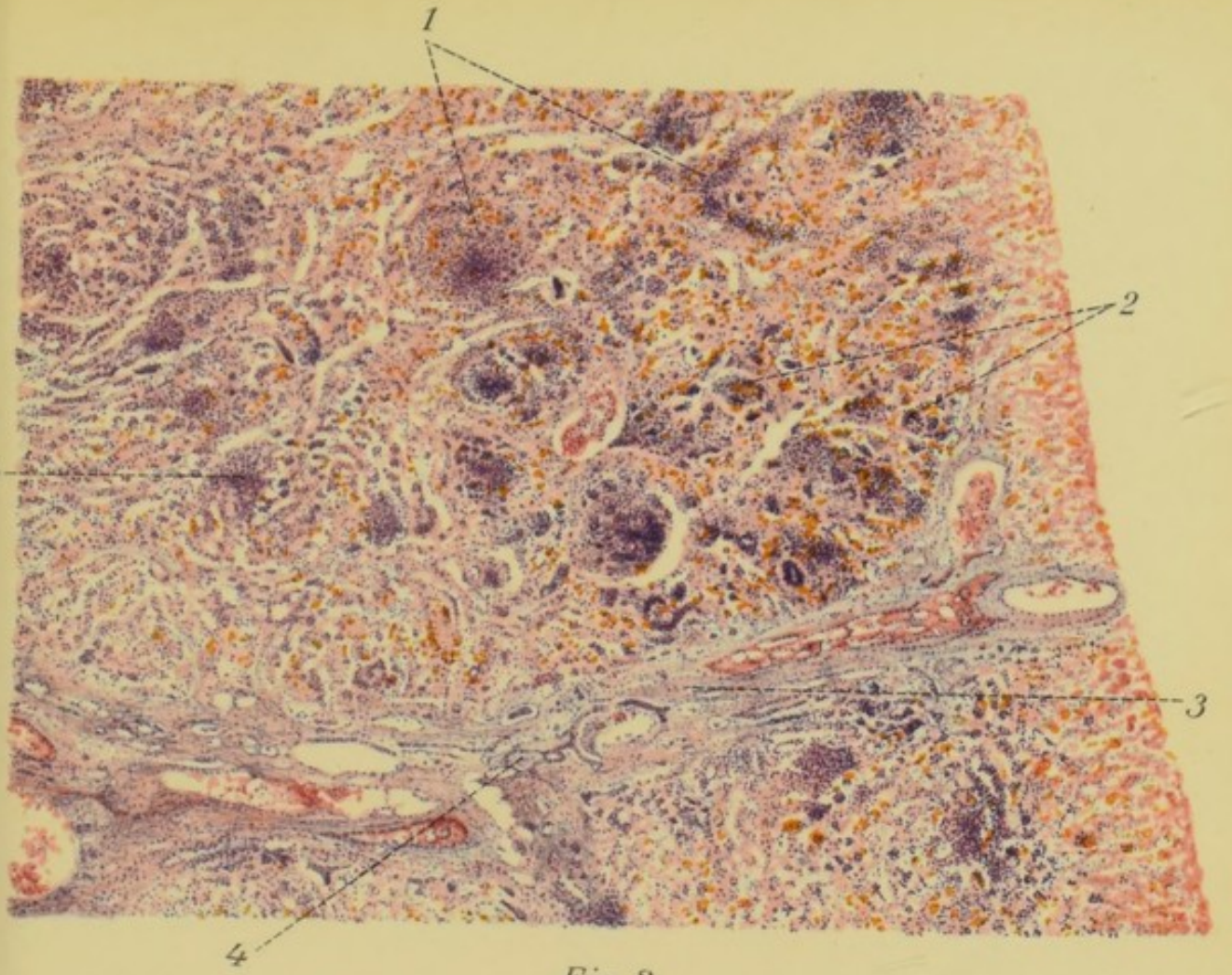


Fig. 2.

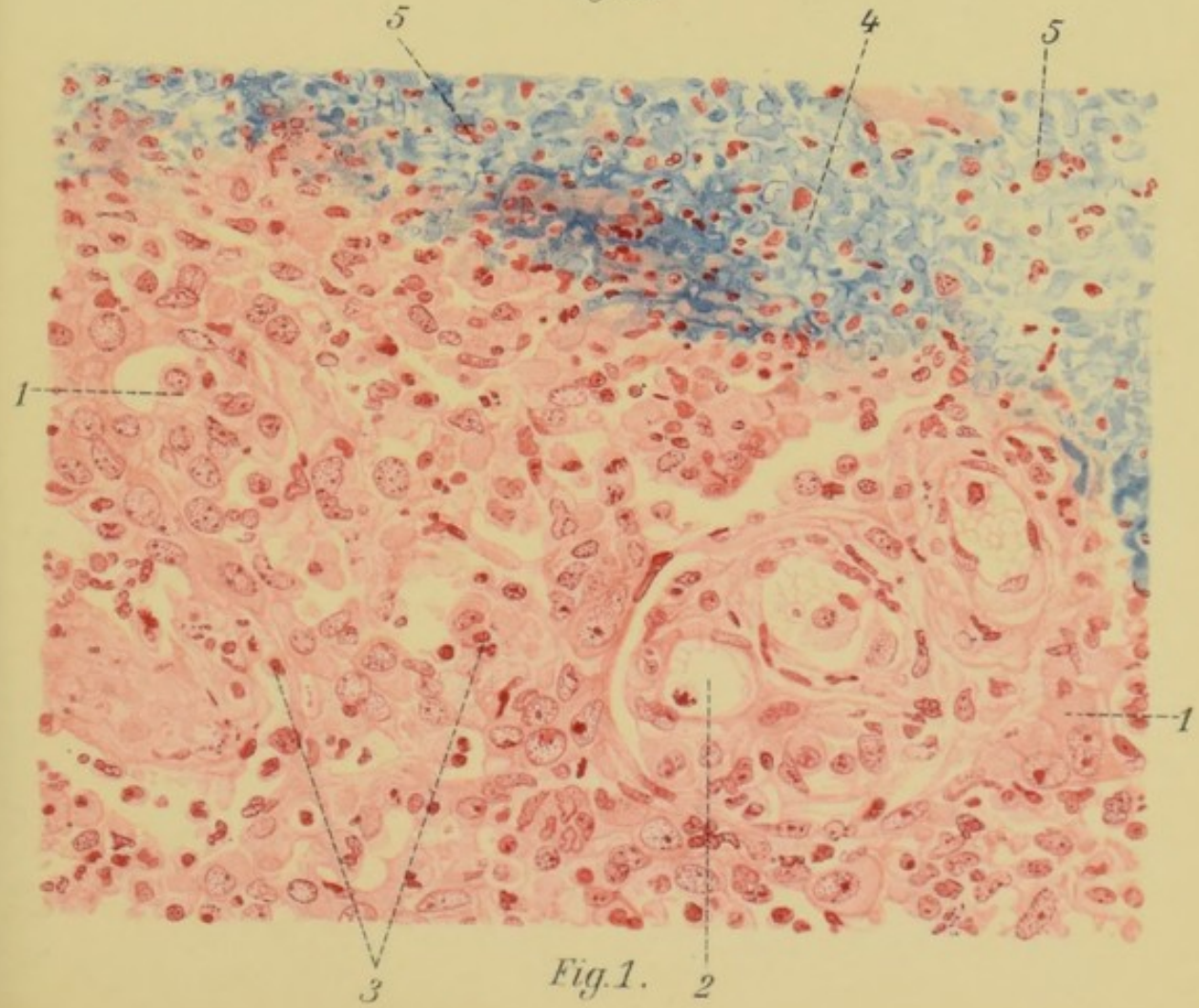


Fig. 1.

tive. The macerated "sanguinolent" condition of the fetal corpse presents in itself no characteristic change. Even children who during life showed the undoubted presence of syphilis, frequently fail after death to present any of the positive symptoms of that disease.

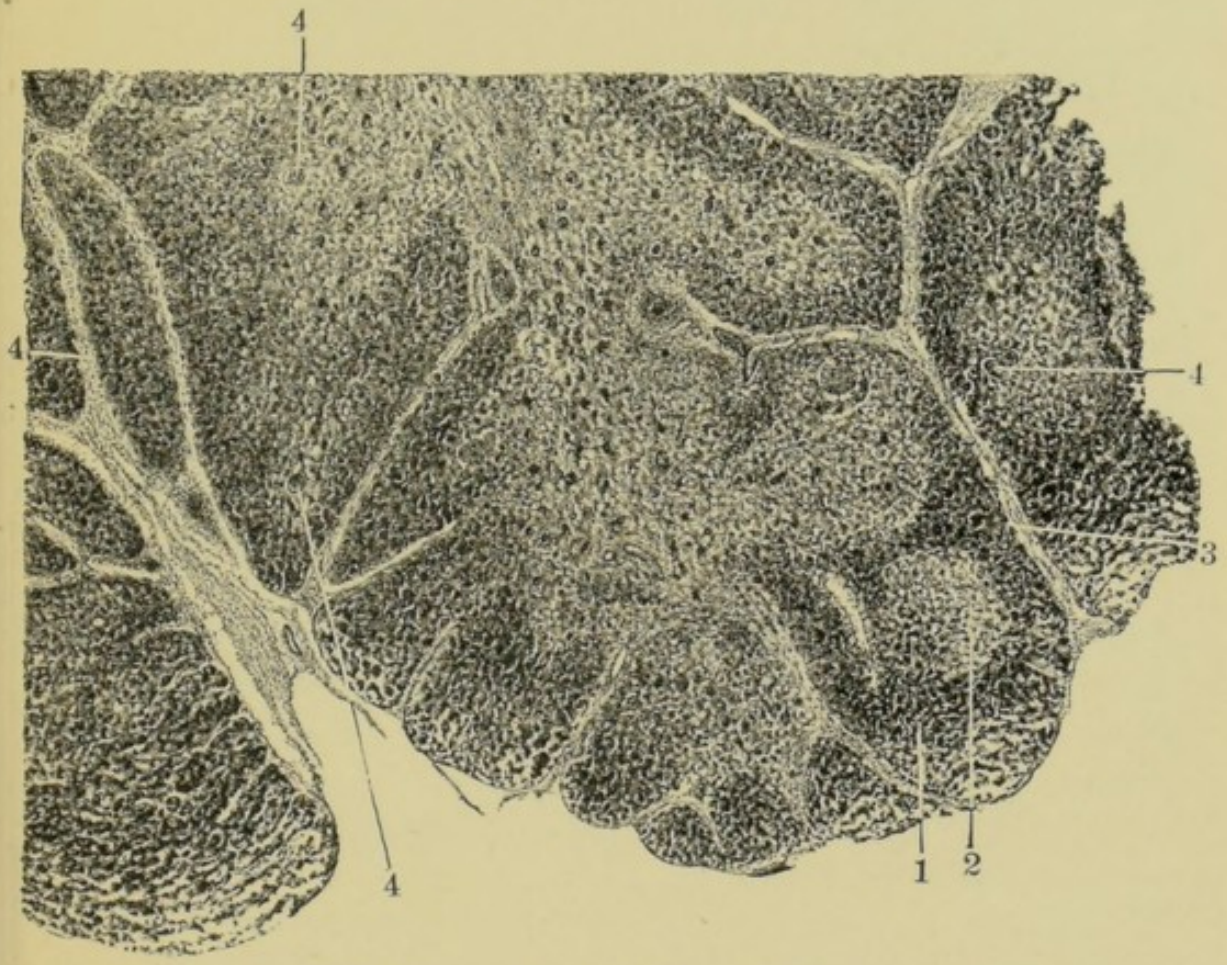


FIG. 57.—Normal thymus of a healthy full-term child which died during birth. The organ is richly supplied with cellular tissue, but sparsely with thin connective-tissue septa. Enlarged 52 times. 1. Peripheral substance. 2. Marrow. 3. Connective-tissue septum. 4. Hassal's corpuscles.

The most important *gross anatomic changes* are: Enlargement, induration, and increase in weight of the large abdominal glands; thus the spleen weighs $\frac{1}{200}$ to $\frac{1}{50}$ of the body weight in comparison with $\frac{1}{360}$ normally; the liver, $\frac{1}{16}$ instead of $\frac{1}{21}$, and the kidney, $\frac{1}{86}$ instead of $\frac{1}{123}$. The weights only hold true for fetal syphilis and that of the early nursing period. Later the organs undergo atrophy under the influence of the cachexia, and hence lose in weight. An exception to this is the thymus,

PLATE 13

Syphilitic Changes in the Kidneys of a Stillborn Congenital Syphilitic (eight to nine months).—Magnified 42 times. Beginning maceration. 1. Thickened and partly obliterated peripheral arteries, whose walls and surrounding tissues show small-cell infiltration. 2. Perivascular small-cell infiltration. 3. The cortex is increased in width and undergoing retrogressive development. 4. Young glomeruli.

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which in fetal syphilis weighs almost constantly less than normal, on an average $\frac{1}{510}$ of the body weight instead of the normal $\frac{1}{235}$. Also note that aside from an increase in weight the liver is more elastic than normal (a piece held between the fingers may be snapped away like a cherry stone); it is dark in appearance, and on cross-section the surface varies from a brownish-violet to a slightly shiny yellow color; the capsule is thickened and opaque.

Syphilitic osteochondritis is the most frequent of the earliest and the longest in duration of the symptoms of congenital syphilis.

Gummata varying in size from a miliary tubercle to that of a hazel-nut, as well as overgrowth of connective tissue and gross cirrhotic processes, are found in all the organs, especially the liver, spleen, lung (interstitial pneumonia), pancreas, thymus, and likewise in the rose-red discolored placenta.

Deserving special mention is the so-called "white pneumonia" of a syphilitic fetus, in which the enlarged firm lung appears grayish white on cross-section; it is generally combined with the interstitial pneumonia. Another condition to be referred to is a rare cherry-sized abscess of the thymus (so called by Dubois), which may be easily mistaken for the normal tissue softening in the fetal thymus.

The histologic changes of hereditary syphilis are uniform in so far as the chief alterations are alike in every organ.

Circumscribed small-cell infiltration, especially in the neighborhood of the large blood-vessels, which has a tendency to undergo central necrosis. This miliary syphiloma may be regarded as the beginning stage of a gumma (see Plate 12, Fig. 2).


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3
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Diffuse cellular infiltration, consisting of irregularly distributed round cells throughout the whole parenchyma.

Diffuse and *circumscribed connective-tissue proliferation*, representing the beginning of cirrhosis (see Plate 12, Fig. 2).

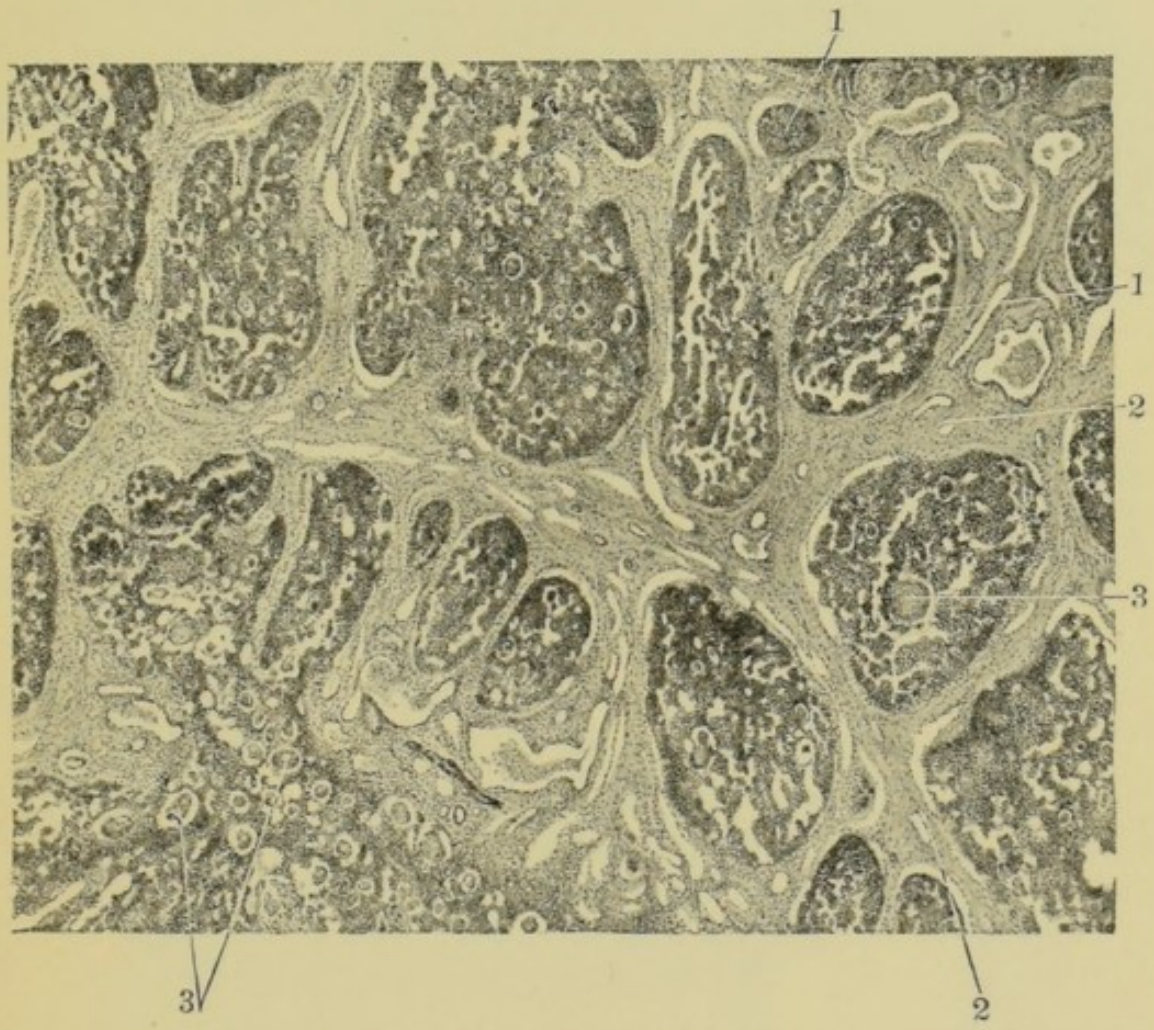


FIG. 58.—The thymus gland in heredosyphilis of an almost full-term dead child. The connective-tissue septa are more numerous and thickened; the glandular structure is shriveled and persists only in certain areas in small foci. The corpuscles of Hassal lie closely crowded together and are notably large. Magnified 52 times. 1. Glandular substance. 2. Connective-tissue septa. 3. Corpuscles of Hassal.

Abnormal epithelial proliferation—collections of epithelium in the liver and, rarely, in the lungs and kidneys.

Individual organs are especially characterized by certain peculiarities: The kidneys of the fetus show cellular infiltration of the walls and neighboring structures

of the smallest peripheral arteries, enlargement of the epithelial marginal zone, and decrease in the size of the glomeruli (see Plate 13); nurslings suffer from acute degenerative nephritis. The spleen undergoes infiltration of the middle and larger sized blood-vessel sheaths. In the thymus there is a thickening of the interlobular septa, with compression and shrinking of the acini (see Fig. 58). The alveoli of the lungs, which are the seat

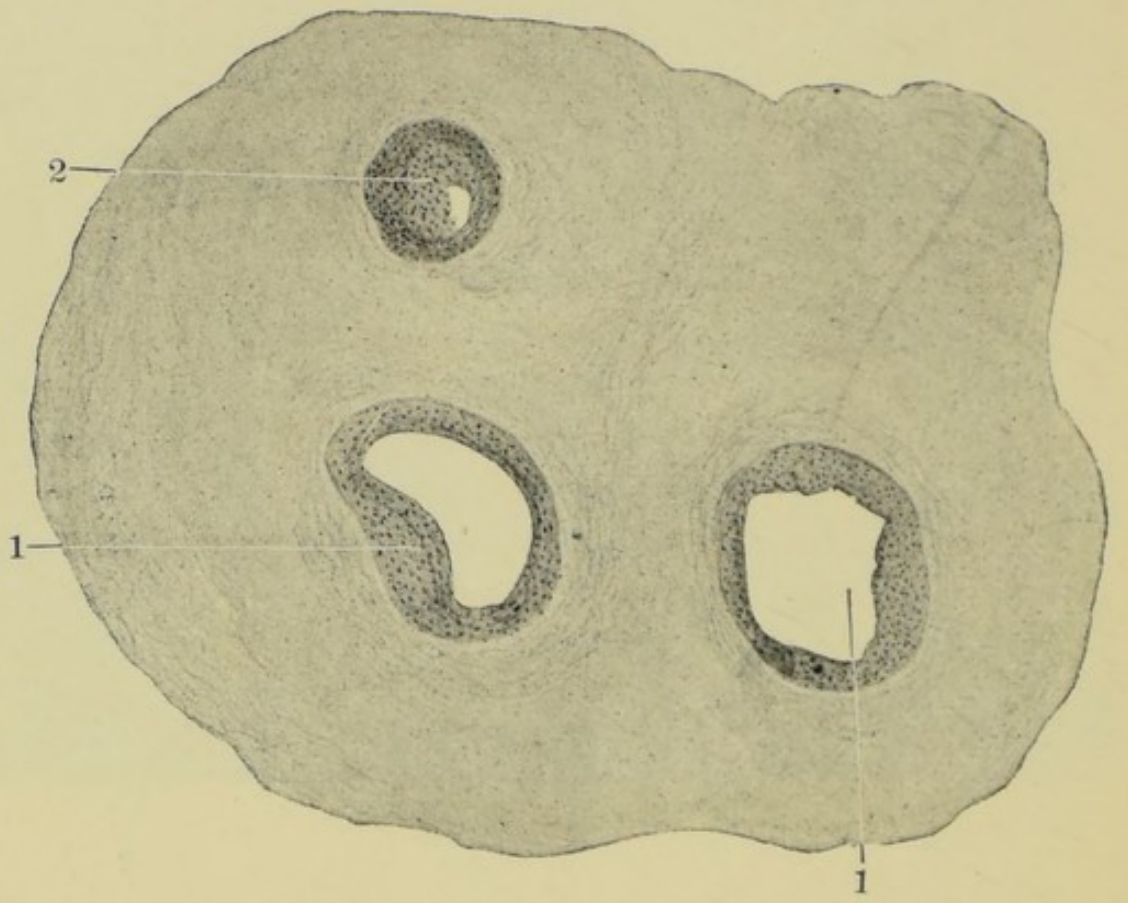


FIG. 59.—Umbilical cord of a healthy newborn child, showing unequal thickening of the vessel walls. Magnified 11 times. 1. Arteries. 2. Vein with thrombus.

of white pneumonia, are filled with desquamated and fatty epithelium. The umbilical cord is infiltrated and the venous and arterial vessel walls are thickened almost sufficiently to cause obliteration.

Syphilitic osteochondritis (Wegner) (see Plate 12, Fig. 1), which attacks chiefly the epiphyses of the long bones, is marked by enlargement of the provisional zone of calcification, and a serrated border between the cartilage

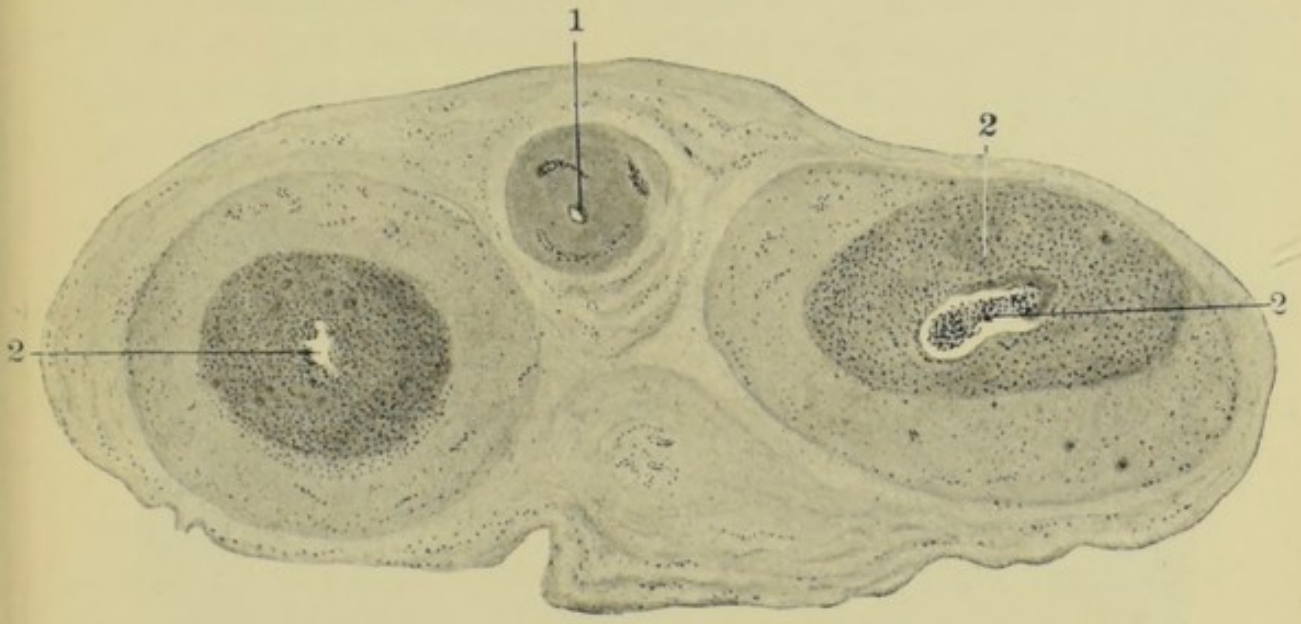


FIG. 60.—Umbilical cord of a full-term syphilitic child which lived to be five days old. Arteritis and phlebitis obliterans. The walls of all three vessels are considerably thickened; proliferation of the intima. The lumen of the vein is much reduced in size; pus in one of the arteries. Magnified 11 times. 1. Vein. 2. Arteries.

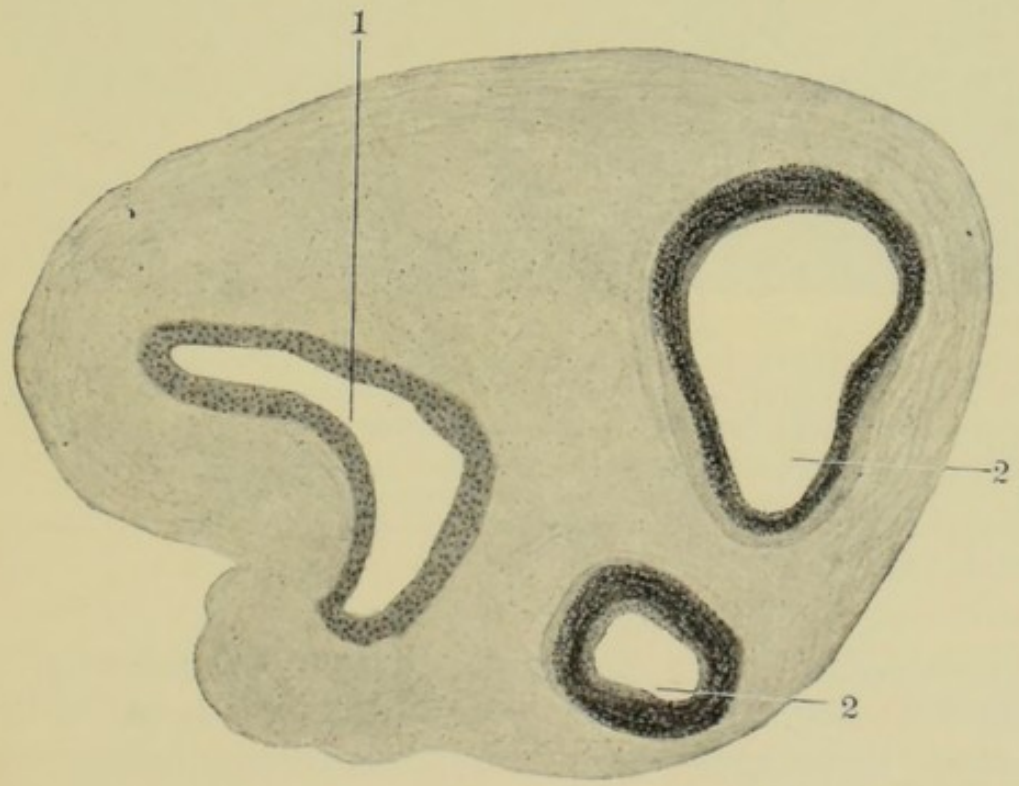


FIG. 61.—Umbilical cord of a syphilitic newborn infant. Small-cell infiltration of the media of both arteries. Magnified 8 times. 1. Vein. 2. Arteries.

and bone; the notches of this border when palpated are felt to be hard and brittle. The bluish and swollen cartilaginous zone of proliferation is diminished in size. Necrosis rapidly sets in, and finally there is inflammation, softening, and sequestration of the calcified and insufficiently nourished tissue, with resulting separation of the epiphysis.

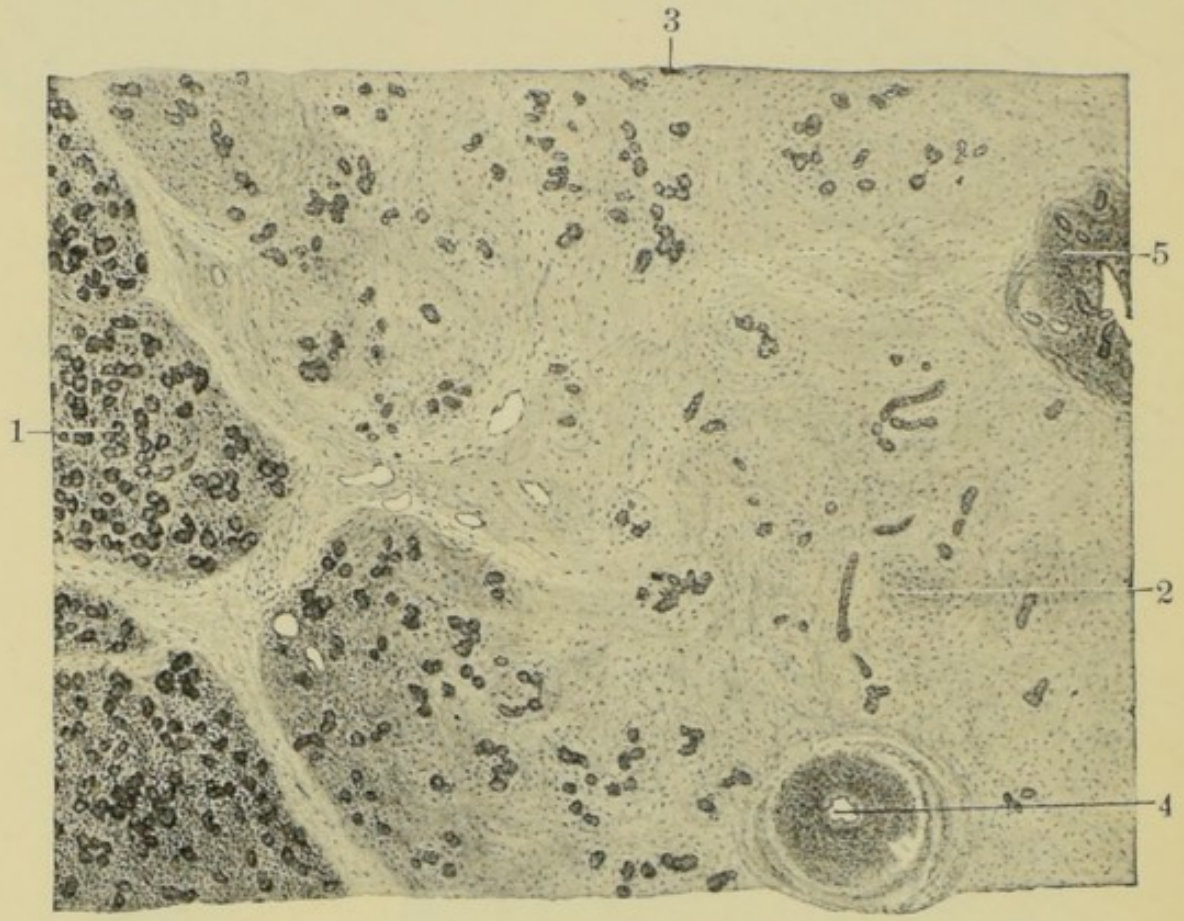


FIG. 62.—Syphilitic interstitial pancreatitis in a dead-born infant from seven to eight months of age. Enormous growth of interstitial connective tissue; inflammatory thickening of the walls of the blood-vessels and excretory ducts. Magnified 25 times. 1. Glandular structure, with beginning proliferation of interstitial tissue. 2. Proliferating interstitial tissue. 3. Rudimentary glandular substance. 4. Thickened arteries. 5. Thickened excretory ducts.

Histologically the process consists of a hardening (see Plate 14, Fig. 2, and Plate 6) of all the bone-building tissues (Heubner). The calcification progresses in an irregular manner into the cartilage, involving not only the cartilage but also the columns of cartilaginous cells and the cells of the bone-marrow. Inasmuch as the portion

of the medullary space which lies next to the epiphysis is filled with granulation tissue in which osteoblasts are absent, there is no deposit of osteoid tissue on the calcified bony trabeculæ. Temporary calcified cartilage and medullary spaces, filled with degenerated granulation tissue, are therefore arranged atypically side by side. In place of permanent bone there is a development of extensively calcified cartilage; this is more easily fractured, especially in the shreath, where it is interrupted by medullary spaces.

Here also occurs the epiphyseal separation. Healing depends upon the fact that the medullary spaces are capable of forming osteoblasts; as a result, permanent bone is built up and the calcified cartilage disappears.

Osteochondritis and Rachitis.—In both conditions an interference with bony development is the cause, only in rachitis there is a defect in the development of inorganic —and in syphilis of organic—constituents of bone. That is, in rachitis there is an insufficient deposition of calcium salts with the undisturbed formation of osteoid tissue, while in syphilis there is no interference with the excretion of lime, but an insufficient development of young bone tissue.

Diagnosis.—In children born dead, without pronounced symptoms of syphilis, the detection of osteochondritis and increased body weight is of significance. Microscopic examination of the kidney for the presence of perivascular infiltration is recommended because maceration of that organ is late in development; likewise examine the thymus. Infiltration must be differentiated from the physiologically rich supply of cells in youthful tissue, especially in the liver, kidneys, and lungs. Care must be observed in the preservation of macerated preparations, which are stained with difficulty.

In living children note the chief symptoms: Snuffles, pemphigus, enlargement of liver and spleen, and insufficient body weight as early manifestations; later maculopapular exanthems, diffuse infiltration of the skin, and rhagades of the nose, mouth, and anus. Of signifi-

PLATE 14

FIG. 1. **Congenital Syphilis of the Intestines.**—Cross-section of the small intestine of a seven-months'-old syphilitic child which was dead when born. Magnified 60 times. A circumscribed specific infiltration, which may even be detected macroscopically, lies between the mucosa and submucosa; it has resulted in loosening of the former. 1. Thickened peritoneum. 2. Muscular coat. 3. Submucosa. 4. Syphilitic infiltration. 5. Loosened mucosa. 6. Normal mucosa. 7. Intestinal contents.

FIG. 2. **Syphilitic Osteochondritis.**—Longitudinal section of the distal epiphysis of the femur of a case of congenital syphilis in a seven-months'-old infant born dead. Magnified 6 times. The border between the bone and cartilage is serrated. The zone of temporary calcification of the cartilaginous ground substance is wider than normal, and extends into the diaphysis as well as into the cartilage. 1. Resting cartilage. 2. Cartilage cells arranged in columns which have been compressed between the calcified cartilage tissue and crowded proliferating cartilage. 3. Large vesicular cartilage cells, in process of beginning ossification. 4. Calcified cartilage ground substance. 5. Primary medullary space, filled with granulation tissue. 6. Calcified cartilage surrounded by granulation tissue.

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cance in doubtful cases, as during the intervals between recurrences, are numerous deaths in the family, lack of body weight, radiating scars on the lips, splenic tumor, yellowish, dirty skin, and swollen cubital lymph-nodes.

Following are the conditions for which syphilis might be mistaken: *Pemphigus vulgaris.* Development after the first week of large soft vesicles, which do not involve the palms nor the soles. Snuffles are absent. The *physiologic desquamation* and *paronychia* on the fingers and toes appear in from two to three weeks, and the other symptoms are absent. *Simple coryza,* a thin abundant secretion. *Congenital hypertrophy of the nasal mucous membrane, adenoid vegetations.* Other syphilitic symptoms are not present. *The glossy reddening of the soles of atrophic children,* the denseness and desquamation of diffuse specific plantar infiltration. *Papular eczema of the anus,* the palms and the plantar surfaces of the feet remain free; also found in the neighborhood of true eczematous parts; the other specific symptoms are absent. *Spina ventosa* (in comparison with specific phalangitis), children older in age, rarely multiple on one hand, fail to localize on the first phalanges, skin also involved, tendency to external rupture, of spheric and cylindric shape.

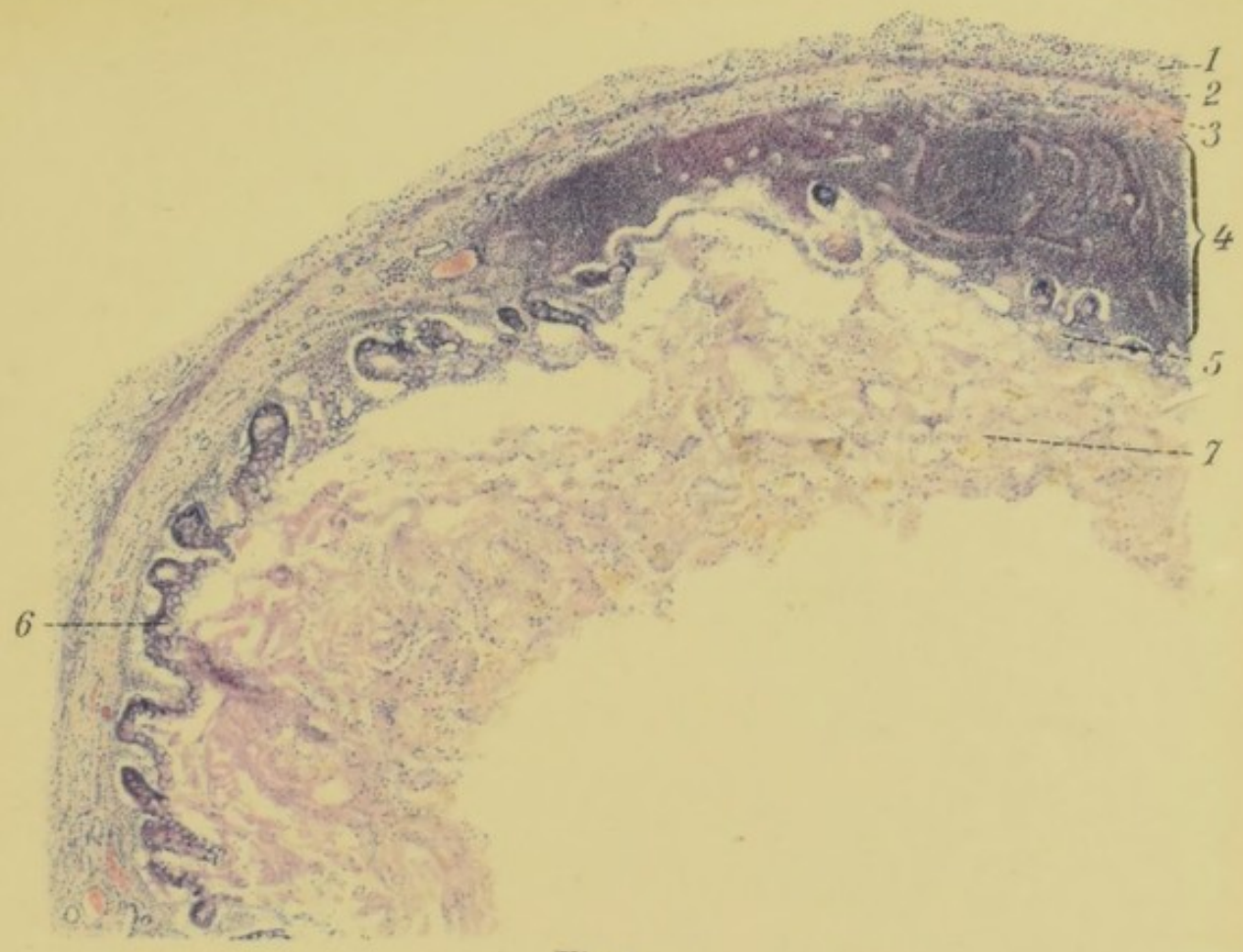


Fig. 1.



Fig. 2.

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Prognosis.—The severe cases die *in utero*, and only the mild forms are born. The prognosis is more favorable the later the symptoms develop after birth, and the slower they follow each other. The outlook is bad in the presence of pemphigus and visceral syphilis which originated during fetal life. Relapses occur in about 30 per cent. of all cases, and almost exclusively within the first years of life. A weakening of the general system cannot be prevented even in the most favorable forms. Death is due to marasmus, septic, enteritic, nephritic, or pneumonic processes. Syphilis tarda usually terminates favorably. Breast-fed children have better prospects than bottle-fed children, yet careful artificial feeding may also offer good results.

Treatment: Prophylaxis.—Marriage is not permissible until at least four years have elapsed since the infection and two years after the last relapse, during which time a thorough course of treatment must have been instituted. When syphilis has been manifestly acquired, the parent should receive energetic treatment. When a mother has been infected during pregnancy, the general treatment should be combined with the local use of mercury by means of vaginal suppositories.

Nourishment.—Whenever possible, feeding should be maintained by means of the mother's milk, at least throughout the acute stage. According to Colles' law the mother is not endangered herself. No wet-nurse should be employed even in a doubtful case. If necessary, milk obtained artificially from the wet-nurse may be used. If human milk is not obtainable, then resort to careful individual artificial feeding, including artificial preparations.

Specific Treatment with Mercury and Iodin: Internally.—Protoiodid of mercury, 0.005 to 0.01 gm. twice daily; or in combination with saccharated ferri carbonate, 0.1 gm.; calomel in like doses, or with opium, 0.003 gm.

Externally.—Sublimate baths, 1.0 to 1.5 gm. to a bath. Caution should be used in case of excoriated skin. Welander's sacks (6 to 10 gm. of mercury and chalk mixture smeared on the woolen side of a piece of lint 20 × 40

PLATE 15

Congenital Tuberculosis.—In the atelectatic lungs of a child which died a few hours after birth was found a single cherry-sized caseous focus surrounded by a connective-tissue capsule; tubercle bacilli were found in this focus. The connective tissue in the neighborhood of the focus was distinctly increased. Other tuberculous processes could not be found. Such a focus might have remained latent throughout life, or it might have been disseminated through an infectious disease, traumatism, etc., and a general tuberculosis caused. (Preparation in the Munich Pathologic Institute.)

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cm. [8 × 16 in.], which is folded, sewed together, and tied in place), which are to be renewed, as in the case of the commercial "mercolint aprons," after from four to six days. Wrap all four extremities at intervals of six days with mercury plasters.

Locally.—Apply calomel to condylomata; 3 per cent. silver nitrate or 10 per cent. chromic acid solution to rhagades; red precipitate or 1 per cent. silver solution to nose. Duration of treatment is about six weeks, in any case for fourteen days after the disappearance of the last symptom. In case of gumma or retarded syphilis give sodium iodid, 1.0 to 2.0 gm. per day, until the disease is influenced. To combat cachexia give arsenic, levico, etc.

TUBERCULOSIS

Frequency.—Next to disease of the digestive tract, tuberculosis is the most frequent cause of death in children (13 to 20 per cent.); nearly 30 per cent. of all children possess latent tuberculosis. Of all deaths due to tuberculosis, 30 per cent. are in children.

Transmission.—Tuberculosis is either congenital or acquired.

Congenital tuberculosis is met with but rarely, and is transmitted usually from a mother who is suffering from a severe form of tuberculosis by way of the placenta, which is also, as a rule, diseased.[1] Transmission of the

[1] The bacilli enter the fetal blood-vessels or the amniotic sac, where the fetus may swallow them with the liquor amnii (Schmorl)—congenital gastro-intestinal tuberculosis.



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tubercle bacillus by means of the human ovum or the spermatozoön has never been determined; the first is possible when tuberculous peritonitis exists, the latter, however, highly improbable (Gärtner).[1] Congenital tuberculosis represents either a general infection of the fetal body shortly after birth (tuberculous bacillemia), or it assumes the form of tuberculous foci disseminated in the organs.

Acquired tuberculosis is the usual form, even in very young children. Caseous foci are rarely seen before the third or fourth month, yet this period of life favors the development of such foci. The infection is transmitted products containing tubercle bacilli, either from the parents' or the child's surroundings. The infection enters the body as follows:

By *inhalation of dried tuberculous sputum* or of fine drops of sputum which have been coughed into the air; the latter occurs only when a person is constantly near the patient. The primary affection lies in the lungs themselves, especially if a bronchitis or bronchopneumonia already exists, or in the peribronchial and mediastinal glands, in which case the bacilli travel through the lungs without injuring them. Occasionally the first deposition of the bacilli occurs in the pharyngeal or palatine tonsils. Inhalation represents the commonest mode of entrance for tuberculosis.

By the *introduction of material containing tubercle bacilli* into the gastro-intestinal tract, by placing utensils, toys, and dirty fingers (tubercle bacilli have been demonstrated in the dirt of the finger-nails) in the mouth, and by the ingestion of raw milk or meat from tuberculous cows. Here also the bacilli may pass through the intestinal walls, and collect in the regional lymph-nodes of the mesentery and peritoneum. It is difficult to determine whether the disease is due purely to the ingestion of infected food—for tuberculosis of the intestines and mesen-

[1] The number of tubercle bacilli in the spermatic fluid is very insignificant in comparison with the enormous number of spermatozoa, and no spermatozoa have ever been found to contain tubercle bacilli.

teric nodes may be secondary to infection through the lymph-channels from bronchial nodes—or to the swallowing of tuberculous sputum.

From the *mucous membrane* of the mouth, pharynx, nose, and genitals, as well as the external skin, when these tissues are damaged or even when they remain uninjured, provided they come in intimate contact with the bacteria (Cornet). The lips, tonsils, and carious teeth are especially prone to admit the infection.

PECULIARITIES OF TUBERCULOSIS IN CHILDREN

Tuberculosis of childhood is nearly always a general disease which involves numerous organs and occurs rarely before the third month, reaching its maximum in from two to four years. Characteristic of infantile tuberculosis is the early and constant involvement of the lymph-nodes, especially the peribronchial, and also the cervical, abdominal, and inguinal nodes. Disease of the lymph-nodes is frequently the only manifestation of tuberculosis—*latent tuberculosis*. In nurslings the lesions usually met with are tuberculous disease of the bronchial nodes, with caseous pneumonia of the neighboring pulmonary tissue or a generalized tuberculosis. The following varied manifestatians of tuberculosis do not occur until later in childhood : Affections of the bone-marrow, serous membranes, meninges, peritoneum, pleura, tendon-sheaths, and joints. As children grow older the symptoms assume the character of adult tuberculosis.

Predisposition.—This is either congenital, on account of a weak constitution, consisting of certain anatomic peculiarities of the skin, mucous membranes, and lymphatics, which are inherited from parents suffering from some dyscrasia, or it is acquired through unhealthy conditions of life, poor nourishment, or wasting diseases. Latent tuberculosis is frequently made active and manifest by febrile diseases, especially measles, whooping-cough, influenza, and inflammation of the lungs.

Paths of Dissemination.—The inhaled tubercle bacillus is deposited on the pharyngeal or palatine tonsils, which

are primarily infected, or penetrated to reach the regional lymph-nodes; or it travels to the finer bronchial tubes, where it begins the primary affection with a caseating bronchitis, peribronchitis, and bronchopneumonia; or even more frequently it pierces the bronchial walls and reaches the lymph-nodes by way of the lymph-channels. Here it causes the formation of minute tubercles, which, becoming confluent, form larger ones; these become swollen and undergo chronic inflammation, and finally, necrosis with caseation, softening, and calcification. Here under certain circumstances the process comes to a standstill—latent tuberculosis. Tuberculosis may extend from the bronchial nodes in the following ways:

By *contiguity* to the neighboring lung tissue—periglandular caseous pneumonia.

By way of the *lymph-channels* to various parts of the lungs—lymphogenic tuberculous peribronchitis—or also to distant structures (abdominal lymph-nodes, bones, and joints).

Rupture of a calcified and softened nodular focus into a bronchus. Dissemination of tuberculous material by aspiration—tuberculous caseous bronchopneumonia.

Rupture of such a focus into the esophagus—*infection of the gastro-intestinal tract* (this may also follow swallowing of infected sputum).

Rupture into the circulation, either by way of a pulmonary artery or a vessel leading to the heart (vein, artery, thoracic duct). In the first case disseminated pulmonary tuberculosis results, and—according to the position of the vessel—the whole lung or only part of it is involved; in the latter case (rupture into a vein, etc.) dissemination throughout the whole body leads to generalized tuberculosis. A particular manifestation of this form is acute miliary tuberculosis, which develops when a large amount of the infective material is discharged into the circulation at one time, or when vital organs like the basilar meninges are attacked by tuberculous meningitis. In both cases the resulting miliary foci have not sufficient time to develop into larger disseminated nodules.

Certain phenomena of tuberculosis are possibly not caused by live bacilli, but by the dead bacilli or their soluble products. Thus experimental investigation has shown that general marasmus, cold abscesses, and caseation are due to dead tubercle bacilli or their extracts.

SYMPTOMS OF GENERAL TUBERCULOSIS

Subacute and **chronic general tuberculosis** are manifested anatomically by foci in process of caseation, varying in size from a hemp-seed or lentil to a hazel-nut, which are spread throughout the lungs, spleen, kidneys, and brain (solitary tubercles), accompanied by primary older foci in the bronchial or mesenteric nodes. The clinical signs are loss of appetite, apathy, cough, sweats, and gastro-intestinal disturbances. The objective symptoms, if any are present, consist of dark circles surrounding the eyes, a slight or hectic fever which, in spite of its persistence, is unaccompanied by febrile disturbances; swelling of the small subcutaneous lymph-nodes (micropolyadenopathy); chronic bronchitis; indications of the involvement of the bronchial lymph-nodes; pneumonic infiltration; enlargement of liver and spleen; nodules in the skin, varying in size, or dirty grayish-brown elevated specks which are dry and subdivided; and, more important than all, a progressive emaciation.

Other significant manifestations are: A caries encircling the teeth (Neumann); small semisoft nodules on the face which resemble incompletely developed furuncles, without a tendency either to suppuration or to resolution (Heubner); considerable growth of hair between the scapulæ (Heubner).

Acute general tuberculosis (miliary tuberculosis) is characterized anatomically by minute gray nodules in nearly all of the organs of the chest and abdomen, the meninges, etc. It is accompanied by marked constitutional disturbances, a high fever, diarrhea, meteorism, splenic tumor, slight cyanosis, dyspnea, and fremitus over both lungs without pronounced pneumonic symptoms. If the brain

coverings are also involved, meningitis predominates the disease picture from beginning to end.

Diagnosis.—Only an approximate diagnosis can be made in case of latent glandular tuberculosis and chronic general tuberculosis. The detection of the tubercle bacilli in young children is difficult, even in the mucus which is removed from the mouth, for the absence of ulcerative processes hinders the appearance of the bacilli in the expectoration. The tuberculin-test presents a harmless and sure method for diagnosis (Schlossmann). [This last statement is not generally accepted. There are a sufficient number of cases on record where an active tuberculosis has been lighted up from a latent one by the injection of tuberculin. Enough so to make clinicians cautious in the use of this test.—Ed.]

TUBERCULOSIS OF THE BRONCHIAL NODES

When tuberculosis of the bronchial nodes is well developed, a symptom-complex results, which is more likely to be characterized by the appearances of a constitutional disease than by pathognomonic local symptoms.

Morbid Anatomy.—Enlargement of the normal nodes lying at the bifurcation of the bronchi and behind the sternum, to a size varying between that of a bean and a walnut; the nodes are joined into clumps. In the different nodes the following individual stages of the tuberculous process may be recognized side by side: Building of tubercles, infiltration, caseation, softening, calcification, and connective-tissue induration.

Symptoms.—A peevish or apathetic disposition; pallor; arrested or gradual loss of body weight, without any real disturbance of the appetite and the intestinal functions; inconstant fever; slight dyspnea (without any evident pulmonary lesion). Enlargement and induration of the cervical nodes, and especially the supraclavicular ones, which join under the clavicles and form a garland of nodes, from which extension into the thoracic cavity may naturally be expected. Small areas of dulness are found at one side of the sternum, and at the sternal end

of the first and second intercostal spaces, and in severe cases also posteriorly between the scapulæ; the respiratory murmur is exaggerated in these areas.

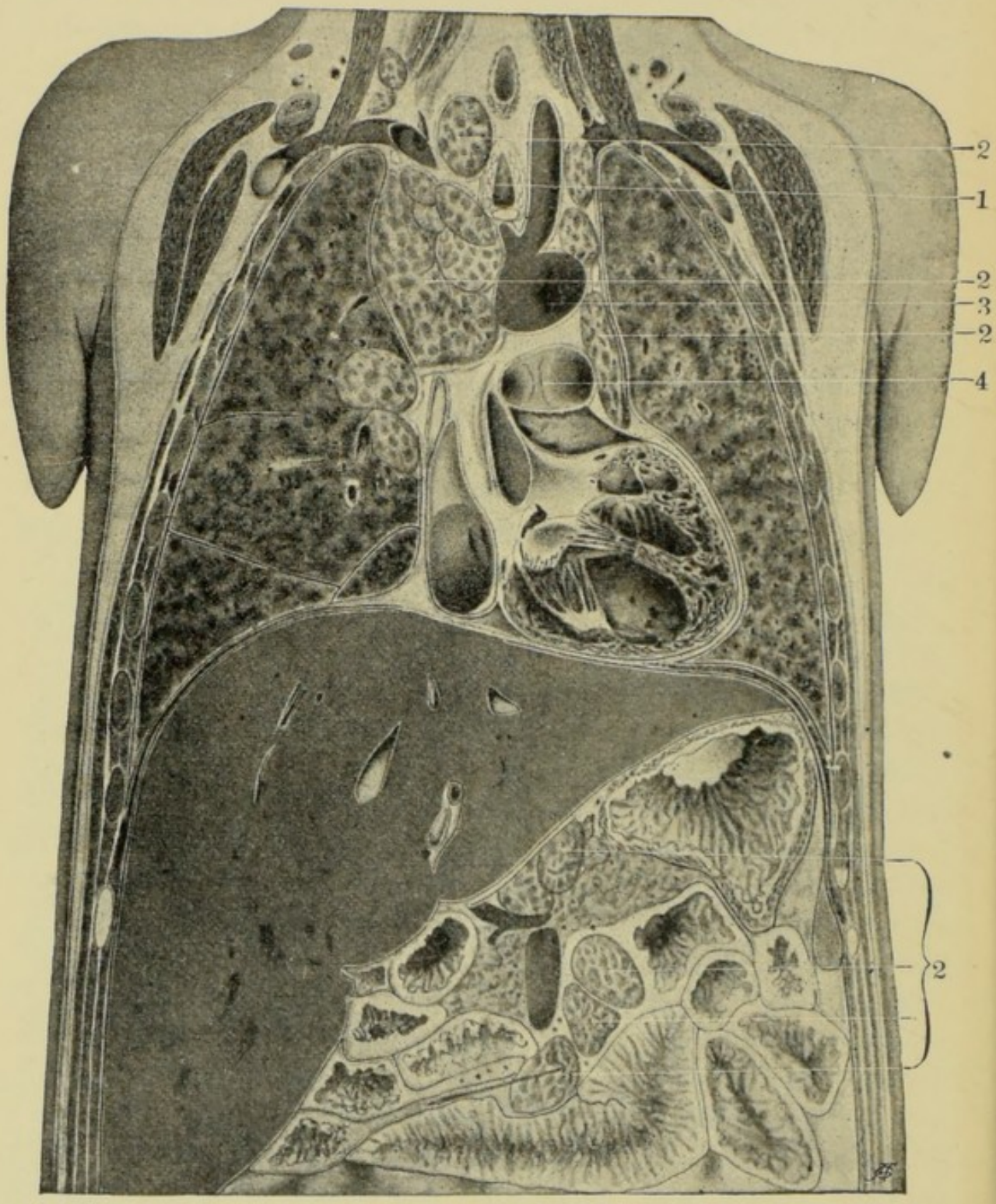


FIG. 63.—Chronic swelling (tuberculous) of the thoracic and abdominal lymph-nodes. A frontal frozen section of a boy four and a half years old, showing the close relationship of the glands to the large blood-vessels, also the position and size of the thoracic and abdominal viscera. 1. Trachea. 2. Lymph-nodes. 3. Aorta. 4. Pulmonary artery. (After J. Symington.)

Symptoms which are caused by the pressure of the clumps of nodes upon nerves, blood-vessels, and air-passages: Paroxysms of cough similar to whooping-cough, but without the inspiratory whoop; hoarseness; increased frequency of the pulse (paralysis of the recurrent and vagus nerves); prominence of the engorged veins of the face, neck, and thorax; clubbing and slight cyanosis of the terminal phalanges of the hand (compression of the large venous trunks); signs of stenosis with respiratory retraction and whistling respiratory murmurs (pressure upon the trachea and bronchi).

Painfulness of several spinous processes between the second and seventh dorsal vertebræ (spinalgia) is claimed to be characteristic (Petruschky).

Although these symptoms are not pathognomonic in themselves, yet they become suspicious when present in children who possess a predisposition to tuberculosis, or in those who have recently passed through an attack of measles, whooping-cough, or influenza, or in those suffering from some other form of tuberculosis or scrofula.

SCROFULA

Scrofula is a combination of chronic swelling of the lymph-nodes with certain inflammatory affections of the skin and mucous membranes, which are characterized by their obstinacy, tendency to relapses, their combined appearance, and their occurrence almost exclusively in childhood.

TUBERCULOSIS AND SCROFULA

Scrofula is closely related to tuberculosis and in certain respects identical. Corroborating this view are: Scrofulous manifestations are very often associated with genuine tuberculous affections, tuberculosis of the bronchial nodes, lupus, caries of the bones, exostoses, and joint disease, in all of which either tuberculosis or scrofula are primary. Simple glandular swelling is frequently observed to develop into true tuberculosis of the lymph-nodes. Post-mortem examinations of scrofulous children always

PLATE 16

Scrofula.—Boy six years old. Chronic rhinitis with excoriations and thickened upper lip; chronic right blepharoconjunctivitis, chronic left keratitis; the facial expression shows sensitiveness to light.

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reveal tuberculosis of the bronchial nodes (Heubner). The majority of scrofulous subjects, including those without evident glandular swelling, react positively to tuberculin (Heubner). Primarily the tubercle bacillus, which because of its minute size can pass through the skin and mucous membranes without causing any damage, enters a bronchial, cervical, or mesenteric node, and sets up a true chronic tuberculosis. The resulting impairment of the general health lessens the resisting power of the body, which, together with the fact that in certain children the permeability of the skin and mucous membranes ("external barrier") to bacteria is already increased, offers but little resistance to the entrance of pus-exciting microorganisms and tubercle bacilli. The scrofulous catarrh of the mucous membranes, eczema, etc., in which sometimes pyogenic cocci and at other times tubercle bacilli are found, may therefore be, but not necessarily, tuberculous. Likewise the swelling of the lymph-nodes ("inner barrier"), depending upon the affection of the region they drain, may be of a pyogenic or a tuberculous character. The presence of a tuberculosis of the internal glands must be excluded.

Symptoms.—*General Manifestations.*—At first the general appearance is still fresh, but later it is nearly always pale; as a rule, there is no emaciation, rather a certain increase of fat; the body and mind are easily fatigued; dull and often irritable temperament; loss of appetite; headache; shooting pains in the chest. Typic facial expression: Thickened nose, which is excoriated at the nostrils by the secretion; thick, protruding upper lip; reddened, thickened eyelids, which are spasmodically contracted on account of photophobia.

Lymph-nodes.—Swelling is at first localized in the superficial nodes of the neck, lower jaw, and angle of the



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jaw. By contiguity the process spreads to neighboring nodes, and at times also by retrograde infection of bronchial nodes from blocking of the lymph-stream. The size of the nodes varies between a lentil, a hazel-nut, or a walnut. Perinodular inflammation causes a number of nodes to form large clumps. At first the nodes are movable underneath the skin, but later they are adherent. Characteristic of this condition are painlessness, gradual increase in growth, and a tendency of the nodular hyperplasia to undergo necrosis; unless resorption occurs, caseation, calcification, or softening and suppuration set in. If suppuration exists, the surrounding connective tissue is infiltrated, the skin becomes thinner, and an abscess is formed, which ruptures externally unless incised. The abscess contains granular pus, shows but little tendency to heal after evacuation, and often leads, after the existence of fistula for a long time, to the formation of radiating scars.

Not every case of lymphadenitis is scrofulous; the secondary glandular involvement of eczema, dental caries, angina, stomatitis, etc., are usually distinguished without difficulty by their acute course, while lymphomatous tumors are recognized by their persistency.

Skin.—The following are the various scrofulous affections of the skin:

Subcutaneous infiltrations which develop slowly, unaccompanied by manifestations of pain or inflammation, in various parts of the body; these infiltrations lead, in the course of time, to indolent ulcers which have serrated edges (scrofuloderma).

Chronic impetiginous eczema of the face, scalp, ear, and surroundings.

Ecthyma pustules on the lower half of the body, with deep-seated ulcers.

Lichen scrofulosus, which is probably miliary tuberculosis of the skin.

Mucous Membranes. — *Eyes.* — Blepharoconjunctivitis, thickening of the eyelids, peripheral phlyctenula, accompanied by photophobia and blepharospasm, tendency of

the infiltrated tissue to undergo ulceration, keratitis, iritis, and finally, more or less permanent corneal opacity (leukoma).

Nose.—Obstinate rhinitis with tough, pus-like secretion, which forms crusts and excoriates the nostrils, leading to nasal obstruction and thickening of the nose and upper lip; in severe cases there is a destructive atrophic ozena.

Pharynx.—Hypertrophic pharyngitis, chronic inflammation, and hyperplasia of the palatine tonsils, and especially the pharyngeal tonsil, with all the consequences of those affections. (See Adenoids.)

Ear.—Fetid, pus-like, and perforative otitis media, usually double, followed by extension, mastoiditis, etc.

Other phenomena noted are caries encircling the neck of the tooth and a persisting gastro-intestinal and bronchial catarrh.

Course and Prognosis.—The course is always chronic, yet it varies according to the individual's strength, the degree of extension, and, above all, the possibilities as to treatment and attention. In favorable cases, although of long duration, complete cure may be achieved; in others the condition is made worse by the advent of bone caries, lupus, pulmonary and general tuberculosis, and meningitis. The prognosis, therefore, is always somewhat doubtful, especially when caseous foci already exist.

Diagnosis.—Although the occurrence of glandular swelling, catarrh of the mucous membrane, and eczema is met with in other conditions than scrofula, yet they point toward the latter by their simultaneous occurrence, their persistence, and tendency to recurrence. Of diagnostic importance is the general habitus and the facial expression. Positive tuberculin reaction would support the diagnosis.

THE TREATMENT OF TUBERCULOSIS AND SCROFULA

Prophylaxis.—Institute careful nursing and feeding; instruct parents as to the hygienic care of their children;

careful hardening of the body against changes in temperature and disease; encourage a certain amount of physical laziness; use pure milk. Discourage marriage between tuberculous individuals; proper ventilation of dwellings; much time to be spent in the open air.

Preventing Infection of Susceptible Children.—Forbid all association with tuberculous subjects; separate from tuberculous parents, and raise in children's sanitaria; when this is not possible the closest attention must be paid to prevent infection. Children should be taught cleanliness—frequent washing of the hands, care of the mouth and nose, and disinfection of utensils and toys. Guard against diseases which predispose to tuberculosis, such as measles, whooping-cough, etc.

General Hygienic and Dietetic Treatment.—Light, well-ventilated dwellings; sojourn in mountainous regions or at the sea; careful supervision of air- and sun-baths; keep the skin in good condition and give alcohol rubs; salt- or peat-baths (see Rachitis), also sand-baths. Institutional treatment in children's sanitaria and sea hospitals; abundant fatty foods: milk, cream, whipped cream, kefir, butter, and infants' meals; together with chopped meat and pressed meat juice. As a rule give a mixed diet, but constantly alternate with green vegetables, salad, fruit, and compote.

Special Treatment.—*Soft-soap Cure.*—Dilute the tincture of green soap with a little warm water and rub it daily into the skin of the trunk and the extremities, and wash it off in ten minutes. To combat the glandular swelling make hydropathic applications with gruel, mud, or decoctions of oak bark. Also smear the body with potassium iodid ointment or iodovasogen, also in combination with equal parts of soft soap, covered with cotton and allowed to remain over night. Large masses of lymph-nodes are extirpated before softening sets in. Adenoid vegetations and hyperplastic tonsils should be removed.

Medication.—One child's spoonful, twice daily, of brown or light cod-liver oil alone or combined with 1 to 3 per cent. creosote carbonate (creosotal). Cod-liver oil

PLATE 17

Acute Disseminated Tuberculosis of the Lungs.—Bronchogenic form—*bronchiolitis nodosa*. The bronchial lumina may be recognized as points within the miliary foci. Chronic caseous tuberculosis and partial softening of the tracheal and bronchial lymph-nodes. Two-year-old child. Duration of disease, four weeks. Clinical history: Remitting fever, dyspnea, cyanosis, no evident dulness, and emaciation.

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may be substituted for lipanin or Mehring's chocolate. Tasteless guaiacol carbonate ("duotal"), 0.1 to 0.3 gm. per dose. Guaiacol valerianate ("geosot"), 4 to 8 drops three times a day; thiocol, sufficient to cover a knife-point, three times a day. Sirolin or sulfusot syrup, 1 coffeespoonful three times a day. Syrup of the iodid of iron with simple syrup, of each 8 to 20 drops; or the iodid of iron with malt extract, iodoferratose. To stimulate the appetite give the compound tincture of cinchona in 1-drop doses, or wine of iron and quinin in coffeespoonful doses after meals.

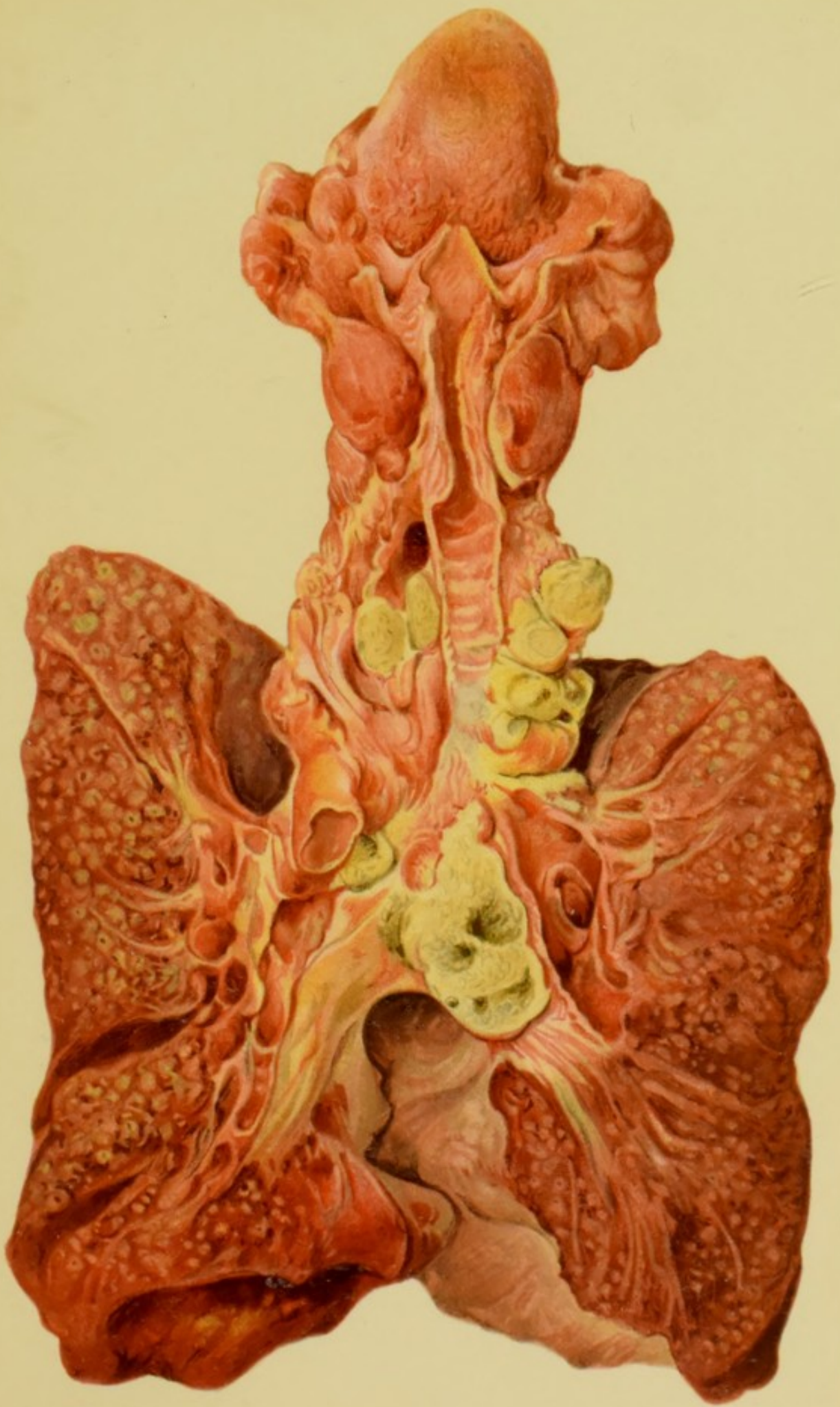
To Combat the Phlyctenular Keratoconjunctivitis.—Yellow mercuric oxid ointment, 1 to 3 per cent.; for obstinate infiltrations, dust with calomel; for the photophobia, hold the head in cold water. Treat the nasal infection with douches of warm salt-water and apply white precipitate salve. (For treatment of the Eczema, see the section on that subject.) The therapy of acute generalized tuberculosis consists in supporting the patient's strength, combating the fever with hydrotherapeutic measures of medication, and relieving the pain with narcotics.

TUBERCULOSIS OF THE LUNGS

Excepting the bronchial glands, the lungs represent the part of the infantile body most frequently affected by tuberculosis.

Morbid Anatomy.—The following are the forms of pulmonary tuberculosis which occur in children. They may occur singly or in combination:

Acute Disseminated Tuberculosis (*Miliary Tuberculosis*).—This type may be either hematogenous or bronchial



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in origin, according to whether the caseous focus ruptures into a blood-vessel or a bronchus. When only isolated portions of the lungs are involved the course is slower and larger nodules are formed—*subacute and chronic disseminated tuberculosis.*

The hematogenous miliary tuberculosis consists of minute nodules scattered throughout the lungs and pleura, not involving the smallest bronchi; hyperemia and consolidation of the lung tissue. It is usually a part of general miliary tuberculosis. In the bronchogenic form the nodules lie in the walls of the smallest bronchi—bronchiolitis nodosa—which finally spread to the neighboring pulmonary tissue.

Caseous peribronchitis is due to extension from old foci along the lymph-vessels of the bronchi, and is accompanied by caseous thickening of the bronchial wall and consolidation in the neighborhood.

Caseous pneumonia is either perinodular, by direct extension of the tuberculous process (frequent in nurslings), or by confluence and spreading of peribronchial foci. The affected area is tense, on cross-section has a yellowish-red to yellowish-white color, and appears granulated because of the exudate from the alveoli.

Secondary tuberculosis develops from pre-existing infiltrations which follow catarrhal or croupous pneumonia.

These various forms may develop into chronic phthisis, with the formation of cavities, connective tissue, induration, calcification, etc. This, the true "consumption of the lungs," attacks the bases of the lungs, as a rule, and is rarer in children than in adults.

Symptoms.—These vary according to the nature of the process. The manifestations of general tuberculosis are always demonstrable, and very frequently before the appearance of the disease itself, in the form of primary glandular tuberculosis. Acute miliary tuberculosis, frequently the termination of other tuberculous affections, usually begins acutely with a high temperature, which later runs the course of a constant or cachectic fever, with increased respiration (40 to 60) of a sighing

character, increased pulse-rate, cyanosis, anemia, and increasing emaciation throughout the course of the disease. There is an irritable cough, usually without expectoration (because the tubercles are mostly extrabronchial); in general the system may be looked upon as being in the typhoidal state. Objectively the lungs present no change or only a dry catarrhal capillary bronchitis; the spleen and liver are swollen at times, and frequently older tuberculous foci are found in the lungs and other organs. Death follows the development of diarrhea, convulsions, and weakening of the heart. The subacute and chronic forms of disseminated bronchial, hematogenic, and lymphogenic tuberculosis present a more gradual onset, a hectic fever, anemia, and emaciation; mild dyspnea, variable areas of dulness, which are localized with difficulty; the respiratory murmur is increased from vesicular to bronchial; whistling and small vesicular râles are heard; yet clinically the picture of bronchopneumonia is not present.

Caseous Pneumonia.—A persistent lobar or lobular pneumonia, especially after measles, whooping-cough, or influenza, accompanied by emaciation and loss of appetite; a recently elevated or hectic fever; or primarily the symptoms of a progressive bronchopneumonia with dulness, râles, bronchial breathing, to which are added the symptoms of the general disease. The expectoration is pus-like and contains abundant tubercle bacilli (obtain by removing with cotton swabs). The dulness, which frequently spreads from the spine toward the apices or to the region of the scapula, often remains unaltered for many months.

Course and Prognosis of Pulmonary Tuberculosis.—The acute and subacute disseminated tuberculosis always ends unfavorably, usually after a few days or weeks. Cure is possible in the mild cases of caseous pneumonia, but never in the severe types. Death follows cardiac weakness, generalized tuberculosis, or meningitis. Chronic disseminated tuberculosis, like true pulmonary phthisis, is more hopeful in children than in adults, and permanent cure is comparatively frequent.

Diagnosis.—The following are the characteristics of a pulmonary disease which is tuberculous in nature: The obstinacy of the condition; the disproportion between the comparatively few local and the severe general symptoms; general habitus; emaciation; anorexia; cyanosis; the existence of other tuberculous affections; chronic swelling of the lymph-nodes, especially the supraclavicular group; the discovery of the tubercle bacillus; positive tuberculin reaction.

Treatment.—As to prophylaxis, predisposed children should be carefully watched after the acute infectious diseases, and sent, if possible, to the country. In the undoubted presence of pulmonary tuberculosis begin the treatment with rest in bed, followed by the open-air cure (protected against the wind). No sea-baths; on the contrary, the child should be sent to a mountainous region.

Medication.—Cod-liver oil, creosote, guaiacol. Stimulate the appetite with the compound tincture of cinchona. To allay expectoration give codein and the extract of belladonna (āā 0.01 to 0.05 gm. a day). To combat the fever make cold applications and give quinin internally; acetic acid for the sweats; or 1 per cent. menthol or spirits of the salicylates. Gelatin internally or injected for hemoptysis, or liquor ferri chloridi, 1 to 2 drops.

TUBERCULOUS PLEURISY

This is usually secondary; when it occurs as a manifestation of miliary tuberculosis the pleura usually contains minute tubercles; when a complication of pulmonary tuberculosis, it is usually of the dry form, with the formation of fibrous indurations and caseating infiltrations. If a watery exudate exists, the pleurisy is of the serous or serosanguineous type; it is purulent in form when a cavity ruptures into the pleura, and in that case the pus contains mononuclear leukocytes.

Symptoms.—The onset is insidious, difficult to recognize, and accompanied by the phenomena of general tuberculosis. Local subjective symptoms are absent.

At first there are slight pains, fever, and dyspnea. Later there is dulness, pleural friction-rub, diminished breath sounds and fremitus, lessened excursion of the diseased side, etc. This process leads quite frequently to empyema. (For treatment, see that of Tuberculosis and Pleurisy.) The diagnosis is of importance because pleurisy is frequently the first manifestation of tuberculosis.

TUBERCULOUS PERICARDITIS

This follows extension of the pulmonary process to the pericardium. If it involves both the parietal and visceral pericardium, they become intimately united. The symptoms are similar to those of ordinary pericarditis. Dry pericarditis in children is always suggestive of tuberculosis.

The *pharyngeal* as well as the *palatine tonsils* may become diseased primarily by aspiration, that is, dirt infection, and secondarily through the expectorated sputum. In the first form nodules are usually found lying deep in the glands, and in the other type superficial latent ulcerations are found.

ABDOMINAL TUBERCULOSIS

Intestines.—*Origin.*—It follows the swallowing of tuberculous material (also congenital), or generalized tuberculosis by way of the blood, and from tuberculous mesenteric glands by way of the lymph-channels.

Morbid Anatomy.—The solitary follicles and Peyer's patches in the lower small and the large intestines are infiltrated, and breaking down, form ulcers, the edges of which are irregular, undermined, and infiltrated. These ulcers spread in a transverse direction in the intestinal walls; they are often circular in outline and are surrounded by minute tubercles. A resultant local peritonitis, with adhesions of the involved section of the intestine and encapsulation of the exudate frequently occur.

Symptoms.—This condition develops either primarily with a gradual onset, or it arises secondarily to already existing tuberculous disease. An intractable diarrhea

sets in, alternating at times with constipation. Vague abdominal pain, with meteorism, nausea, and loss of appetite. Aside from these the following constitutional symptoms exist: Emaciation, sweats, and irregular elevations of the temperature.

Course and Prognosis.—The course is always protracted throughout months and years. The outlook is unfavorable because of the increase of the diarrhea and cachexia.

Treatment.—Observe general hygienic and dietetic principles. Preserve and increase the body strength; hydropathic applications; feed as in case of catarrh of the large intestine. Of the astringents give: Bismuth subcarbonate, subnitrate, subgallate; silver nitrate (0.05 to 100.0 gm.); calumba, tannigen, etc. [In persistent cases opium must be used in sufficient quantity to control diarrhea.—Ed.]

Mesenteric Nodes.—Tuberculous disease of the mesenteric nodes arises primarily by the entrance of the bacilli through the intact intestinal walls, or secondarily from an already existing tuberculosis of the intestines or peritoneum. As in the case of the bronchial nodes, tuberculosis of the mesenteric nodes may arise as an independent disease—tabes mesenterica. The swollen nodes form large masses, which are packed closely together.

Symptoms.—Those of the general condition, together with a rounded dome-shaped abdomen, the apex of which is at the umbilicus; dilated abdominal veins, enlarged inguinal glands, and abdominal pain. If the glands are palpable (which is not always the case), they may be felt as movable tumors deep in the abdomen near the umbilicus. The diagnosis, because of the possibility of fecal masses, can only be established after the intestines are evacuated. The course is usually unfavorable.

Treatment.—Hot applications, inunctions with soft soap; otherwise like that of tuberculosis of the bronchial glands.

Peritoneum.—*Origin and Morbid Anatomy.*—Tuberculous peritonitis arises either secondarily to general tuber-

PLATE 18

FIG. 1. **Chronic Tuberculous Peritonitis.** — Semidome-shaped abdomen. Flattened, chronically infiltrated, and pigmented periomphalitis. Four-year-old girl. (Clinic of Escherich, Vienna.)

FIG. 2. **Umbilical Fungus.**—(See text, p. 84.)

culosis or it is lymphogenic in origin, following tuberculous ulcerations of the intestines, abdominal glands, vertebræ, genitalia, lungs, etc.

In the first case miliary and submiliary nodules are distributed on both visceral and parietal layers, the presence of which are unrecognizable clinically. In the second case—true tuberculous peritonitis—there is at first the secretion of a thin serous fluid, this is followed by a serofibrinous, pus-like, sanguineous, or ichorous (in intestinal perforation) exudate. Next, fibrinous and caseous deposits are formed; the intestinal coils adhere to each other and to the abdominal wall, and encapsulated abscesses are formed. Occasionally the peritonitis is of the dry form, accompanied by extreme thickening and wrinkling of the omentum. A fatty or cirrhotic liver or an amyloid liver and spleen are often met with.

Symptoms.—As a rule the onset is gradual, but occasionally it presents the picture of an acute and, later, chronic peritonitis.

Chief Symptoms.—Gradual increase in the size of the abdomen, which presently assumes an oval or semi dome shape, in marked contrast to the emaciation of the rest of the body. The abdominal skin is tightly stretched, and through it the veins are visible. The umbilicus, instead of being flattened, may protrude and be infiltrated—periumbilical inflammation. Usually the presence of free fluid is demonstrable (light percussion and palpation). At times a dense resistance, and the shape of exudative tumors are palpable. There is but slight tenderness to pressure, but considerable intermittent abdominal pain. Sometimes a respiratory friction-rub is heard over the spleen and liver. The stools are clay-like, acid in reaction, and fatty. Otherwise it presents the picture of the general disease.





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Course and Prognosis.—The disease lasts for months and years, with intervals of improvement or of arrest, during which time the exudate may be increased or decreased. Sometimes abscesses may rupture at the umbilicus or into the intestines, an event which does not favorably influence the course of the disease. Death may occur through a progressive marasmus, acute peritonitis, general tuberculosis, or meningitis. In milder cases spontaneous healing gradually occurs, with permanent encapsulation; the latter makes recurrence of the condition possible. The prognosis is, therefore, always doubtful, and is less hopeful in the presence of caseous products or other tuberculous affections.

Diagnosis.—Of significance is the enlargement of the abdomen while the rest of the body undergoes emaciation; symptoms of general involvement, the subsequent development of tuberculous processes, and the discovery of solid or fluid exudates.

Treatment.—That of the general disease. Rest in bed, a non-irritating but strength-producing diet, milk, infant meals, eggs, bouillon, chopped meat, meat juice, somatose, and fruit jellies. For the pain, make warm applications, opium. To absorb the exudates anoint with soft soap or vasogen. Internally give creosote, guaiacol. If these procedures remain ineffectual perform a laparotomy, simply remove the exudate, and institute drainage. This operation gives good results in the serous, encapsulated, and dry form (mortality-rate of operation 27 per cent.), but is contra-indicated in generalized tuberculosis and advanced cachexia.

TUBERCULOUS MENINGITIS

Basilar Meningitis. Acute Hydrocephalus.—Tuberculous meningitis, a local phenomenon of acute miliary tuberculosis, consists of a tuberculous inflammation of the coverings of the brain, to which the infection is transmitted by the circulation. It is frequently the terminal stage of tuberculosis of any organ.

PLATE 19

Acute Tuberculous Basilar Meningitis.—The blood-vessels of the pia are considerably congested. A deposit of a grayish-yellow or greenish gelatinous exudate in the meshes of the pia between the chiasm and the medulla oblongata. Slight exudates along both Sylvian fossæ, where a large number of miliary tubercles were found. The dilated ventricles of the brain contained a slightly turbid exudate (Hydrocephalus internus). Other changes in the body: Chronic caseous tuberculosis and softening of the bronchial glands. Beginning miliary tuberculosis in the lungs and spleen.

Morbid Anatomy.—Hyperemia of the meninges, especially at the base and in the Sylvian fossæ. The arachnoid is stretched, the convolutions flattened; the peripheral brain substance is often softened and is torn when the pia is pulled off. Deposit of a gelatinous, slightly yellow or grayish-green pus-like exudate between the meninges lying between the chiasma and medulla oblongata. This exudate surrounds the cranial nerves after their exit and extends into the Sylvian fossæ. The pia, especially along the blood-vessels, contains miliary and submiliary tubercles, in which the bacilli may be detected. Tubercles are also found on the convexity, but rarely. At times the tubercles cannot be observed macroscopically. Fluid is found in the ventricles and the subdural spaces—*hydrocephalus internus* and *externus;* dilatation of the ventricles and softening of their walls. Anemia of the brain substance. Beginning miliary tuberculosis in the lungs, liver, spleen, and bone-marrow; the latter is positively demonstrable only at times (Henoch). Caseating foci in several bronchial or mesenteric nodes; there is also often advanced tuberculosis of other organs.

Symptoms.—These are manifold. The disease is usually divided for description into the following three stages: Irritation of the brain, cerebral pressure, and cerebral paralysis. This is not always tenable, for one form tends to merge into the other. A division of the various stages from a psychic point of view is more suitable, for then the groups of symptoms may be placed in a more constantly uniform and definite classification. Accordingly there is (1) a prodromal stage, with depression of the mind; (2)



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an initial stage, with apathy of the mind, but with consciousness remaining; (3) a stage of somnolence, and (4) a stage of stupor.

Following is a picture of the typic course of tuberculous meningitis. Weeks and even months before the onset of the disease certain prodromal symptoms appear: The disposition, which has hitherto been cheerful, becomes moody, melancholy, and fearful. The child becomes weak and quiet, loses its appetite, its skin turns pale, and undergoes superficial emaciation. Its sleep is restless and disturbed by dreams. The patient yawns constantly, has a desire to support its head and to sleep during the day, and presents a dragging, uncertain gait. Occasional headaches; disturbance of the intestinal functions; it tends to be constipated and vomits from time to time. These symptoms are especially noticeable in children who have been particularly healthy, less so in those already subject to tuberculosis.

The following initial symptoms occur at the beginning of the disease: Continuous vomiting, which is independent of the ingestion of food and which continues in spite of dietetic management; it either ceases after a few days or continues until the end; headache chiefly localized in the forehead or occiput, which is also described as pain in the neck; obstinate constipation; increased pulse-rate; hyperesthesia, photophobia, sensitiveness to noises and movement. An apathetic and vacant expression, with intervals of restlessness and loud crying. In general, reasoning is maintained; the child plays about, talks, and looks at books. The sleep is restless and interrupted by sighing and sudden awakening. The tongue is coated, there is anorexia and swelling of the spleen. After a few days the characteristic symptoms set in: The pulse becomes slower and beats only 100, 90, 80, or even 70 times a minute; it is irregular, intermittent, and of varying frequency within a minute. The pupils are contracted and unequal. The respiration varies in frequency and depth and is accompanied by deep sighs. The apathy increases and the next stage is entered. The apathy and

drowsiness lead into a state of perpetual somnolence, from which, however, the patient may be aroused; the child is still able to answer questions, recognize its surroundings, but soon falls asleep again. The eyes are then usually only half closed. Sleep is interrupted by restless tossing, mild delirium, or shrill outcries—"cri hydrencephalique"—while the pulse and respiration still maintain the above changes.

Development of Symptoms of Cerebral Irritation.—Converging strabismus, which may disappear again; also nystagmus. Dilatation and undulation of the pupils, that is, contracted when exposed to the light and immediate dilatation in spite of the presence of light. By

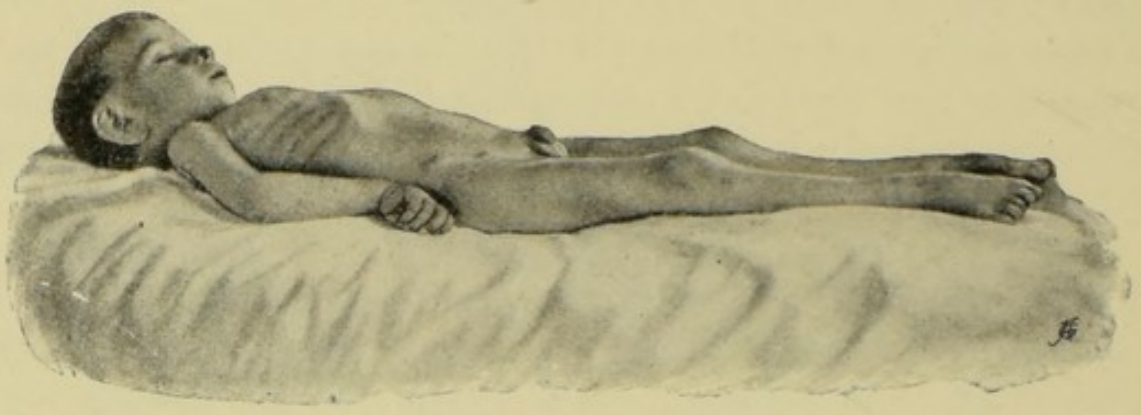


FIG. 64.—Tuberculous basilar meningitis. Five-year-old boy. Stage of stupor. Marked emaciation; contracted abdomen; tonic spasm of both lower extremities; spasm of right hand in pronation. The left arm was paralyzed. Spasms and palsies were not permanent, but alternated with each other.

ophthalmoscopic examination choked optic disk and choroidal tubercles are detected. Loud gnashing of the teeth; movements of mastication. A wandering movement of the hands; twitching of the lips and skin. Oscillatory movements of the extremities, which are lifted widely apart. *Kernig's sign:* The leg when flexed at the hip and knee cannot be extended in the sitting posture. The tendon and skin reflexes are increased. *Tache cérébrales:* Drawing the finger-nail over the skin is followed by dark red stripes, which continue for some time (Trousseau).

The abdomen gradually undergoes a scaphoid retraction on account of lack of nutrition and contraction of the



FIG. 65.—Tuberculous basilar meningitis. Boy three and a quarter years old. Stage of stupor. Eyelids only partly closed. Corneæ are beginning to dry up. Ptosis of the right eyelid. Converging strabismus. The lower jaw hangs relaxed; the nasolabial folds have disappeared (paralysis of the labial and maxillary muscles). Dry lips and tongue. (Clinic of Escherich, Vienna.)

intestines (irritation of the vagus, Heubner). Rigidity

of the neck is usually not very pronounced. The influence of light upon the eyes lessens and soon ceases entirely. The somnolence is changed into stupor. The patient becomes wholly unconscious and fails to respond even to the strongest stimulation.

Final Stage.—At times there is a short return to consciousness just before death. Eyes half closed; cornea insensible; palpebral fissures absent; flakes of mucus in the eyes; loss of sight and hearing. The pulse begins to grow more rapid, and its frequency may reach 200 or even higher (cardiac weakness); its rhythm becomes regular. The respiration is of the Cheyne-Stokes type, with long pauses, which may last fifty seconds. In consequence of the cardiac weakness and the insufficient oxidation we plainly see cyanosis, peaked nose, thin lips, and cold extremities. Excessive emaciation until the body is no more than a skeleton; paralysis in the regions supplied by the cranial nerves, including ptosis, facial palsy, hemi- and monoplegic palsies, which may again disappear, and be replaced by chronic or epileptiform convulsions and extreme tremor. Complete anuria; incontinence of feces. Death often sets in after days of coma. The fever presents no characteristic curve; it is higher at the beginning than during the rest of its course; it remits irregularly and rises in the evening, and is always of a moderate degree, but as the end approaches it frequently rises abnormally high—41° or 42° C. [105.8°–107.6° F.]—(paralysis of the heat-moderating center, Henoch). Vomiting and constipation may be absent.

Course and Prognosis.—The prodromata last several weeks or months; the duration of the disease itself from the first vomiting attack is three weeks on an average, but it may be shorter or as long as eight weeks (Monti). The somnolent stage is the longest. The development of cerebral irritation marks about the middle of the disease itself. The increase in frequency of the pulse begins about two and a half or, at the most, four days before death. The prognosis is hopeless. The cases are rare in which the patients recover from the first attack

and live long enough to experience a second attack, to which they are sure to succumb.

Diagnosis.—Chief characteristics: *Vomiting*, independent of the ingestion of food; *headache* and *constipation* in a child whose general health and state of nourishment have been disturbed for several weeks. Abnormally slow and unequal *pulse* and irregular *respiration*. Gradual mental failure, languor and drowsiness, apathy, somnolence, and stupor. Tuberculosis in the child itself or in its ancestors strengthens the diagnosis. The detection of the tubercle bacillus in the cerebrospinal fluid (by centrifuge) makes the diagnosis positive. Lumbar puncture shows increased pressure of the cerebrospinal fluid, which at the beginning is clear, but later cloudy, as if a fine dust were held in suspension; the fluid also contains a high and constantly increasing percentage of albumin (1 to 6 per cent., instead of 0.2 to 0.4 per cent. normally) and mononuclear leukocytes.

Possible Errors.—*Typhoid fever:* Typic elevation of fever, splenic tumor, roseola, bronchitis, meteorism, diarrhea, presence of typhoid bacillus in a drop of the blood, or a positive Gruber-Widal reaction. *Dyspepsia* or *constipation:* Absence of prodromata and influence of therapy. *Intestinal parasites* with *cerebral irritation:* Examination of stools; administration of anthelmintics. *Hereditary syphilitic cerebral processes:* Coryza, syphilids, and other specific symptoms. *Uremia:* Examine urine.

Differential Diagnosis between Various Forms of Meningitis.—*Epidemic Cerebrospinal Meningitis.*—Acute onset without long prodromal stage, more intense headache, pronounced and painful stiffness of the neck and spinal column, hyperesthesia of the skin, early somnolence, no abnormal slowness or irregularity of the pulse. The cerebrospinal fluid is made turbid by the presence of pus and contains the Meningococcus intracellularis. High albumin-content (3 to 6 per cent.).

Purulent Meningitis.—Sudden onset with high fever, convulsions, and headache following an injury to or a suppurative process of the skull. Slight stiffness of the neck,

spinous processes of the vertebræ not sensitive to pressure; the spinal fluid clouded with pus and containing polynuclear leukocytes, pus bacteria, and no meningococci. It runs a rapidly fatal course; blood shows an inflammatory leukocytosis.

Serous Meningitis.—Spinal fluid clear, sterile, contains more than 1 per cent. of albumin; relieved by lumbar puncture.

Treatment.—In case of doubtful diagnosis give calomel, 0.05 gm., repeatedly; a leech applied to the mastoid process; rub the head with unguentum cinereum or unguentum Credé. If syphilis is suspected give potassium iodid, 0.5 to 1.5 gm. per day (Fischer). In every case darken the room and prevent noises; cold applications to the head or ice-caps, but avoid pressure; for the headache give sodium bromid and ammonium bromid, of each 5.0 gm. : 100.0 gm., a coffeespoonful three times a day (Bendix). Combat the nervousness and convulsions at the beginning with warm baths, followed by a cold douche; later give chloral hydrate (1.0 gm. : 50.0 mucilage) in three rectal injections. Apply cold pack for high fever. Careful nursing of the eyes and mouth; water cushions; light, appetizing, and varied diet.

TUBERCULOSIS OF THE BONES AND JOINTS

Tuberculosis seldom attacks the osseous and articular systems primarily, as a rule it occurs secondarily by transmission through the circulation from a tuberculous gland—as the first manifestation of latent tuberculosis. Injuries are of predisposing influence. The affection begins usually in the bones, and then extends by continuity or by way of the lymph-channels to the joints. In the long bones the epiphyses, and in the short bones the diaphyses, are especially liable to be involved.

Morbid Anatomy.—The bones show tuberculous deposits in the marrow, chronic caseating osteomyelitis, caseous necrosis of the spongiosa, and chronic periostitis. The tubercles grow in size, undergo caseation, and form large caseous infiltrations in the spongiosa with isolated frag-

ments of necrosed bone. These foci soften and become converted into cavities lined with granulation tissue and filled with caseous pus, in which the bony sequestra lie free. Simultaneous proliferation of the periosteum causes a thickening of that membrane. Local tuberculous infection of the periosteum results in superficial or deep caries, in the formation of caseating periosteal nodules, or in the development of cold abscesses. These usually rupture externally by means of fistulæ, either at the site of their origin or, tunneling through the tissue, they appear in an altogether different location.

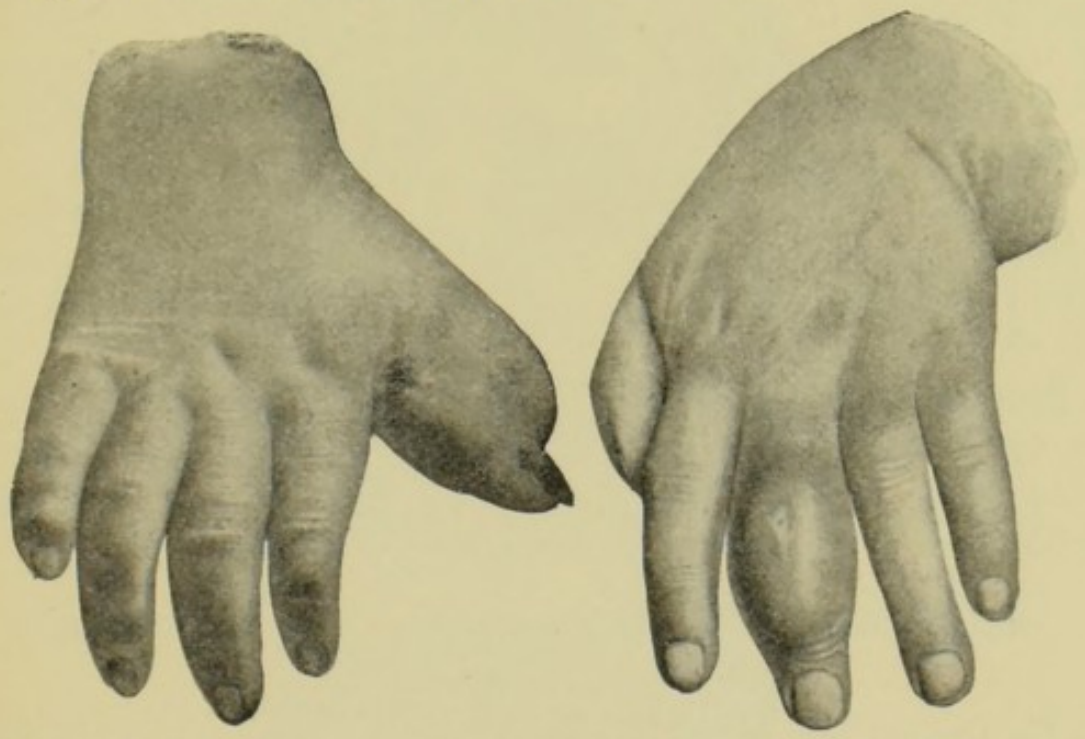


FIG. 66.—Spina ventosa of the right thumb and left middle finger of a three-year-old child.

In the *joints* we note the eruption of disseminated tubercles on the synovial membrane; the latter is converted into a hyperemic, infiltrated, or soft grayish-red granulation tissue, which is permeated with tubercles—*arthritis fungosa;* a serofibrinous or pus-like exudate fills the articular cavities. The surrounding soft parts are edematous and infiltrated, the skin pale, smooth, and shiny—*tumor albus;* development of cold abscesses and fistulous tracts.

The constitutional disturbance in the mild cases is

slight, but in all severe forms, especially in repeated attacks, it is considerable and simulates general tuberculous infection. The course is always prolonged. Cure (relative) is possible in all stages, but is usually accompanied by anomalies of position and function. Tendency to recurrences. Constant danger of general tuberculosis, meningitis, etc.

The most important symptoms of bone and joint tuberculosis are :

SPINA VENTOSA

This is a tuberculous osteomyelitis of the phalanges, consisting of a suppurative absorption and enlargement of the marrow spaces accompanied by a simultaneous periosteal bone formation. This causes a swelling of the bone.

Clinical Symptoms.—A slowly developing, painless, spindle-shaped swelling of the shaft of the bone ; the skin becomes red and thin ; eruption occurs and fistulæ are formed.

Treatment.—Compress with circular bands of adhesive plaster, iodovasogen, comfortable position ; surgical intervention to remove diseased tissue ; in severe cases disarticulation. Spontaneous cure is possible.

SPONDYLITIS. TUBERCULOUS CARIES OF THE VERTEBRÆ

(*Pott's Disease*)

This consists of tuberculous inflammation and caries of a single vertebra or of the intervertebral disks ; the lower dorsal or the lower cervical vertebræ are usually involved. The spinal column breaks down gradually or suddenly (by pressure from above). A pointed, and in case of disease of several vertebræ a rounded, hump results—"gibbus." The pus which collects in the neighborhood of the diseased bone seeks to gravitate externally ; in caries of the cervical vertebræ it appears as a retropharyngeal abscess ; in disease of the dorsal vertebræ it travels down along the spinal column to the lower portion of the pelvis and back, and most frequently forms a typic psoas abscess at the inner side of the femur.

Symptoms.—Vague pains in the spine, disinclination to walk and stand, frequent desire to lean against objects.

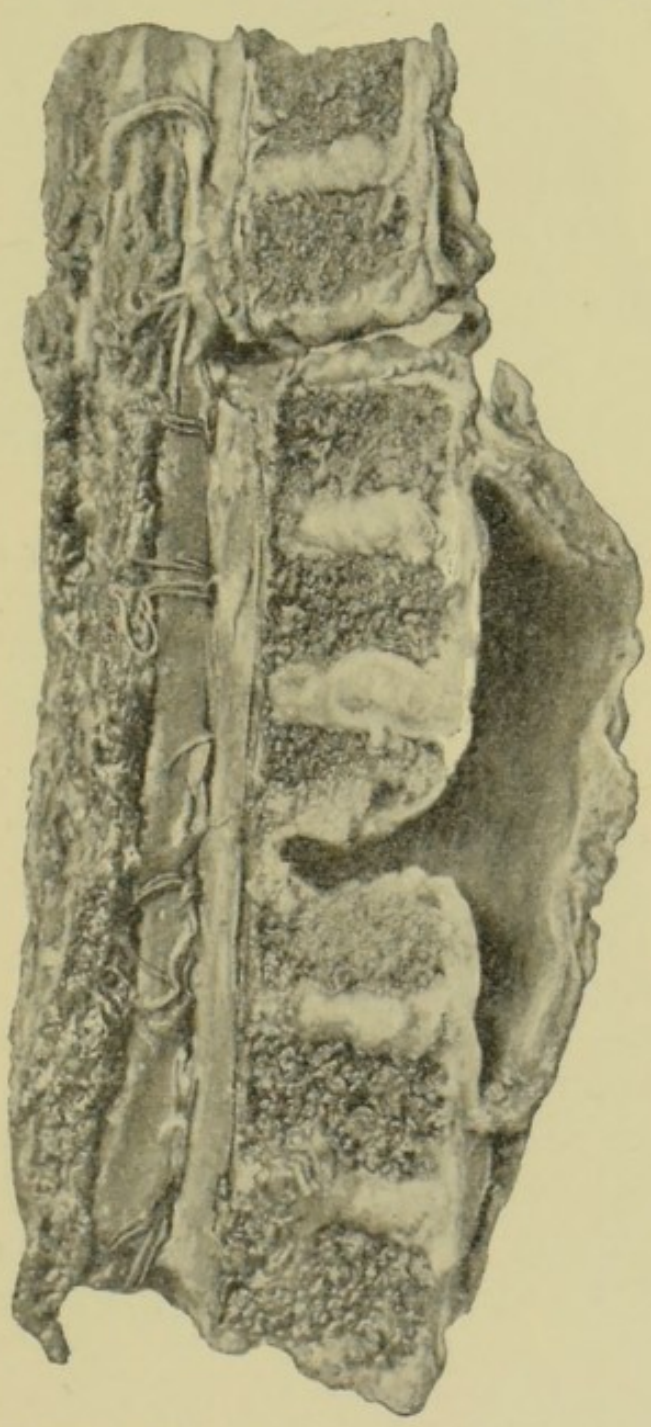


FIG. 67.—Tuberculous caries of a vertebral body (spondylitis), with the formation of a psoas abscess.

Gradual stiffening of the spinal column, which is especially noticable at an early stage when picking an object up from the floor. Localized pressure tenderness when the spinous processes are palpated; a gradual or rapid

FIG. 68.—Spondylitis of the upper dorsal vertebræ. Sharp-angled kyphosis. This eight-year-old girl showed phenomena of transverse myelitis (spastic paraplegia of the legs with increased reflexes), which under orthopedic management disappeared, with the cure of the spondylitis. (Clinic of von Ranke-Herzog, Munich.)

development of any one of these symptoms. Formation of a more or less pointed hump, which does not disappear when the patient lies on the abdomen. As the process progresses the symptoms of the general disease gradually arise. In severe cases extension of the inflammation or compression of the spinal cord leads to manifestations of myelitis, which varies according to the height of the diseased area. In cervical spondylitis disturbances of swallowing and speech also co-exist.

Results.—Cure, with a remaining large or small hump; death due to exhaustion, amyloid disease, peritonitis, general tuberculosis, myelitis, or meningitis. The treatment should strive to relieve pressure and to set the spinal column at rest by means of extension beds and orthopedic corsets; also general hygienic management. Osteomyelitis and periostitis of the long bones, of the malar bones, of the temporal bones, orbital, etc., are manifested by chronic swellings, cold abscesses, fistulous tracts, and caries.

Treatment.—Where possible, make alcoholic applications, maintain rest, inject iodoformol.

COXITIS

Coxitis is a tuberculous inflammation of the hip-joint, which is usually an extension from the bony portions of the joint.

Symptoms.—At first there is a slight dragging, later a pronounced favoring of the diseased leg and over use of the healthy leg—voluntary limping; pressure tenderness at the trochanter; pains in the knee; diminished mobility of the hip-joint. The leg is held contracted, at first in abduction, flexion, and external rotation, with apparent elongation; later in adduction, flexion, and internal rotation, with apparent shortening. When an attempt is

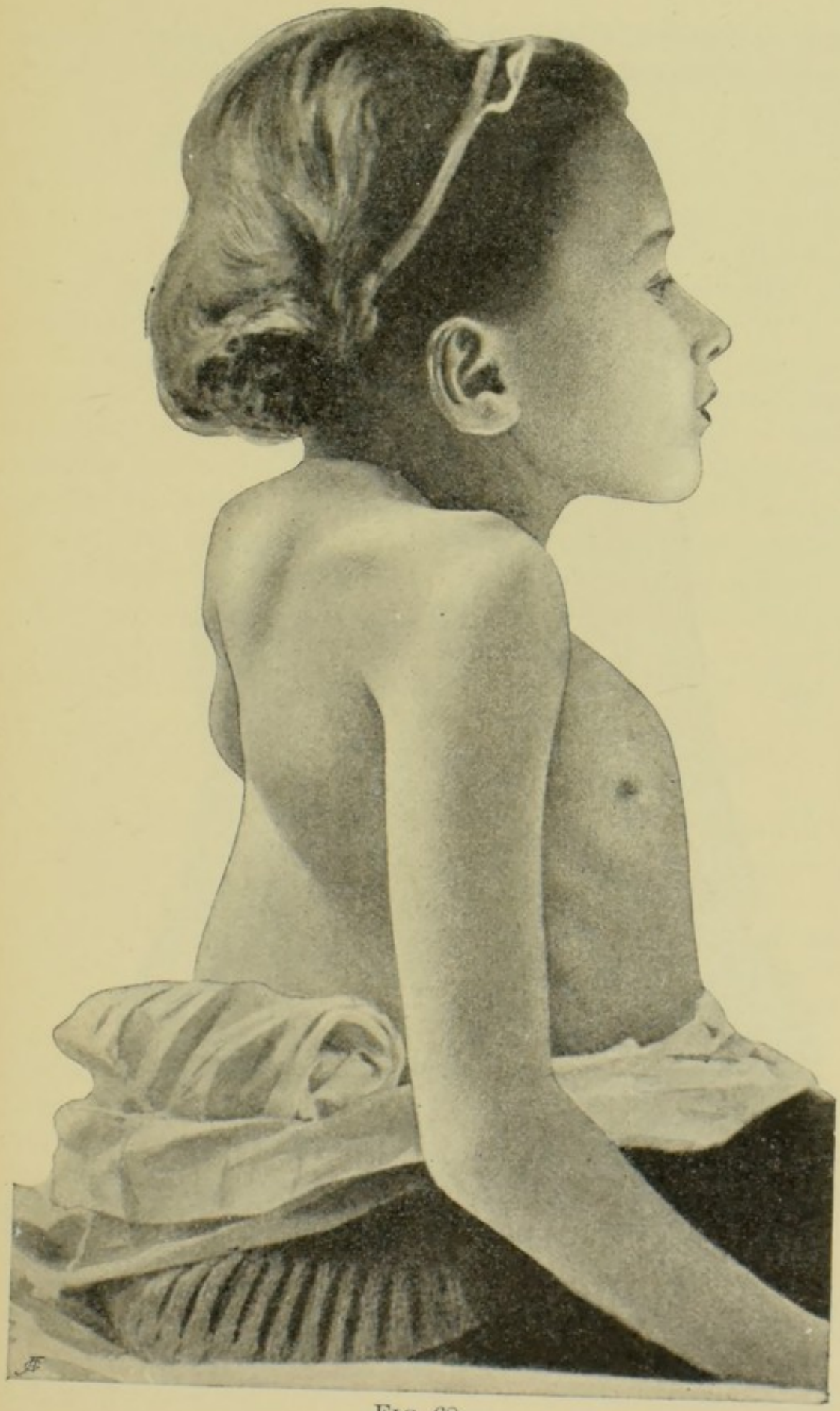


FIG. 68.

made to extend the flexed leg, with the patient in the dorsal posture, the spine is lifted and becomes lordosed

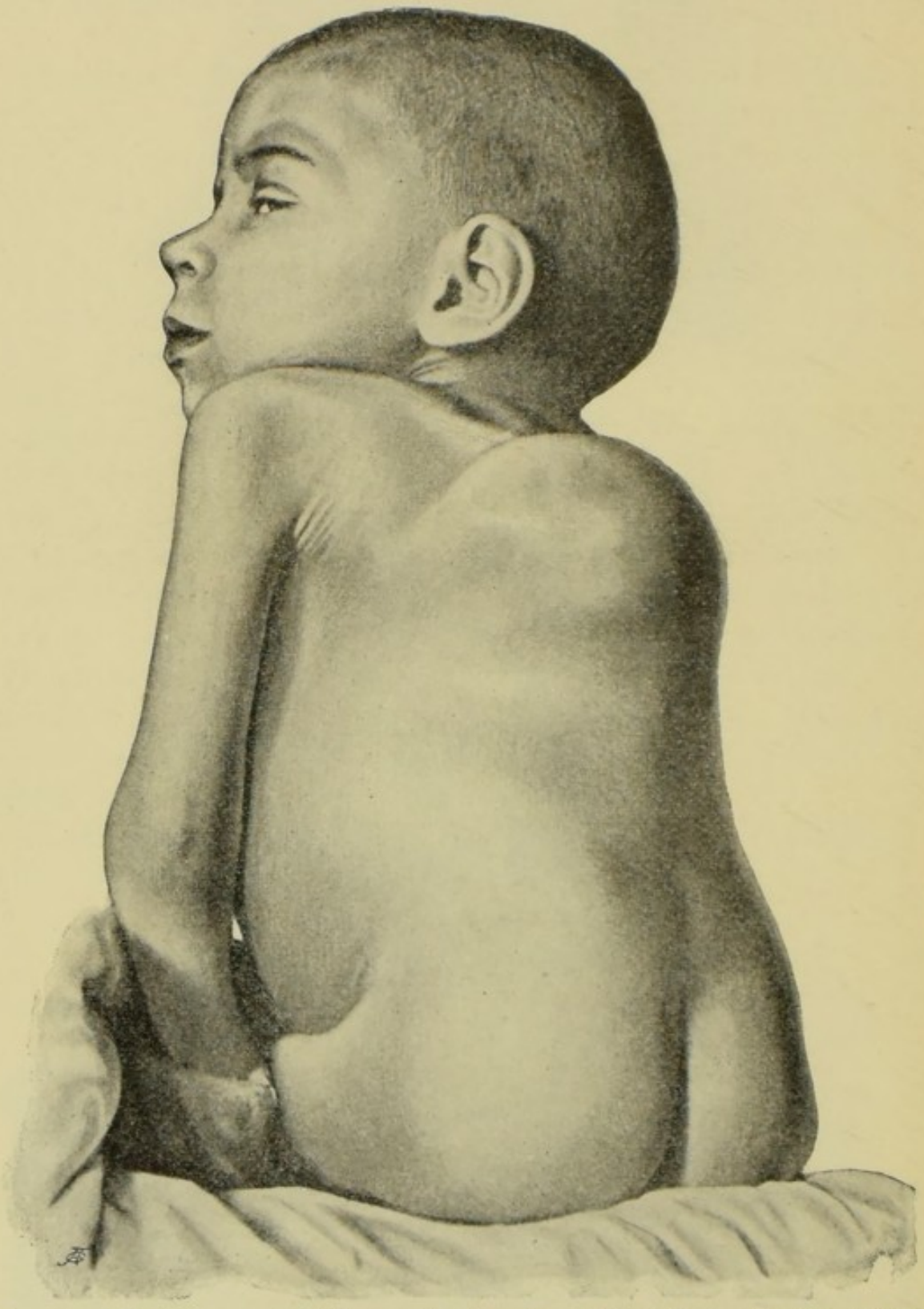


FIG. 69.—Dorsal spondylitis, with the formation of an abscess at the summit of the hump.

and the pelvis moves with the joint; when attempting to

flex the leg the pelvis is raised and the lordosis disappears. Interference with posterior rotation of the affected leg is also of importance; this is tested for by grasping the tip of the foot and rotating first one leg, then the other, while the patient is in the recumbent position. The process progresses with swelling of the hip and gluteal regions and with the development of burrowing abscesses, which commonly rupture at the posterior and outer side of the femur.

Treatment.—Rest by means of plaster-of-Paris cast; permanent extension. As soon as possible institute such orthopedic procedures as will permit the child to walk, and yet correct the deformity and transmit the support of the body weight to the pelvis. The general disease is treated by sojourn in the open air and at the seacoast. The disease may be cured at any stage; the prognosis in the early stages is the most favorable, while in the more advanced periods the cure is only relative and is accompanied by deformities, ankylosis, and pseudo-arthrosis.

TUBERCULOSIS OF THE KNEE-JOINT ("WHITE SWELLING" OF THE KNEE)

Tuberculous disease of the knee-joint begins with stiffness, lessened mobility, and slight pains. Early swelling of the joint may be recognized by the disappearance of the two fossæ at the sides of the patellar tendon, as well as by filling out of the popliteal space. Later the knee is flexed and becomes painful when moved, and also spontaneously. The growing swelling assumes a spindle form, is elastic, and its skin covering is shiny and stretched; there is fluctuation or pseudofluctuation; the patella becomes immobile. The development of an abscess increases the size of the swelling considerably and causes severe pain; the abscess ruptures in the region of the joint itself or in that of the femur or the tibia. Destruction of the joint leads frequently to subluxation and luxation of the tibia backward. Healing is possible in all stages, but, as a rule, connective-tissue overgrowth or true bony ankylosis results.

PLATE 20

FIG. 1. **Tuberculosis of the knee-joint,** which led to destruction of the joint and subluxation of the tibia. Tuberculous osteomyelitis and periostitis of the tibia, with multiple fistulous tracts.

FIG. 2. **Tuberculosis of the Knee-joint.**—Disappearance of the contour of the joint. Doughy swelling at the anterior aspect of the knee covered by tense, pale skin. Fistula formation. Subluxation of the tibia.

Treatment.—Place at rest in plaster-of-Paris cast in the position of flexion. Inject iodoformol; later apply port-



FIG. 70.—Tuberculosis of the right ankle-joint, with doughy swelling of the joint and disappearance of the bony contours.



FIG. 71.—Tuberculous caries of the left tarsal bones, with the formation of a fistula.

able apparatus, with the support at the tuberosity of the ischium.



Fig. 1.

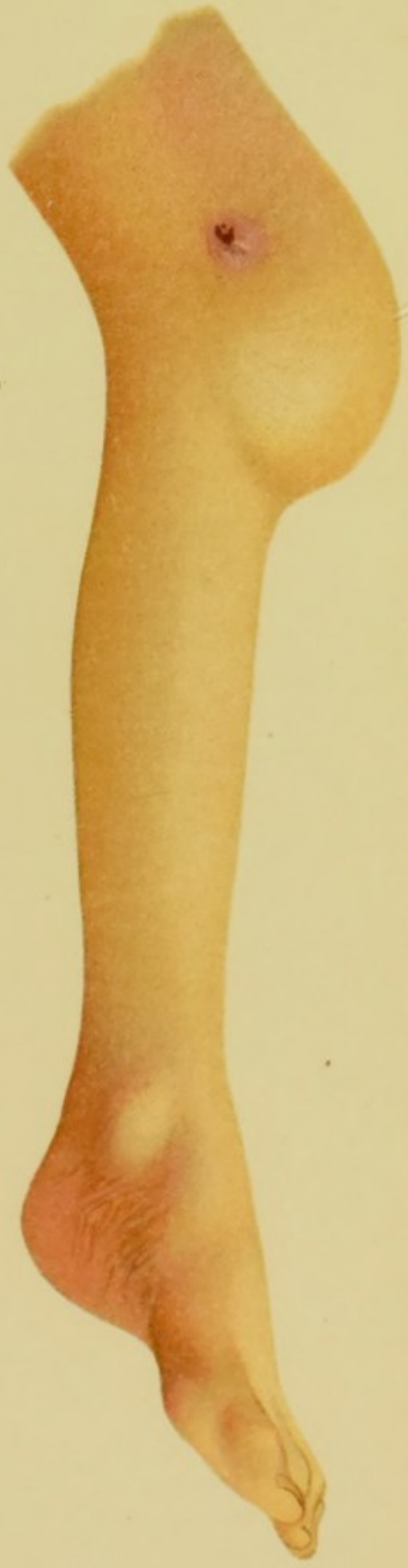


Fig. 2.

TUBERCULOSIS OF THE JOINTS OF THE FEET (TUMOR ALBUS PEDIS)

The ankle-joint is the one most commonly involved. It is accompanied by localized pain in front and at the sides of the foot on standing, which later may also occur spontaneously. The tissue in front and back of each bone is somewhat swollen and the bony contours are lost. Later a distinct and diffuse elastic swelling is noted around the joint; suppuration, cold abscesses, fistula formation.

Treatment.—Put at rest. Alcohol applications. Iodoformol.

TUBERCULOSIS OF THE ELBOW

Pain, interference with movement, and spindle-shaped swelling, which forms a marked contrast to the emaciated upper forearm. The forearm is flexed midway between pronation and supination.

DISEASES OF THE NERVOUS SYSTEM

DISEASES OF THE BRAIN AND ITS MEMBRANES

CEREBROSPINAL MENINGITIS

CEREBROSPINAL meningitis is an epidemic and sporadic suppurative inflammation of the cerebrospinal meninges, which attacks by preference young children and nursing infants; its direct cause is the Meningococcus intracellularis. This is similar to the gonococcus, inasmuch as it occurs in pairs, and is found in groups of twenty or more pairs within the cell. It is stained by methylene-blue, but in the meningeal exudate it fails to stain by Gram's method, whereas it may be detected by means of Gram's stain in smear-cultures. It grows in glycerin-agar; when it is injected intradurally into goats it sets up a typic case of meningitis (Heubner); the meningococcus has been demonstrated in the nasal discharge of patients suffering from meningitis.

Morbid Anatomy.—Hyperemia of the cranium and of the meninges of the brain and spinal cord. A gelatinous, serous, fibrous, or purulent exudate collects between the pia and arachnoid, preferably at the convexity between the convolutions, and at the posterior surface of the cervical and lumbar spine. The brain appears as if it were "smeared with butter." Inflammation and softening of the superficial portions of the brain. A cloudy, seropurulent exudate fills the ventricles.

Symptoms.—Its onset is sudden during the enjoyment of perfect health, or it begins after a short period of prodromal symptoms, consisting of weakness and loss of appetite, with a high fever, convulsions, vomiting, extreme pains in the neck and back, accompanied by loud sighing. Extreme hypersensitiveness to movement, light, and noise. The cardinal symptoms are: Intense stiffness

of the neck and of the spine—opisthotonos. Spasms of the extensor muscles of the extremities, followed finally by tonic rigidity of the whole body. There is also an early clonic twitching and tremor in the various groups of muscles; nystagmus (Kernig's symptom). Partial palsy of the lower extremities, of the muscles supplied by the facial nerve, and of the ocular muscles. Consciousness is soon lost and the patient enters a somnolent stage, which is interrupted by shrill cries and jactitation. The pupils are contracted and the abdomen retracted. Herpes facialis (in 50 per cent.), various erythemata, petechiæ, and urticaria. The pulse and respiration rate are usually considerably increased. At times the former is irregular, and later in the course of the disease it becomes slower than normal. The fever rises rapidly to 40° C. [104° F.] and over, and is irregularly remittent or intermittent.

Course.—Very acute cases are sometimes met with which run a course of only a few hours or days, accompanied by a sudden loss of consciousness, convulsions, subnormal or hyperpyretic temperature, and apoplectiform palsies. In contradistinction we meet abortive forms, presenting a headache, moderate cervical rigidity, and fever, which frequently cannot be recognized except during an epidemic. The average course is protracted over weeks and months and associated, as a rule, with remissions and fresh relapses. Convalescence sets in gradually and is considerably protracted; individual symptoms may persist for a long time. Cerebral disturbances, deafness, blindness, hydrocephalus, and psychoses are frequently the after-effects of this disease. For this reason and on account of the high mortality rate (60 to 70 per cent.) the prognosis must be doubtful. Death occurs during coma, or on account of cardiac weakness, or because of complications, such as disease of the lungs, intestines, heart, kidneys, etc.

Treatment.—Absolute rest. Prevent external irritations; carefully selected, appetizing diet. Hot baths (35° to 40° C. [95°–104° F.]), with cold applications to the head once or twice a day. To relieve the pressure

perform lumbar puncture; repeat every few days. Apply unguentum Credé to the neck, temples, and back. Subcutaneous infusions of sublimate (0.005 to 0.01 gm. per day) in the gluteal region, daily at the beginning, later, every two days (Dazia, Consalvi). Keep nose clean by bathing.

PURULENT MENINGITIS; SIMPLE MENINGITIS

Purulent meningitis is a suppurative inflammation of the membranes of the brain caused by injuries to the skull, extension of suppurative processes in the middle ear, nose, etc. The direct cause is one of the various micro-organisms, especially the pneumococcus, streptococci, and staphylococci, the bacillus of influenza, colon bacillus, typhoid, and pyocyaneus. It attacks children at any age or of any constitution.

Morbid Anatomy.—The convexity of the brain is covered, as if by a hood, with a yellowish-green, purulent, seropurulent, or fibrinous exudate, which lies in the subarachnoid space. The adjacent portions of the brain are inflamed and a turbid fluid is found in the ventricles; the latter may, however, be absent.

Symptoms.—Sudden onset; chills, vomiting; high fever (40° C. [104° F.]); severe convulsions of a tonic and clonic character, which appear at intervals; loss of consciousness; expanded fontanels in nurslings; pupils contracted and unequal; staring eyes; torturing headache; great thirst; rigidity of the neck; Kernig's symptom; temporary erythema. The pulse and respiration rate are extraordinarily rapid; incontinence of feces and urine. Death, at the latest, at the end of a week. Very rarely the patient recovers after a prolonged convalescence, which is, however, nearly always followed by permanent sequelæ. The prognosis is, therefore, serious.

Diagnosis.—Meningitis is differentiated from acute infections by the following symptoms: Expanded fontanels; severe headache; Kernig's symptom, and pupillary contraction.

Treatment.—Prevent external irritation; ice-cold or hot applications; hot baths; leeching; laxatives (calomel, rhubarb); febrile diet; lumbar puncture to relieve the pressure.

SEROUS MENINGITIS. MENINGISMUS

Serous meningitis consists of an infiltration of the pia and the presence of a clear serous fluid in the ventricles, which is accompanied by the symptoms of meningitis. It occurs in tumors and injuries of the skull, as the termination of acute infections and gastro-intestinal diseases, and in otitis media. The symptoms are those of a meningitis, but show no specific characteristics; sometimes they simulate the epidemic form, sometimes the tuberculous or purulent meningitis. The course is, however, usually favorable.

Treatment.—Repeated lumbar punctures. [It is doubtful if this form of treatment would receive unanimous approval from clinicians. True serous meningitis tends to spontaneous recovery.—Ed.]

The spinal cord may be involved in every form of meningitis, and this extension may be recognized by the following manifestations: Rigidity of the spine, muscular twitching in the extremities, hyperesthesia of the skin, and paralysis of the bladder and rectum.

THROMBOSIS OF THE CEREBRAL SINUSES

The following forms of thrombosis are distinguished:

Inflammatory thrombosis, following extension from peripheral purulent processes, mostly from caries of the petrous portion of the temporal bone, head wounds, and eczema. The petrous and transverse sinuses are most frequently involved, more rarely, the cavernous and longitudinal sinuses.

Marantic thrombosis, following interference with the circulation by tumors of the skull and brain, or from slowing of the blood-current in exhausting diseases; the longitudinal sinus is the one most frequently involved. In many cases a bacterial phlebitis is also the cause.

Morbid Anatomy.—The diseased sinus is felt as a tense, thickened cord, which contains at a certain point an adherent thrombotic mass, whose appearance and consistency vary with age and cause; thus it may be homogeneous or stratified, red, gray, or yellow; hard or soft, also purulent. Not rarely there is also a thrombosis of neighboring veins, hyperemia, and also hemorrhages of the meninges and the brain.

Symptoms. — The *characteristic symptoms* are few. Manifestations of a cerebral disease, including convulsions, muscle palsies, etc. Signs of general sepsis are often present. Of the local symptoms the following are important: Bulging of the previously sunken fontanels; hemorrhagic condition of the spinal fluid which has been obtained by lumbar puncture; extension of the thrombotic process to the jugular vein; unilateral swelling of the eyelids and face; protruding eyeballs (cavernous sinus), cyanosis of face and forehead (longitudinal sinus); one jugular less full of blood than the other, and swelling of the mastoid process (transverse sinus). The result is usually fatal; cure with remaining defects is possible in marantic thrombosis (permanent disturbances of cerebrum). Operative treatment of otitic thrombosis sometimes gives good results; other treatment consists in applying antiphlogistics and in depleting the part.

CIRCULATORY DISTURBANCES OF THE BRAIN

HYPEREMIA

Active hyperemia follows increase of arterial blood-pressure in traumatism, sunstroke, at the beginning of the acute infectious diseases, in meningitis, alcoholic intoxications, psychic excitement, and dentition.

Symptoms.—Hot, flushed head, reddened eyes, headache, ringing in the ears, arterial pulsation, vomiting, excitement, delirium, somnolence, coma, and increased pulse rate.

Treatment of Active Hyperemia.—Antiphlogistics; leech-

ing back of the ear; ice-caps; depletion by purging with calomel or compound infusion of senna.

Passive hyperemia is due to venous obstruction in pulmonary and cardiac diseases, struma, whooping-cough, spasm of the glottis, or holding of the head in bent position.

Symptoms.—Languor, drowsiness, cyanosis, weak tension of pulse, and expanded fontanels.

Treatment of Passive Hyperemia.—Treat the causal condition; administer stimulants—camphor, alcoholics.

ANEMIA

Cerebral anemia occurs in acute loss of blood, in cardiac weakness, and as an associated phenomenon of the various forms of anemia.

Symptoms.—Pallor of the face; tossing of the head to and fro, numbness; eyes rotated upward; cloudiness of corneæ; tonic contractures of the extremities which are usually in position of flexion; the fontanels are retracted (in contradistinction to hydrocephalus and meningitis); the pulse is small and very rapid; the respiration is increased in rapidity and the temperature is low.

A peculiar form of cerebral anemia is the *hydrocephaloid* (Marshall Hall). This is a cerebral state which follows the loss of considerable fluid in an exhausting intestinal catarrh, which is characterized anatomically by anemia and a watery condition of the brain without the collection of fluids in the ventricles.

Treatment.—External and internal stimulation; infusion of normal salt solution hypodermically and by rectum. (See also Cholera Infantum.)

CHRONIC HYDROCEPHALUS

Chronic hydrocephalus is a condition due to the collection of an abnormal amount of fluids within the skull, either in the cerebral ventricles (*hydrocephalus internus*) or between the dura and arachnoid (*hydrocephalus externus* or

FIGURE 72

Atrophic Brain in Hydrocephalus Externus.—Boy two and a half months old. (For explanation, see Fig. 73.)



FIG. 73.—Hydrocephalus externus (congenital). Probably due to congenital syphilis. Boy two and a half months old. The child, which was born spontaneously, showed a rapid increase in the size of the skull; at the age of one month he was picked up by an ambulance while suffering from eclampsia infantum. On examination he was found to have tonic spasms of the arms and legs; the circumference of his head was 40.5 cm. [16.4 in.] (normal 35.4 cm. [14.3 in.]); the large fontanel, which measured 11×13 cm. [4.4×5.4 in.], was bulging, enlarged, and considerably expanded, and its edges irregular, rough, and notched. A lumbar puncture was performed and 40 ccm. of a clear, sterile fluid was removed, which showed a specific gravity of 1.006 and an albumin-content of 1 per cent. A temporary improvement resulted. The child was lost to observation and died at the age of two and a half months from bronchopneumonia. At necropsy 600 ccm. of fluid were removed from the subdural space. The brain was atrophied and lay on the floor of the skull, compressed to the size of a woman's fist (see Fig. 72). It had lost its normal shape. Numerous tense connective-tissue strands extended from its surface to the dura in the region of the large fontanel. Other conditions which were found were a bronchopneumonia and enlargement and consolidation of the spleen and liver; the latter was discolored greenish yellow.

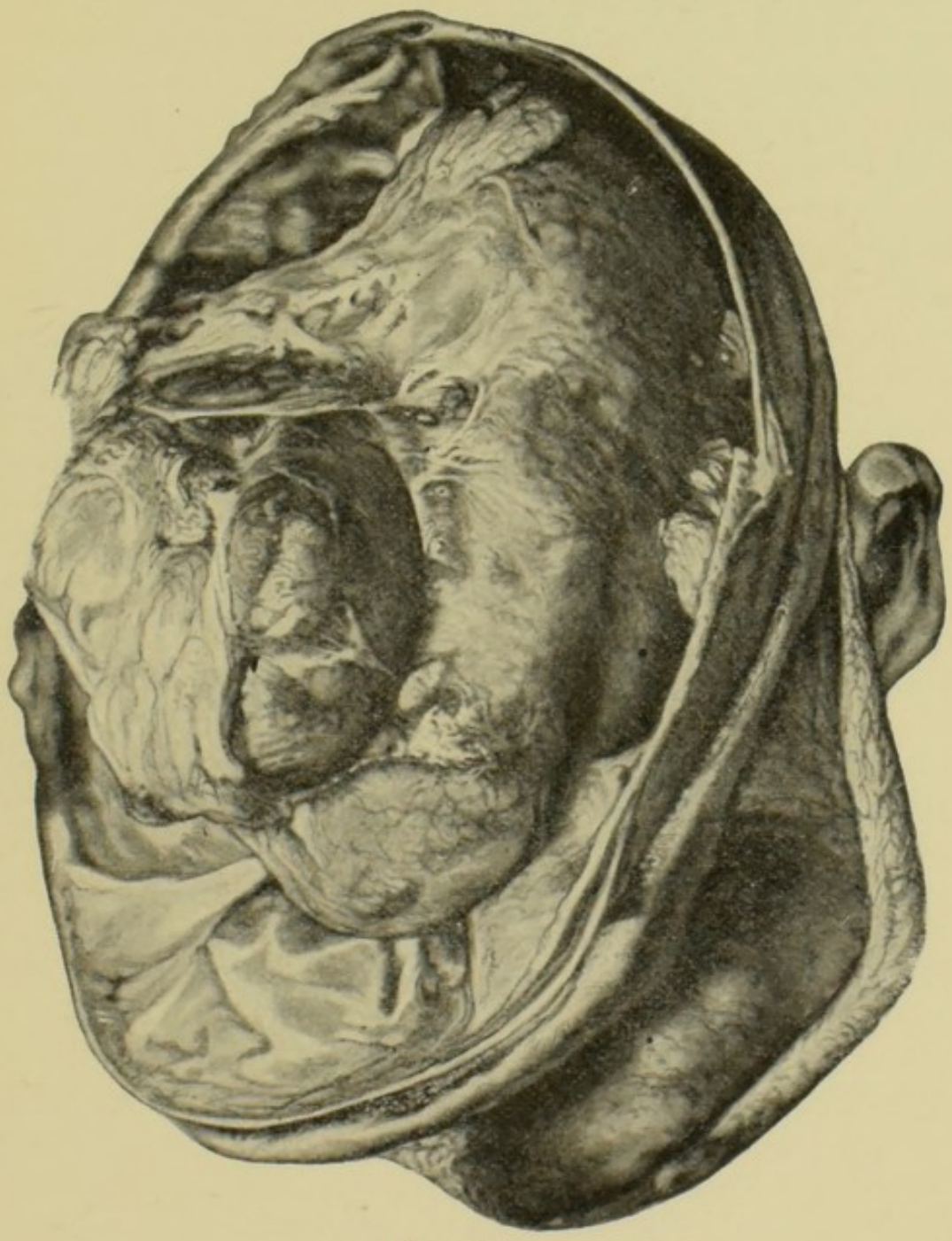


FIG. 72.

intrameningealis); the latter form is by far the rarer. Hydrocephalus is either congenital or acquired.

Etiology.—The internal form of hydrocephalus occurs passively through obstruction to the outflow of the cerebral venous blood on account of pressure upon the vein of Galen, or it may occur actively, following inflammatory disease of the ventricular ependyma and choroid plexus. The external form of hydrocephalus is the result either of intra-uterine or acquired inflammatory processes of the dura and arachnoid, or it arises on account of imperfect development of the brain (agenesis). The true causes of these various changes are unknown. Of etiologic significance are: Congenital syphilitic disease of the vessels and ependyma; rachitis; brain tumors, especially when located at the base; cervical and mediastinal tumors; meningitis; traumatism; pertussis; acute infectious diseases; disturbances in development of the adrenal bodies (Czerny). It frequently occurs in neuro- and psychopathic families. The acquired form of hydrocephalus may develop before as well as after the cranial bones have become fully ossified.

Morbid Anatomy.—Enlarged skull; gaping sutures; thin and partially membranous cranial walls; flattening of the orbital roof and of the sella turcica. In hydrocephalus externus the space between the dura and arachnoid is filled with a fluid which connects the two membranes by means of tense threads and cords. The small pear-shaped brain lies on the floor of the cranial cavity undeveloped, or perfectly formed but compressed. In hydrocephalus internus the convolutions are flattened, the hemispheres fluctuate, the lateral and fourth ventricles are distended with fluid, and in some severe cases the hemispheres are converted into large, thin-walled cysts. The brain substance is atrophied. The fluid is colorless or a light green, it contains a small amount of albumin ($\frac{1}{2}$ per cent.), and occurs in amounts varying from 100 gm. to 2 liters [4.4 pints] and even more (as many as 36 liters [79 pints] have been observed).

Symptoms.—Congenital hydrocephalus may interfere

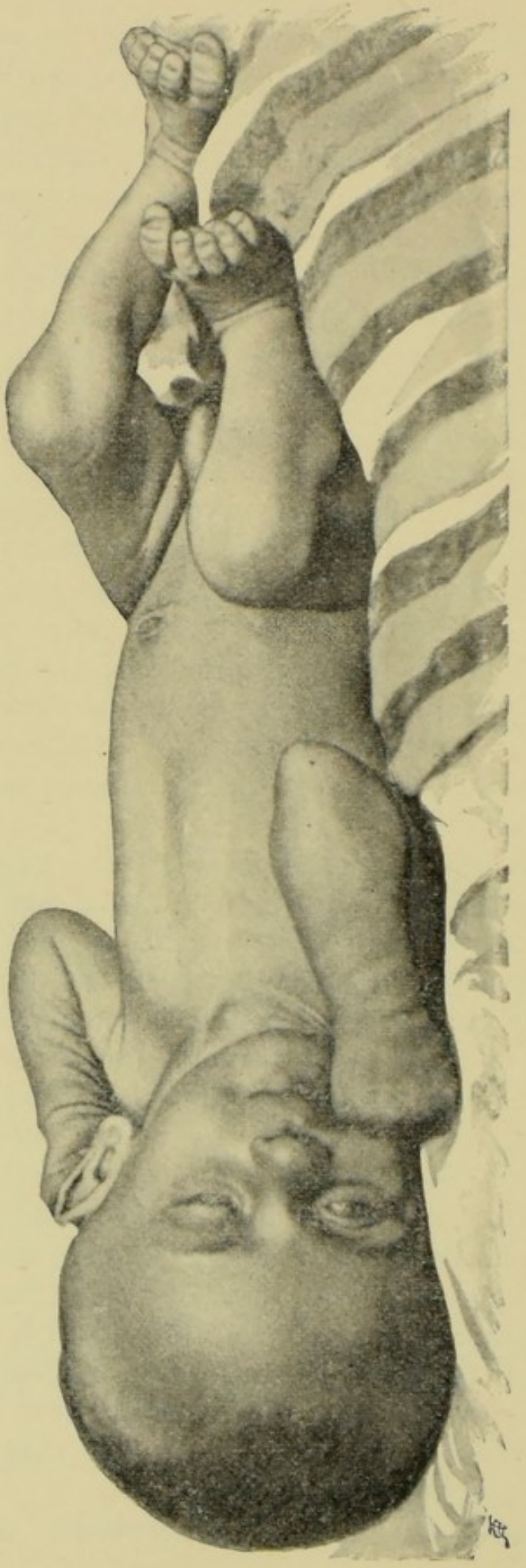


FIG. 74.—Chronic hydrocephalus in an eight-months'-old child. An acute exacerbation occurred during the course of a chronic enteritis, resulting in the increase of the circumference to the extent of 1 cm. [.8 in.]. Flexor spasm in all four extremities ; spasmodic closure of the mouth ; ptosis of the left eye. Further course unknown.

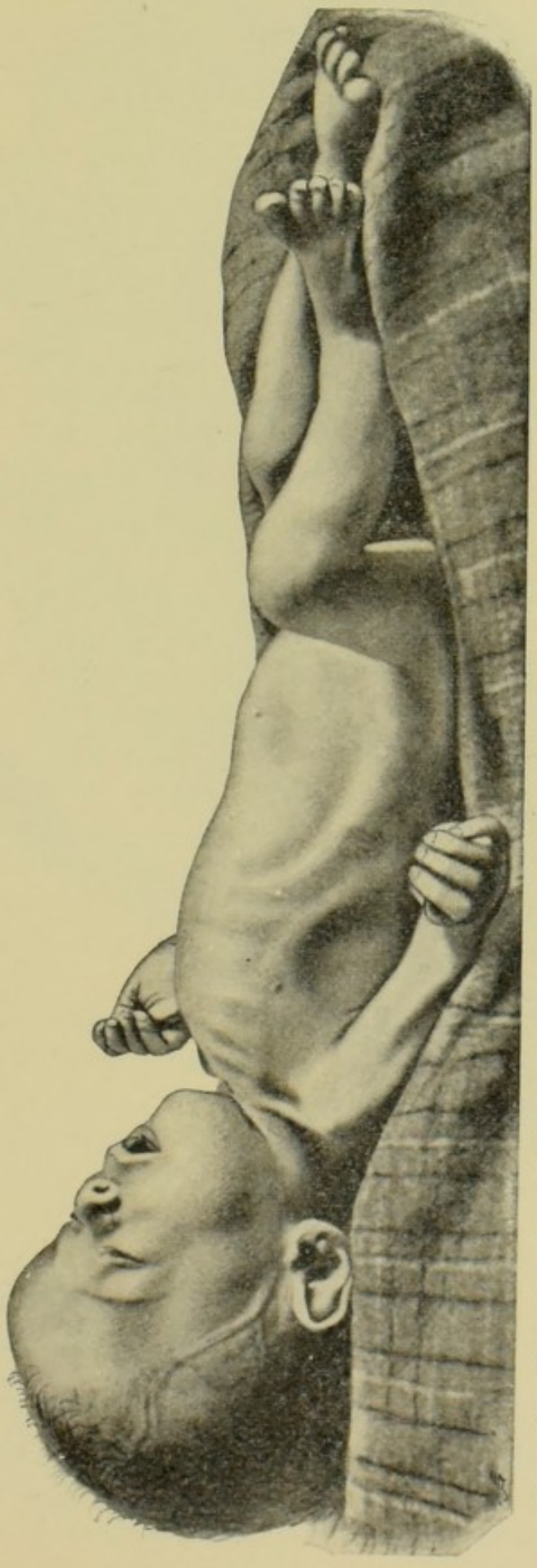


FIG. 75.—Chronic hydrocephalus in a boy fifteen months old, who was decidedly rachitic. Extreme distention of the temporal veins; the considerably enlarged head presents a rachitic bulging of the frontal protuberances; absolute unconsciousness. Photographed one day before death. (Clinic of Escherich, Vienna.)

FIG. 76.—Congenital chronic hydrocephalus; sacral spina bifida. Marked tonic spasms of all four extremities. Opisthotonos. Photographed two days before death. (Clinic of Escherich, Vienna.)

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with birth or it may not become noticeable until after birth. The chief symptom is the constant increase in size of the skull, which in a fully developed case of hydrocephalus presents the following characteristics: The skull, which is increased on all sides, presents a marked contrast with the small and senile face; the forehead bulges forward; the occipital bone is more horizontal than normal and the temporal and parietal bones project laterally. The large fontanel is wider than normal, it is tense and pulsates actively; the sutures gape and the bony plates are soft. Sutures which had closed may again open; veins congested and feel elastic; protruding eyes; a staring, downcast expression of the eyes, with the white sclera visible from above. Frequently the children are unable to support their head. The body may be imperfectly developed; the nourishment is poor, the skin inelastic, yet the appetite is retained and the digestion is good. Although the eyes are usually badly affected (strabismus, nystagmus, choked disk, atrophy of optic nerve, blindness), yet the other organs of special sense remain intact.

Disturbances of Motion.—Tremor and choreic movements in the upper extremities and a spastic—more rarely a paretic—condition of the lower extremities. Contractures, convulsive twitching of single muscle groups, and true eclamptic attacks may arise at any stage of the process. A spastic rigidity of the tendons, especially in the legs, is characteristic of the beginning of the disease (v. Ranke). There is faulty development or retrogression of the psychic function, resulting in various degrees of idiocy.

Course.—The course is chronic and progresses with an increase in the circumference of the skull and the various physical and mental changes. Death is brought on by eclampsia, collapse, or intercurrent diseases. More rarely



FIG. 76.

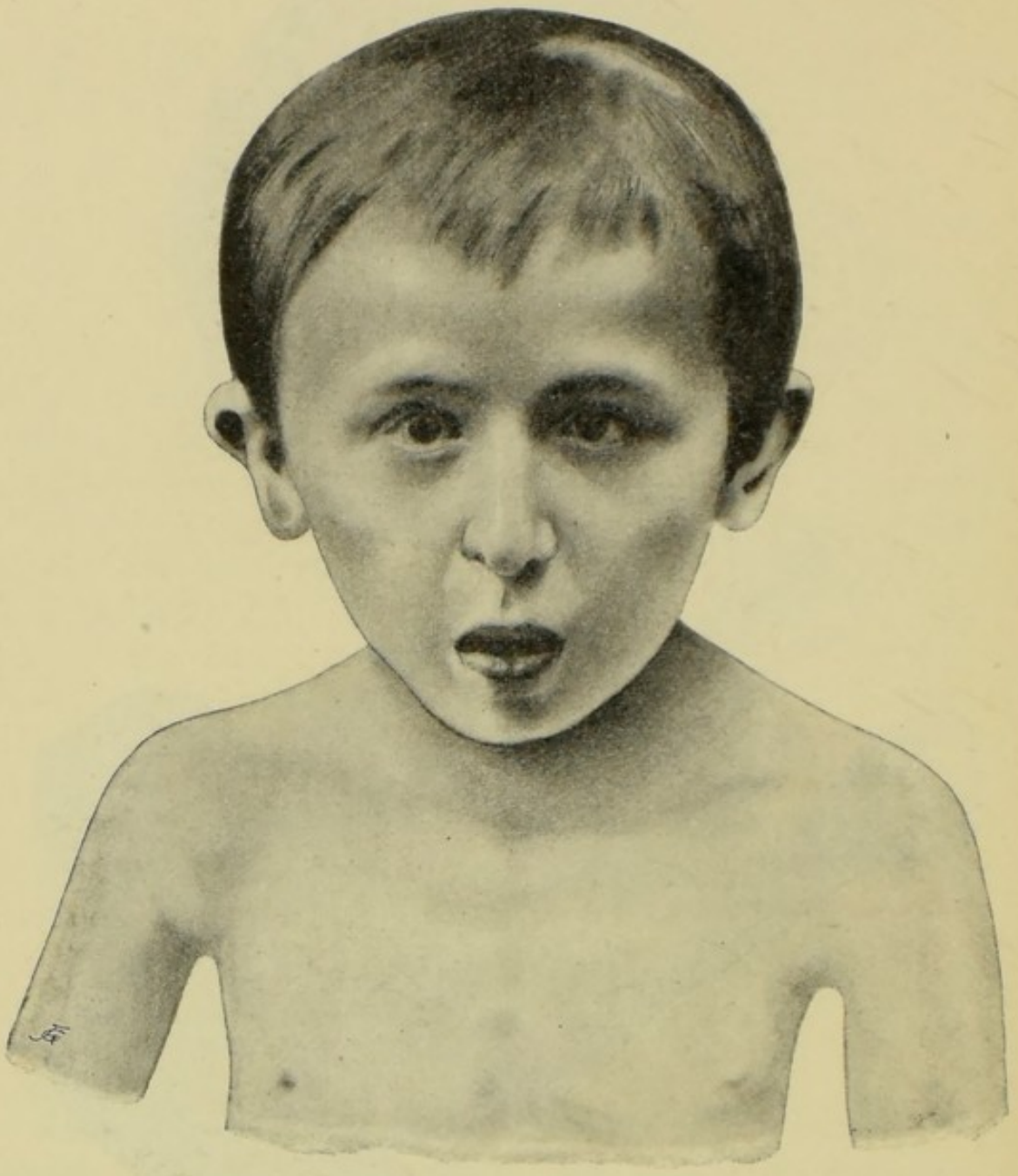


FIG. 77.—Chronic hydrocephalus which has run its course. Imbecility; adenoid vegetations. Boy nine years old. Born prematurely, between seven and eight months. The head was observed to be too large immediately after birth; the fontanels closed in three years. No convulsions. Was taught to walk when two years old, but lost the faculty (rachitis), and did not again learn to walk until the fifth year. Unable to talk correctly until six years. Much headache. The boy is now attending the first school class for the second time, and is making but tolerable headway. Of a phlegmatic yet fearful disposition. The facial expression is somewhat stupid on account of the adenoid vegetations. The cranium is enlarged and its protuberances are prominent; the mouth is held open; mild convergent strabismus. Carious teeth (see Fig. 39); pointed palate.

the disease may take the following courses: Spontaneous cure may occur, but only when the fluid which has col-

lected is small in amount; the condition may also remain at a standstill with a gradual further development of the intellect; rupture externally, either through the nose, eyes, ears, or fontanels, may lead to a cure.

Diagnosis.—This is impossible in mild cases; in doubtful cases it is important to take regular measurements of the head.

Treatment.—If syphilis is suspected, resort to specific treatment externally and internally, with mercury or potassium iodid. Lumbar puncture repeated every few weeks, with the removal of small amounts of fluid, about 30 cc. (Bokay). Puncture the lateral ventricles by way of the large fontanel to one side of the middle line by means of a trocar or aspirating needle and inject tincture of iodin (Pott, von Ranke, Phokas, Gross); paracentesis with drainage (Biedert); suitable training and methodic teaching; carry out rules of general hygiene.

ENCEPHALITIS

The following forms of encephalitis may arise:

Acute, non-suppurative encephalitis, with cerebral irritation, convulsions, fever, etc. The prognosis is not unfavorable. Many of the favorable forms of cerebral irritation without paralysis belong to this division.

Acute suppurative encephalitis (brain abscess) follows injuries to the skull, suppuration in the head, especially of the ear, and septicemia. The onset is sudden and is accompanied by fever and general meningitic symptoms, together with focal phenomena. The differentiation of brain tumor from meningitis is difficult. Treatment is operative when the exact site is known; otherwise it is that of meningitis.

CEREBRAL INFANTILE PALSY

This is no uniform disease process; it represents a group of chronic disturbances of motility, the nature of which indicates the site of the lesion to be in the brain.

FIGS. 78, 79.—Case of hemiplegic type of cerebral infantile paralysis, which has run its course. Thirteen-year-old boy. Contractures of the flexors of the right upper and lower extremities, with typic attitude and slight atrophy of the whole right half of the body. No reactions of degeneration. Mentality slightly defective.

Cerebral infantile palsy develops before birth or during the first three years of life.

Morbid Anatomy.—The primary pathologic changes are meningeal or cerebral hemorrhages, accompanied by a reactive inflammation of the adjacent portions of the brain, or encephalitic processes and thrombosis. As a result we find destruction of the section of the brain involved, including softening, fatty degeneration, and resorption, which lead to loss of brain substance, and the substitution of the latter by serous cysts or hyperostoses (porencephaly) and scar-tissue. Aside from the above changes a diffuse sclerosis (*i. e.*, chronic inflammation of the supporting tissue) is met with; there is frequently a secondary degeneration and atrophy of the pyramidal tracts.

Etiology.—*Before Birth.*—Traumatism to the body or brain of the mother; congenital syphilis, and premature birth.

During Birth.—Continued asphyxiation during protracted labor; premature discharge of the liquor amnii, and compression by the obstetric forceps (Little's disease).

After Birth.—Injuries to the skull; acute infectious diseases, such as scarlet fever, measles, influenza, and meningitis. Some cases present a certain neuropathic predisposition.

Symptoms.—Two types are distinguished, the hemiplegic and the diplegic (Freud).

Hemiplegic Type; Spastic Infantile Hemiplegia; Acute Polioencephalitis (Strümpell).—This type begins suddenly, presenting the picture of an acute infectious disease with high fever, vomiting, delirium, and convulsions. In from a few days to weeks a one-sided flaccid paralysis of the body is found to be present, and it will be noted that

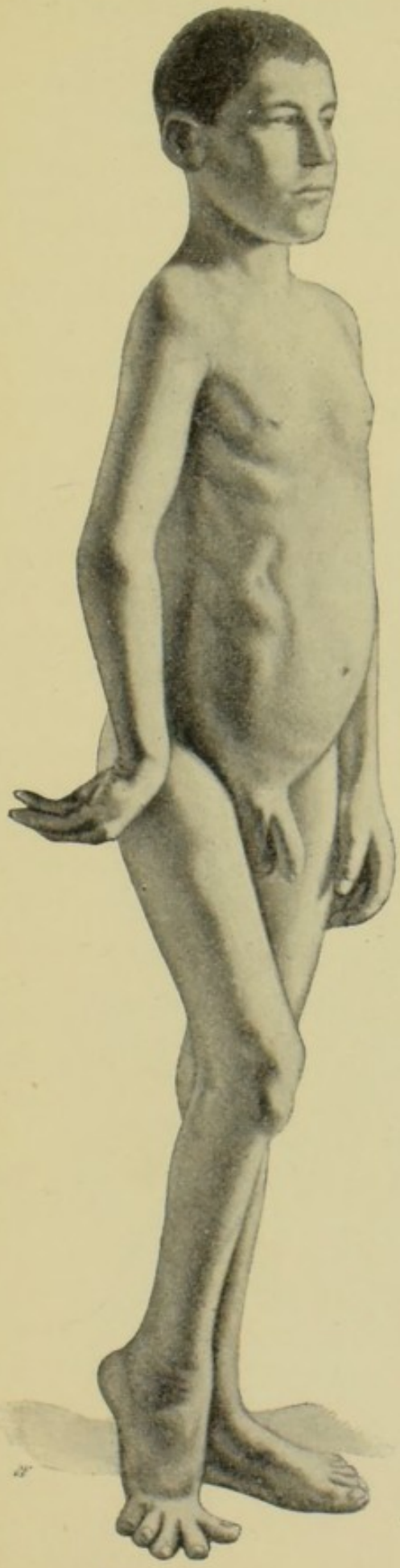


FIG. 78.

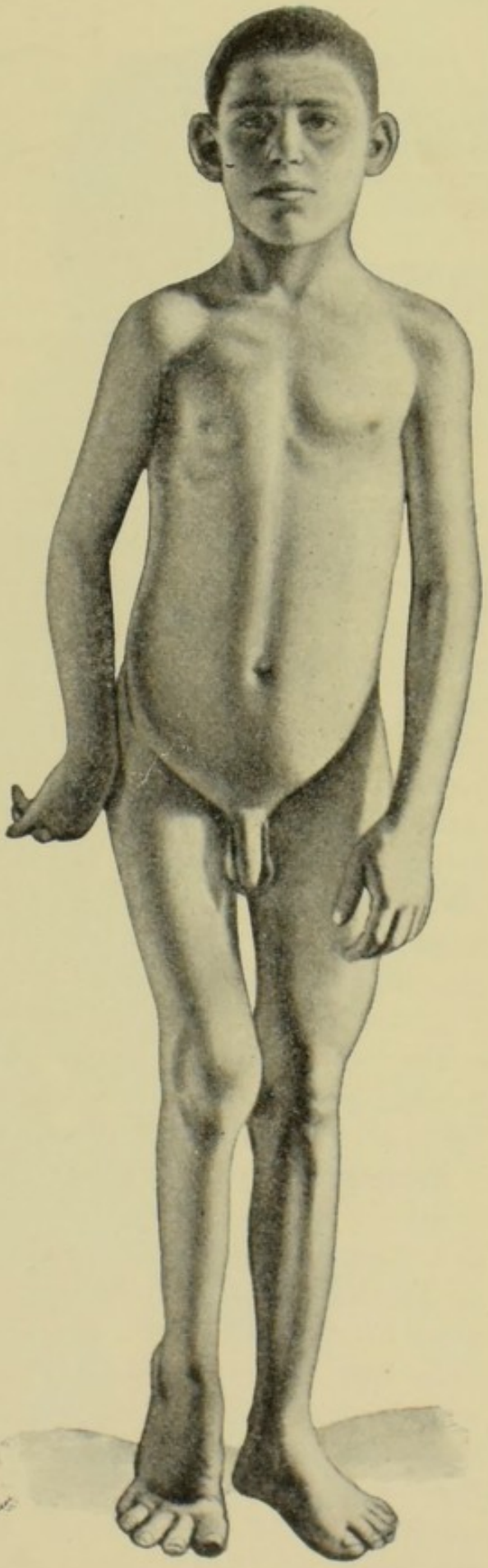


FIG. 79.

FIG. 80.—Congenital spastic rigidity of the extremities (Little's disease). Girl one and a half years old. The rigidity involves all four extremities, as well as the musculature of the neck and face. The legs show the characteristic crossed position on account of marked involvement of the adductors. The left arm is more markedly affected than the right; mask-like appearance of the face. Further course not known.

the arms, legs, and face are involved to a variable extent. The palsy is partial or complete and improves in the course of time to a certain degree. In some cases only a slight helplessness and a tremor of one side are demonstrable.

Resulting Phenomena.—Flexor contractures of the involved extremities, which are held in a characteristic position: The arm is pressed against the trunk, the forearm is held semipronated and bent at right angles at the elbows, the hand is flexed and curved toward the ulna, while the fingers are flexed. The legs are slightly bent at the knee, the foot assumes the equinovarus position, and the toes undergo dorsal flexion. The involved members show athetosis and choreic movements; disturbances of speech exist, also aphasia and defective intelligence, which varies in grade from moral degeneracy to idiocy. Epilepsy develops in the later stages. The muscles are atrophied, but fail to show the reactions of degeneration; the tendon reflexes are increased.

The *diplegic type* includes all of the remaining large variety of forms of cerebral palsies, especially the congenital spastic form of muscular rigidity; the general form of infantile chorea and athetosis.

Congenital spastic rigidity of the muscles (Little's disease) represents a condition which is characterized by the development of marked stiffness and spastic contractures of the legs, with a peculiar gait, within a certain time after birth, usually at the time of mental development. The legs are rotated inwardly, strongly adducted, and often cross each other; the feet are in the position of equinovarus; the upper portion of the body is rigid and bowed forward. The spasms, which are due to increased excitability of the reflexes, tend to disappear upon rest in bed. In mild cases they may only be elicited by



FIG. 80.



FIGS. 81, 82.—Alternating convulsive seizures of the facial musculature following porencephaly in a child four days old, spontaneously born at full term. It presented respiratory disturbances from the first day on. The respiratory pauses lasted from one-half to one minute, were accompanied by marked cyanosis and a disappearing pulse, and alternated with periods of similar or shorter length of fleeting respiration. On the third day the whole right half of the face was seized with tonic spasms, which lasted several hours. On the fourth day similar spasms attacked the left half of the face. These convulsive seizures continued until the sixteenth day, when death occurred. First one side and then the other was involved, but more frequently the right. The necropsy revealed (Prof. Dürck) a loss of the superficial substance (porencephaly) in the region of the lower surface of the pons and the cerebellum, together with connective-tissue and mucoid degeneration of the brain substance in the region of the defect.

rapid passive movements. Reactions of degeneration are absent. The muscular rigidity may be confined to the

legs, in which case the mentality remains intact; or it involves all extremities, converting the children into rigid dolls, and causes cerebral disturbances, strabismus, and defects of intelligence. This general rigidity is usually congenital.

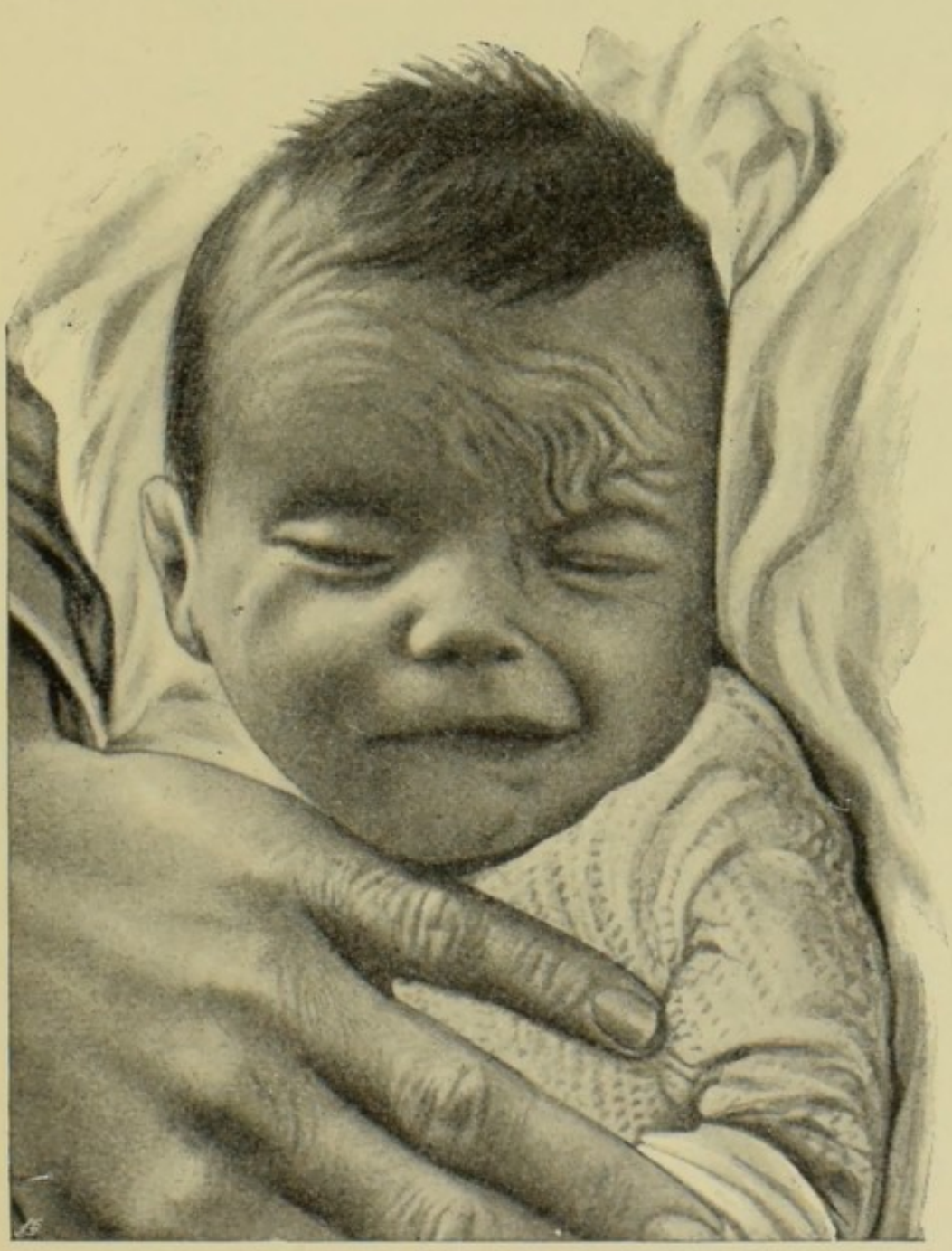


FIG. 82.—See page 222.

General infantile chorea is distinguished from rheumatic chorea by its early appearance, its stationary course, and the development of cerebral manifestations.

Athetosis may be recognized by the relaxation of the contractures, the presence of palsy-like signs, and the spontaneous movements. The fingers are almost constantly in motion, either spreading out, flexing, or grasping.

Diffuse sclerosis probably possesses its own morbid anatomy (diminished size and dense consistence of the cerebral cortex, later, also of the white substance; proliferation of the glia and degeneration of the ganglion cells), but presents few clinical characteristics, so that it is proper to classify it with the infantile palsies (diplegic type).

Disseminated sclerosis, with its circumscribed dense foci in the brain and spinal cord, offers the same symptoms as in adults. The prognosis of cerebral infantile palsies as regards recovery is, with rare exceptions, bad, but good as concerns life; yet cases have been recorded in which death occurred during the convulsions. As a rule incomplete recovery follows with permanent contractures, athetosis, and defective intelligence. The outlook is much brighter when modern orthopedic surgery is resorted to.

Diagnosis.—This is impossible during the acute stages of the hemiplegic form. Of importance later in contradistinction to encephalitis and meningitis are the absence of fever; in contradistinction to tumors, the absence of choked optic disk and the initial manifestations; and in comparison with spinal infantile palsies, the hemiplegic or paraplegic and simultaneous spastic form of the paralysis, the typic contractures, the associated movements, the increased reflexes, the strabismus, and the defective mentality.

Treatment.—In the acute stages depletion and antiphlogistics; later, faradization, massage, dry heat, alcohol rubs, warm baths, and passive movements. (For treatment of the contractures, see Poliomyelitis; of defective mentality, see Idiocy.)

TUMORS

By far the commonest tumors are tubercles, solitary and multiple, which are chiefly located in the cerebellum

and pons; they are sharply outlined, quite dense, and vary in size up to that of a walnut. Other cerebral growths are sarcoma, glioma, gumma, psammoma, and those due to the cysticercus and echinococcus. The symptomatology and therapy present no peculiarities characteristic of childhood. The presence of cerebral tubercles may be suspected when tuberculosis exists elsewhere in the body, and when chronic meningeal manifestations and symptoms of cerebral foci arise.

DISEASES OF THE SPINAL CORD

SPINAL INFANTILE PARALYSIS

(*Acute Anterior Poliomyelitis*)

This is a degenerative paralysis of single extremities, which has an acute onset, runs a subsiding course, and is probably due to an infectious myelitic process in the anterior horns of the cord. The most frequent subjects with this condition are children from one and a half to four years.

Morbid Anatomy.—In recent cases the substance of the anterior horns is softened; microscopically the multipolar cells are seen to be degenerated and the interstitial tissue inflamed. In older cases we note atrophy and sclerosis of one anterior horn, with disappearance of all ganglion cells, secondary degeneration of the anterior motor roots and of the nerves, muscles, and tendons supplied by them. [According to the researches of Marie and Goldscheider, it is shown that the anterior horns are supplied by the anterior branches of the spinal arteries. The areas supplied by these branches have been found necrosed and softened, the vessels blocked, and the nerve-cells completely destroyed.—Ed.]

Symptoms.—The disease may be divided into four stages (Fischl):

Initial Stage.—It begins in the midst of perfect health, apparently as an acute infectious disease, including high fever, headache, slight somnolence, and, more rarely, con-

vulsions and stupor. The duration is from thirty-six to forty-eight hours.

Stage of Fully Developed Paralysis.—The acute symptoms disappear and a flaccid paralysis remains, which involves several extremities. As a rule the paralysis affects both legs and one arm, an arm and a leg on opposite sides, both legs, or all four extremities. The excitability of the muscles to the faradic current rapidly lessens and the paralysis reaches its highest point of development. The duration of this stage is from one to—at the most—two weeks.

Stage of Abatement of Paralysis.—The paralyses improve by degrees, and the improvement affects a whole member or only individual muscle groups. The paralyzed parts show beginning reactions of degeneration, and the diseased muscles react to the galvanic current with sluggish vermicular twitchings; the An.Cl.C. exceeds the Ca.Cl.C. The improvement ceases after one, two, or more months, and the following stage is reached:

Stage of Completed Paralysis and Sequelæ.—The paralysis of a leg or an arm or both legs now becomes permanent. Of the upper extremities, the deltoid and shoulder muscles, the extensors or flexors of the forearm are most frequently involved, while in the lower extremities the extensors and peroneal muscles are mostly attacked.

The usual **sequelæ** are: Atrophy and fatty degeneration of the muscles and tendons; sometimes these are thickened because of increased fatty growth; bony growth is delayed or bony absorption takes place. The paralyzed extremities are wasted, loose at the joints, the muscles are pale and withered, the tendons are thin and relax. The shoulder, when attacked, appears flattened and the finger can be introduced between the acromion and the humerus.

The skin temperature is subnormal and the affected extremities are cyanosed. Muscular reaction to electric stimulation is lessened or wholly absent. The tendon and skin reflexes are absent, but sensation remains normal. Deformities result from the action of antagonistic muscles, from the weight of individual parts and of the



FIG. 83.—Spinal infantile paralysis in the stage of fully developed palsy. Three-year-old girl. The flaccid paralysis of the right leg is shared by the quadriceps, peroneal, and extensor communis digitorum muscles. The use of the leg was restored by periosteal tendon transplantation (F. Lange).

whole body. These include paralytic club-foot and talipes equinus and, more rarely, talipes calcaneus and club-hand. After the formation of these deformities spontaneous cure is hopeless and the disease has reached its termination.

Prognosis.—Complete cure as well as an unfavorable ending are rare. As a rule the disease leads to some permanent deformity. By means of timely and subsequent treatment, especially by resorting to modern orthopedic methods, it is possible to secure functional improvement and cure. In the third stage the condition of the muscles as regards electric reactions is of prognostic significance.

Diagnosis.—This cannot be established in the acute stages. Indicative of this disease are: The flaccid paralysis, which at the beginning is widely spread, but later limited and stationary, and which, accordingly, runs a retrogressive course; the degenerative atrophy; the loss of reflexes; the retained sensibility and sphincter function. A conclusion as to what muscles are paralyzed and to what extent they are affected may be reached by determining what movements are possible by palpation of the contracted tendons (F. Lange).

Treatment.—In the acute stage resort to an antiphlogistic regimen, deplete through the bowels, and keep at rest in bed for several weeks. If the paralyses are evident and the fever has disappeared, use electricity over a long period of time, also massage, passive movements, and gymnastics. At the beginning apply a weak current of electricity by passing the cathodal electrode over the paralyzed muscle, while the anodal pole is held over the part of the spinal cord which represents the affected area. Later employ stronger stimulation by means of the faradic current (at first every other day, later, daily). Prevent contractures by the application of splints, which fix and hold the member in a correct position. (The splints are only worn at night.) Corrective manipulation is also recommended; periosteal transplantation of tendons, with the insertion of silk tendons, according to the

recent simplified operative methods (Lange); fix loose joints by means of arthrodesis.

TRANSVERSE MYELITIS

Transverse inflammation of the spinal cord is especially likely to follow a spondylitis—the so-called *compression myelitis*. It is then due to the direct pressure of the caseous exudate, the deformed vertebræ, to disturbance of the circulation, or it is an extension of the inflammatory process. Aside from these etiologic factors it may also develop as a termination of an acute infectious disease, or it may be caused by traumatism, exposure to wet, or syphilis.

Morbid Anatomy.—In recent cases there is a slight discoloration and softening of the cord, while in older cases the spinal cord is smaller and harder than normal. Microscopically we note a small-celled infiltration and swelling of the axis cylinders and of the connective tissue; degeneration of the medullary sheaths and ganglion cells; fatty granules; later the connective tissue and glia show proliferation; ascending and descending degeneration of the spinal tracts.

Symptoms.—Transverse myelitis may be sudden or gradual in onset. Paresthesiæ, pains, and, later, hyperesthesiæ and anesthesiæ. Spasmodic twitchings of the extremities. Paralysis of that portion of the body innervated by the spinal cord, varying according to the location of the affection. In diseases of the lumbar cord, flaccid paralysis of the lower extremities with atrophy, reactions of degeneration, loss of reflexes, disturbances of sensation, paralysis of the rectum, and decubitus. When the dorsal cord is involved, spastic paraplegia with increased reflexes, unaccompanied by atrophy and reactions of degeneration; otherwise the symptoms are the same. In involvement of the cervical cord, paralysis of the arms is added to the symptoms of disease of the dorsal cord. The so-called "Brown-Sequard paralysis," or unilateral lesions, consisting of motor palsy and increased reflexes on the diseased side and sympathetic

palsy on the sound side, occurs in disease of one side of the spinal cord (crossing of sensory fibers after entering the cord, straight course of the motor fibers).

The **prognosis** is usually unfavorable, excepting in cases following syphilis or infectious diseases. The course is chronic and is dependent upon the causal condition.

Treatment.—If due to spondylitis or syphilis, direct treatment for those diseases. In case of syphilis and also in other forms of myelitis which are not tuberculous, administer potassium iodid internally and paint the site externally with iodin. Massage and electricity for the muscles. Careful nursing in order to avoid decubitus, which is so frequent, and the disturbances of the bladder with their sequelæ.

FRIEDREICH'S (HEREDITARY) ATAXIA

This is a family disease occurring before puberty, which is caused by degeneration of the posterior columns of the cord. It is characterized by ataxic movements of the arms and legs, nystagmus, muscular disturbance of speech, loss of knee-jerks, extremely chronic and an incurable course.

SPASTIC SPINAL PARALYSIS

This is a disease of later childhood, consisting of a gradually developing spastic paralysis of the legs accompanied by contractures of the adductors of the femur and of the muscles of the calves, crossing of the legs, pes equinus, increased reflexes without atrophy, reactions of degeneration or cerebral manifestations. (Little's disease occurs congenitally, or it arises during the first period of childhood.)

FUNCTIONAL NERVOUS DISEASES

ECLAMPSIA

(*Convulsions; Spasms; Eclampsia Infantum*)

Clonic spasmodic convulsions accompanied by *unconsciousness.* These represent no distinct disease, but

rather a symptom, the cause of which is still unknown in many cases.

Etiology.—*Primary Reflex or Functional Eclampsia.*—The convulsions arise spontaneously or as the result of sensory disturbances (intestinal parasites, foreign bodies, injuries, psychic or sensory impressions). Of predisposing influence is the physiologic increased tendency to convulsions in children (spasmophilia), the cause of which, according to Soltmann, lies in the imperfect development of the psychomotor inhibitory center together with increased reflex excitability of the peripheral nerves. Eclampsia may also precede true epilepsy.

Secondary symptomatic eclampsia occurs in diseases of the brain, meningitis, tumors, hydrocephalus, encephalitis, anemia, hyperemia (pertussis), and in otitis media. It may also be of hematogenic origin, that is, the convulsions may follow the presence of toxins in the blood, or the poison of intestinal bacteria, or they may be brought on by fever, anomalies of metabolism, rachitis, affections of the gastro-intestinal tract, or overfeeding. Convulsions have been known to occur at the onset of acute infectious diseases instead of the initial chill; on account of carbonic-acid intoxication in laryngospasm or pneumonia; in uremia or when the blood contains an insufficient amount of water. Eclampsia is also a frequent complication of tetany and laryngospasm, especially during the first eighteen months of life.

Symptoms.—The attack begins suddenly, with pallor of the face, a vacant stare, and rolling of the eyes. The following conditions arise simultaneously: Loss of consciousness, tonic rigidity of the head and of the extremities; flexion of the fingers, extension of the legs, and the pes equinus or talipes calcaneus position of the feet. After a few seconds we observe a clonic twitching around the angles of the mouth, distorted facial expression, tightly set jaws; in older children gnashing of teeth; tossing of the head to and fro; rhythmic twitching of the extremities, as if electric shocks were given. Cyanosis around the mouth and nose, escape of froth and, fre-

quently, bloody saliva; the pupils are dilated; the corneal reflex is lost, complete failure of reaction to all external stimulation; incontinence of urine and feces. The respiration is shallow and interrupted by spasmodic pauses, while the pulse is irregular and unequal.

Such an attack lasts but a few moments, after which the spasms successively disappear, the face becomes flushed and quiet, the child falls into a sleep, the beginning of which is interrupted by single twitchings. A single attack is very rare; as a rule, on the contrary, the convulsive seizures recur at longer or shorter intervals of days, weeks, or months. In some cases a series of convulsions occur in quick succession, even before the patient awakens from a previous attack. In the severest forms the patient is in a continuous convulsive state for several hours, which is only interrupted by short periods of sleep. Exhaustion and venous stasis, on account of inhibition of respiration, may lead to death. The intensity of the attacks varies from the severest to the lightest, in which the turning of the eyes and the slight twitchings (as may also be seen in healthy nurslings when asleep) are barely noticed by the parents. Loss of consciousness is, however, constantly present.

Diagnosis.—Inasmuch as many diseases may begin with convulsions, the diagnosis of true eclampsia is difficult at the commencement. Careful examination of all organs will prevent errors and the further course of the disease will establish the diagnosis. Eclampsia is distinguished from organic brain disease by permanent tension of the fontanels, whereas in convulsions the tension is only during the attack; long duration (more than twelve hours) of the attack, as well as prolonged unilateral convulsions, indicate cerebral affection. In favor of epilepsy are: The recurrence of attacks over a long period of time without any apparent cause; the presence of an hereditary predisposition, and the late occurrence at the end of the second year of life.

Prognosis.—This should be guarded and not made until after long observation of the child, both during the

attack and the interval. The prospects are dependent upon the severity and frequency of the attacks, as well as the nature of the original causal condition. The reflex and hematogenic forms of convulsion run, as a rule, a favorable course. Death may occur during the attack because of asphyxiation or cerebral hemorrhage. Convulsions are not infrequently followed by paralysis, defective mentality, or true epilepsy.

Treatment of the Attack.—Remove clothing; stimulate with cold water; chloroform inhalations, chloral enemata (1.0 gm. to 30.0 cc. milk, starch-water, for two injections); tepid baths with cold applications to the head. In hyperemia of the brain compress the carotids (Seitz) or apply leeches. After the attack deplete by way of the bowels (calomel) and skin (stimulation of the skin, heat). Low diet. Should the attacks frequently recur, give the bromids with or without chloral; if rachitis exists administer phosphorus and resort to antirachitic treatment. [In those cases associated with high fever, antipyrin combined with bromids sometimes gives excellent results. In the cases of continuous or protracted convulsions small doses of morphin—$\frac{1}{500}$ to $\frac{1}{100}$ gr.—may be given hypodermically.—Ed.]

TETANY

Tetany is a functional neurosis depending upon a hyperexcitability of the peripheral nervous system, which chiefly attacks the rachitic children of poor parents during the first two years of life, especially in the spring months.

Etiology.—Disturbances of the gastro-intestinal tract, foul atmosphere, artificial (cows' milk) feeding (Finkelstein).

Morbid Anatomy.—Thus far no uniform change has been noted.

Symptoms.—A symmetric tonic muscular contraction, beginning with the fingers, then the hands and toes, and which does not involve the arms and legs until after the lapse of considerable time; the musculature of the trunk is very rarely attacked. Characteristic position of the

hands—the fingers are extended at the phalangeal joints and flexed at the metacarpal joints, while the thumb is turned in a volar direction—*obstetric hand* (Fig. 85); the legs are extended and the toes flexed. These contractures

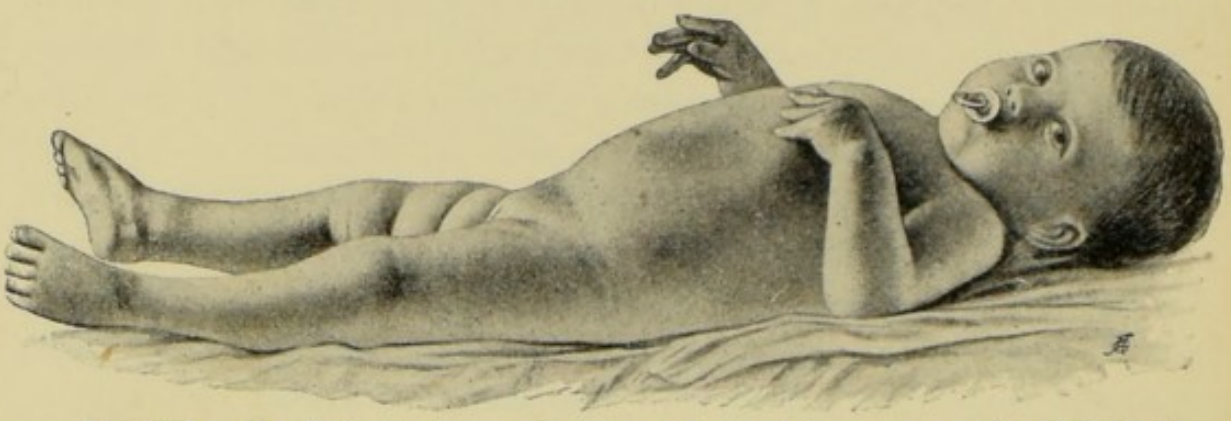


FIG. 84.—Persistent form of tetany in a girl a year and a half old. Tetanic contractures of the arms and legs; hands in the "obstetric" position; feet in plantar flexion. The convulsions lasted three days uninterruptedly and disappeared after thorough purging. Trousseau's sign, the facial phenomenon, and the heightened electric excitability (Ca.Cl.C. 1.0 M. A.: Ca.O.C. 3.2 M.A.) remained demonstrable for a long time—*latent form*. Etiology: Chronic constipation in a child living under unfavorable conditions of life.

may continue as a permanent form of tetany (Escherich) or they disappear after a certain time—*intermittent form;* the latter type is most frequently observed. Dur-

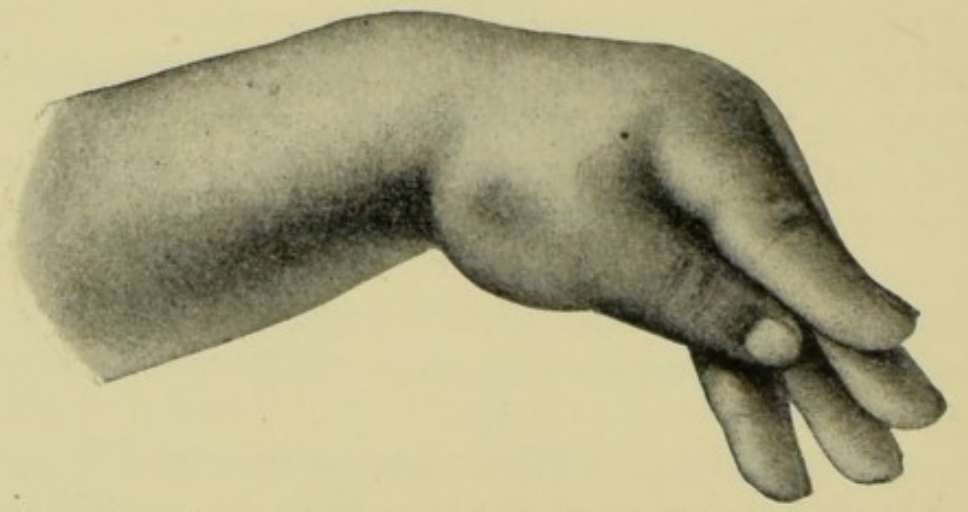


FIG. 85.—The obstetric hand of tetany. (From Fig. 84.)

ing the interval the hypersensitiveness persists and is manifested by the so-called *latent symptoms*. These are, moreover, in some cases the only expression of the disease—*latent tetany*. The symptoms of latent tetany are:

Trousseau's Phenomenon.—Pressure upon the internal bicipital groove produces after a few minutes the typic obstetric hand. This sign when present is pathognomonic, but it may be absent in some cases.

Erb's Phenomenon.—Increased excitability of the motor nerves to the galvanic current. Tested by means of the Stinzing normal electrode placed at the median nerve in the elbow and the indifferent electrode on the sternum. It is noted that the Ca.Cl.C. requires less than 0.7 milliampères (about 1.5 milliampères normal); the An.O.C. is greater than the An.Cl.C., and, above all things, the weak current required for the Ca.O.C. (1.94 milliampères in manifest and 2 23 milliampères in latent tetany; 8.22 = normal). This is a constant and likewise pathognomonic symptom (Thiemich).

The Chvostek, or Facial Nerve, Phenomenon.—Heightened excitability to mechanical stimuli. Tapping or palpating the facial nerve between the zygoma and the angle of the mouth causes a lightning-like twitching of the whole or part of the facial musculature. The symptom is usually present, but occurs also in other neuroses.

Laryngospasm.—Spasm of the glottis is noted in many cases of tetany, especially in the latent form. The first two symptoms are called obligate, the last two, facultative latent symptoms. Eclamptic attacks are also observed throughout the course of tetany.

Course and Prognosis.—The duration is from three to five weeks (Loos, Kirchgässer). The prognosis is favorable, but serious in case of laryngospasm, eclampsia, and severe rachitis. Marked improvement of the tetanic and laryngospastic phenomena follows the substitution of cows' milk by nourishment with infants' foods (Finkelstein).

Diagnosis.—In the absence of manifest contractures one of the "obligate" symptoms suffices for the diagnosis of latent tetany. Every case of laryngospasm should be tested for latent symptoms.

Treatment.—Empty the intestinal tract; correct the diet. Combat the nervous hyperexcitability with phos-

phorus, bromids, and chloral. Attend to hygienic conditions of the dwelling; tepid baths. Temporary substitution of the cows' milk by feeding with infants' foods. [This last statement is not in accord with the most accepted teaching in regard to the treatment of these neuroses. Fresh cows' milk, properly modified or diluted, is considered the best substitute for mother's milk and is particularly indicated in these cases.—ED.]

PSEUDOTETANUS (ESCHERICH)

Pseudotetanus presents a condition similar to traumatic tetanus, which is characterized by an ascending rigidity of the whole body and face, without affecting the arms and hands. Trousseau's and Erb's signs are absent; the convulsion is lessened when the body is at rest, but increased by external stimuli. It runs a favorable course of from four to six weeks.

LARYNGOSPASM

(*Spasm of the Glottis; Laryngismus Stridulus*)

Laryngospasm is an apnea occurring in attacks due to convulsive seizures of the glottic muscles and the other muscles of respiration. It frequently runs in families and occurs during the first years of life. It is an independent disease as well as a nervous phenomenon of rachitis, and a partial manifestation of eclampsia and tetany. Laryngospasm occurs most frequently in association with these three affections during the spring months of the year; and may also follow whooping-cough, disturbances of digestion, and difficult dentition. As the direct cause of the attack we note any mental alteration, also crying, drinking, exposure to cold, and catarrh. At the height of the disease attacks occur apparently without cause.

Symptoms.—The laryngospastic attack begins with sudden cessation of respiration, the head is hyperextended, the eyes have a staring expression, the patient is in fear of impending death, and the face is pale and cyanotic; to

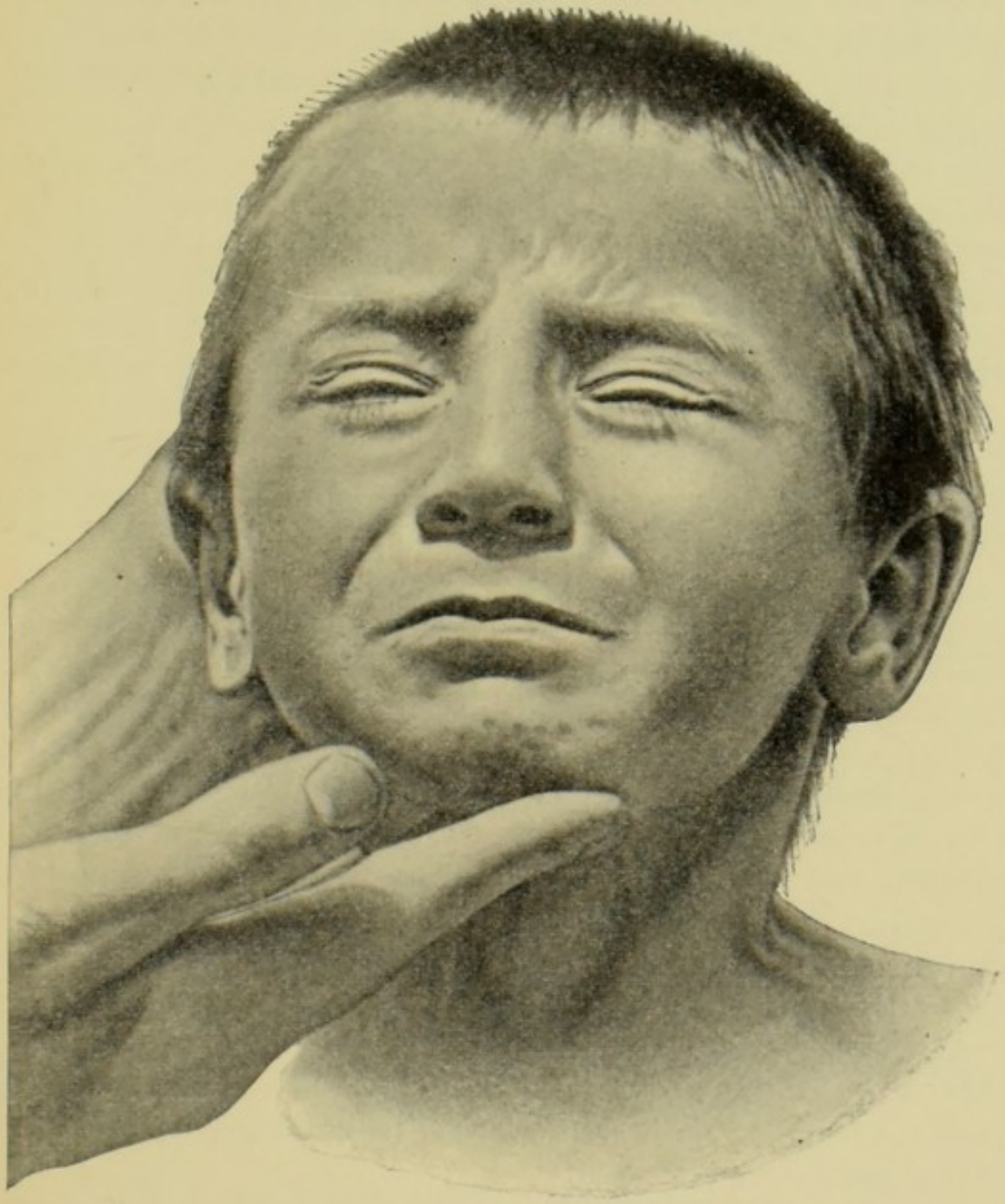


FIG. 86.—Pseudotetanus. Nine-year-old boy. Tonic spasms of the facial muscles; characteristic grinning expression; the teeth are tightly pressed upon each other. Contraction of the platysma muscle. The patient shows rigidity of the whole body, with the exception of the arms, hands, and eyes. The convulsion disappears largely during rest. The ingestion of food is never prevented. Recovery in eight weeks. (Escherich's Clinic, Vienna.)

these are added frequent twitchings of the face and extremities or a tonic rigidity of the latter. After the lapse of a few seconds (ten to sixty) breathing is begun in

the form of short, rapidly repeated, loud and crowing inspirations, which are followed by an expiratory movement. Respiration then sets in, and with its return all other symptoms disappear and the child becomes drowsy. The attacks vary considerably in number and in severity. We may see the various stages, from the mildest form, in which the apnea lasts but a few seconds with a few sighing respiratory movements, to the severest forms, which terminate fatally. They occur either every few days or daily and, indeed, as many as twenty, thirty, or more may occur in one day.

Course and Prognosis.—The disease lasts at least three months. Three stages may be distinguished (Bendix): The attacks increase in intensity and frequency for three or four weeks, then the symptoms remain stationary for from four to eight weeks, after which they gradually disappear for four more weeks. The prognosis is likely to be favorable, yet there is a possibility of fatal asphyxiation during an attack, and we must not, therefore, neglect to watch the tongue to prevent aspiration. The outlook is less favorable in severe rachitis or when the disease is complicated by pneumonia or pertussis. [Laryngitis occurring during an attack may lead to laryngeal stenosis and necessitate surgical intervention. The prognosis in these cases is very grave.—Ed.]

Diagnosis.—The sudden attack of apnea with the crowing inspiration is absolutely characteristic. The so-called apnea of many children is not laryngospasm, but an almost harmless affection.

Treatment of the Attack.—Spray or douche with cold water; introduce the finger into the mouth, lift the epiglottis, thus encouraging swallowing movements, and draw the tongue forward to prevent the possibility of its being aspirated. Slap the back; apply a hot sponge to the neck and, if necessary, artificial respiration, intubation, or tracheotomy. When the attacks follow each other in close succession give chloral enemata (0.5 gm.) and inhalations of oxygen. To lessen the nervous excitability administer phosphorus, the bromids, infants'

foods (Fischbein, Finkelstein). To improve the general condition institute an antirachitic, general hygienic, and dietetic regimen.

SPASMUS NUTANS

During the months of primary dentition rhythmic contractions are noted in the region of the sternomastoid muscle and the rotators of the head which are supplied by the brachial plexus. They consist of an almost ceaseless rotatory and nodding movement of the head, accompanied by nystagmus. The movements cease when the head is held tightly by the physician (which increases the nystagmus), during sleep, and darkness. After a course of months the condition terminates favorably.

Etiology.—The disease is undoubtedly a nutritional disorder. Whether due to a disturbance in the ocular dynamics or to insufficiently lighted rooms is not definitely settled.

Treatment.—Well-lighted dwellings; phosphorus.

SALAAM CONVULSIONS

These consist of a nodding of the head and upper part of the body, unaccompanied by nystagmus, but associated with disturbances of intelligence and epileptiform seizures. The condition is frequently fatal.

CONGENITAL MYOTONIA

(*Thomsen's Disease*)

This is an hereditary primary parenchymatous myopathy occurring in families, which is characterized by a tonic rigidity and contraction of the voluntary muscles when voluntary movements are made. It runs a progressive, very chronic, and incurable course.

Treatment.—Tepid baths and corrective exercises.

PERIPHERAL PARALYSES

Facial paralysis is due to disease of the petrous portion of the temporal bone, hemorrhages, cerebral affections, traumatism during birth, and exposure to cold. Typic obstetric paralysis follows any form of injury or obstruction at birth. The palsy may involve the deltoid, the infraspinatus, the brachialis anticus, or biceps muscles. A flaccid paralysis of the arm and the shoulder is noted, while movement of the hand and finger is preserved.

Treatment.—Electricity and massage.

CHOREA MINOR

(*St. Vitus' Dance*)

This is a psychomotor neurosis characterized by short involuntary movements of the different voluntary muscles. These motions are similar to those to which the child has already been trained and occur in children from two to fifteen years of age, but most frequently after the seventh year. Girls are more often affected than boys (70 per cent.).

Etiology.—Chorea is an infectious disease due to the injurious effects of bacteria, which are identical with those of rheumatic arthritis and endocarditis and are localized in the psychomotor centers of the cerebral cortex and in the pyramidal tracts. The affection, therefore, corresponds to rheumatism and endocarditis, and may occur simultaneously with them or in the form of rheumatic recurrence, or as the first manifestation of a later rheumatic affection (Henry Meyer). Likewise it may develop after diseases which are closely related to rheumatism, such as gonorrhea, angina lacunaris, and erythema nodosum. According to Heubner, chorea is the "infantile rheumatic equivalent." Aside from these conditions it may also follow influenza, scarlet fever, or measles.

Morbid Anatomy.—The findings in the brain are indefinite and consist in arteritis (Reichard) and the deposition of colloid bodies (Hudo-Vernig); minute recent endocardial deposits in the mitral valve with insufficiency of

that valve and acute nephritis. Various forms of bacteria (bacilli, streptococci, and staphylococci) are found in the blood, in the brain, in the endocardial deposits, and in the joints.

Symptoms.—The general health fails, the disposition is altered, and abnormal movements gradually set in. The face begins to twitch and there is involuntary shrugging of the shoulders; the child is restless when sitting and plays with the fingers. Later the arms and fingers are in constant motion; the arms and shoulders are thrown about and the face is distorted into all possible grimaces. Together with these are noted various expressions of emotion which the child has already been trained to show naturally, such as grief, anger, mirth, and fear; these movements exhibit somewhat of the theatrical and are often comical. They present themselves during voluntary movements of the hands and interfere considerably with them. Likewise there is a disturbance in speech, mastication, writing, dressing, gait, respiration, etc., and often of the movements of the heart. The phenomena are increased by voluntary actions, in psychic impressions and affections, and when the child knows it is being observed, but cease almost entirely during sleep. As the disease progresses the appetite is lost, emaciation, pallor, and irritability increase, and the ill humor and the fearful disposition grow worse. In the severest types we note flail-like movements of the extremities, inability to stand, walk, or swallow—*muscle anarchy* (Eulenberg). Such cases also usually present psychic dissociation, confusion, and halluciations. St. Vitus' dance, which, as a rule, is worse on one side at the beginning, may continue permanently on one side as hemichorea.

Paralytic chorea is a rare form, in which the choreiform movements are associated with marked weakness of the muscles of the extremities.

Electric chorea consists in jerky and rhythmic movements of the head and extremities (usually upper), which set in when the muscles are comparatively at rest. This form, like chorea major and imitative chorea, which tends

to become rapidly widespread in schools, should be classified with hysteria.

Course and Prognosis.—Chorea lasts from two to three months (forty-four days on the average, Heubner), but may continue for years. It has a tendency to recurrence, which does not disappear until puberty. The prognosis is, on the whole, favorable, provided it does not set in with endocarditis or rheumatism. The choreic forms of psychoses always disappear.

Diagnosis.—The coördinated and involuntary movements, which cease during sleep, and the undisturbed mentality are of significance.

Treatment.—Rest in bed for several weeks (followed by sitting up in bed); mental rest. Nourishing, but easily digestible diet. Try salicylic acid; good results follow the use of arsenic, with or without iron. In severe cases give the bromids, chloral, or morphin. In convalescence employ suggestive treatment; rest the mind; country life.

EPILEPSY

This consists of attacks of unconsciousness, occurring at intervals, accompanied by clonic and tonic convulsions and followed by loss of memory. About 60 per cent. of all cases of epilepsy occur before the sixteenth year. In the earlier years of childhood they present largely the symptoms of eclampsia, from which they are distinguished with difficulty. At this age the attacks do not recur continually and, indeed, the convulsions of the nursing period may recur after the lapse of years.

Etiology.—Hereditary influence, especially when the parents suffer from epilepsy or alcoholism; injuries to the head; increase of intracranial pressure on account of new growths, exudates, or thickening of the cranial bones; irritation of the peripheral nerves by painful scars, foreign bodies, new growths, and auto-intoxication (Monti)—*reflex epilepsy;* abuse of alcohol in children; persistent infantile cerebral palsies, which are especially likely to cause Jacksonian epilepsy. Immediate causes

of the attacks are severe emotions, mental strain, and dietetic indiscretions; yet these factors frequently do not exist. The attack is usually preceded by an aura, which may assume many forms: Headache, hallucinations, ringing in the ears, nodding movements of the head, cardiac palpitation; sensation of strangulation, tremor, paresthesiæ, etc. The aura may persist in children as an independent affection for many years.

Symptoms.—The attack itself begins with a fixed expression of the face and, as a rule, with a loud cry, and the child falls to the floor unconscious; the latter is accompanied by considerable danger of severe injury. The body is then seized by a tonic convulsion, in which the head is bent back, the legs are extended, the arms convulsively extended or flexed, the jaws tightly closed, and the thorax set in the expiratory position. The face is pale, the pupils widely dilated and do not react, and the eyes are turned upward. This stage lasts only a few seconds or a minute. Then, while the face reddens and froth appears at the mouth, clonic spasms involve the head, trunk, and extremities. Gnashing of the teeth and a rattling respiration are heard. The face is covered with perspiration. The tongue is frequently caught between the teeth and bitten. Not infrequently there is incontinence of feces and urine. After a few minutes the twitching ceases and the child awakens or falls into a long state of unconsciousness. The attacks return at very irregular intervals, sometimes daily and at other times only once a year.

Abortive attacks occur more frequently in children than in adults, the so-called *epileptic vertigo* or petit mal. These are characterized by temporary disturbance of consciousness, with a fixed expression and loss of memory; at times they last but a few seconds or they are not noticed at all, then again they may continue for a longer period of time, when they are accompanied by vertigo, a feeling of fear, and also by twitchings of the face. Jacksonian epilepsy is a term applied to attacks which consist only of clonic spasms of individual muscles

or of a distinct muscle group, as the face, arm, or leg of one side. The initial cry is usually absent and consciousness is generally preserved at the beginning.

Course and Prognosis.—The course is always chronic and complete recovery is rare. The patient either remains normal mentally, and may develop to an extraordinary degree of intellectual power (Julius Cæsar, Napoleon), or in the course of time the character is altered and the child becomes peevish, irritable, and ill humored. We may also observe moral insanity, with a tendency to telling falsehoods, adventures, stealing, violent acts; or the mind may gradually decline until imbecility and complete idiocy are reached. The disease usually continues until death, and in many cases epilepsy is followed by other cerebral affections. The prognosis is the more favorable the earlier treatment is begun; it is better in the reflex type than in the genuine form, which can be attributed to no recognizable cause. If recovery occurs, the attacks gradually decrease in frequency and in intensity, become converted into petit mal, and finally cease altogether.

Diagnosis.—Hysteric seizures almost always follow emotional disturbances, generally during the day, may last for hours, and are unaccompanied by the initial cry, biting of the tongue, incontinence of feces and urine, and are not followed by the somnolent state; nor does complete loss of consciousness occur. The epileptic attack is very often independent of emotion, continues at the most for seven minutes, occurs frequently at night, and often leaves traces of blood on the pillow, due to biting of the tongue. Inquiry into the anamnesis and a careful examination is important in every case.

Treatment. — Vegetable and easily digestible diet; large quantities of milk and farinaceous foods; prohibition of alcohol, tea, and coffee; wash with warm water and follow by cool douches; tepid baths (25° to 22° R. [88.2°–81.5° F.]). Avoid mental strain. The bromid cure:

℞ Sodium bromid,
Potassium bromid, āā 1.0–2.0 gm.;
Ammonium bromid, 0.5 gm.—M.

Give this mixture daily in one dose, together with ¼ to ½ pint of water, followed later by more water. If these preparations remain ineffectual, try the Flechsig cure (the results of which are indeed uncertain); for four or five weeks administer 0.005 gm. of opium twice daily, gradually increasing the dose until from 0.01 to 0.03 gm. are given, then follow immediately by a course of the above bromid mixture for two or three months. A portion of the salt of the food may be substituted by sodium bromid. If bromism develops, stop the bromids and give large amounts of alkaline waters.

NERVOUSNESS. NEURASTHENIA. HYSTERIA

Etiology of Nervousness.—Inherited predisposition to nervous diseases of any kind, development of puberty, injuries, heat prostration, chronic intoxication from alcohol, coffee, tobacco, excessive use of meat, and following acute or chronic diseases; affections of the brain and spinal cord. Bad example set by the parents or hysteria; strong mental impressions, fright, punishment, religious impressions; masturbation; improper training; physical and mental fatigue while at school.

NEURASTHENIA

Neurasthenia represents a state of physical and mental fatigue and irritability.

Chief Symptoms. — Drowsiness and weariness after slight exertion, poor memory, inability to concentrate the thoughts, sensitiveness to loud noises and strong light; headache, which is already present upon awakening and increases during the day, but improves toward night. Nervous asthenopia (Wilbrand, Sänger); nervous dyspepsia, with constipation or diarrhea, gastralgia, hyper- or anacidity, with normal appetite; disposition

grows worse; tremor of the eyelids when the eyes are tightly closed (Rosenbach's sign).

HYSTERIA

Hysteria is a condition characterized by an abnormally excitable temperature and sensitiveness of the body, which shows a pronounced tendency to respond to any occurrence or event with decided psychic and physical disturbances of varying intensity. Peculiar to hysteria of childhood is the occurrence of individual symptoms; stigmata are met with. Frequently a former symptom of organic origin persists, that is, after the organic condition has disappeared it remains as an hysteric symptom. For example, hysteric contractures after rheumatism or the habituation of the so-called "tubards" to the laryngeal tube, even after the disappearance of the original stenosis; in these cases the respiration is perfectly free when the tube is not employed during anesthetization. Characteristic of hysteria is the fact that the severest symptoms disappear in a short time, leaving no trace, and reappear in another place.

Symptoms. — The most significant of the manifold symptoms are: Continual change of disposition from one to the other extreme and morbid introspection; absent-mindedness; autosuggestibility, as, for instance, a permanent impression of the inability to walk, to lift the arms, or to speak after a single overexertion of the muscles involved; a tendency to lie; vague pains; hyperesthesiæ; paralyses and spasms of the extremities, the voice, speech, and muscles of respiration, etc.; such palsies in contradistinction to those following organic lesions occur unsystematically, and never lead to changes in the reflexes or to electric excitability; spasms of yawning, laughter, crying, and shrieking. The affected child may jump about, dance, or throw itself upon the floor—*chorea major;* it may make grimaces—spasm of customary expressions; atasia and abasia, that is, inability to stand or walk although all movements of the legs are active when the patient is resting in bed; pronounced convulsive

seizures accompanied by more or less disturbance of consciousness for several hours or less, with partial or complete preservation of memory; catalepsy; outcries at night; somnambulism. The morning vomiting of school children is also hysteric, especially of ambitious children, and is usually brought on for the first time by emotion or worry over lessons.

Course and Prognosis of Neurasthenia and Hysteria.—Neurasthenia, which may stretch over a period of months and years, runs a variably intermittent course. The prognosis is dependent upon the severity of the individual case. The course in hysteria is likewise usually intermittent, and frequently one symptom is replaced by another. The prognosis, when external influence can be eliminated, is comparatively good, especially in the presence of single symptoms; severe and incurable cases are, however, met with.

Diagnosis.—To avoid error, before making the diagnosis of neurasthenia or hysteria a most careful and conscientious examination of the body must be made and all organic diseases excluded.

By the term mental or psychopathic inferiority (Koch) is meant a lack of resistance of the general nervous system of children who inherit a neuropathic predisposition to external influences, so that, on the one hand, these children are more likely to be affected by nervous disturbances; on the other, they do not possess sufficient strength to combat them. Patients of this class, who are often talented, suffer later from want of steadiness of character and purpose, and many commit suicide.

Prophylaxis and Treatment of Nervousness.—Prohibit or discourage marriage between neuropathic persons. Combat the predisposition by the proper physical and mental training.

Treatment.—Remove from the hysterogenic surroundings and avoid any overexertion; psychic treatment; stimulate the child's will power and, under certain circumstances, do not pay any regard to the various manifestations; easily digestible diet and hydrotherapy. If indicated, give iron, quinin, and valerian.

NIGHT TERRORS

Etiology.—Anemia, hysteria, alcoholism, adenoid vegetations, punishment, and dreams with excitable imaginations.

Symptoms.—The child awakens suddenly from a peaceful sleep, showing great fear, cries out, sits up in bed, has hallucinations, fails to recognize its surroundings, and appears to be still dreaming. After a time he again lies down quietly, falls asleep, and the next morning remembers nothing, or only vaguely, of what transpired during the night. The attacks are repeated at shorter or longer intervals.

Treatment.—A non-irritating bodily food; avoid any mental irritation. Horrible stories must not be told and the child should be raised amid peaceful surroundings. Alcohol and coffee should be prohibited. The bladder and rectum are to be emptied at the proper time. Treat the causal condition and attend to the hygiene of the bedroom and of the bed. In pronounced cases give the bromids.

MASTURBATION

Masturbation occurs in every phase of childhood, even during the nursing age.

Etiology.—Itching of the anus in eczema and oxaluria; phimosis; balanoposthitis; suggestion by example or reading. It is performed by a rocking or rubbing movement of the legs or by direct manipulation. It results locally in enlargement of the penis, reddening of the prepuce or of the labiæ, and is finally manifested by all possible forms of infantile neurasthenia.

Treatment.—Light, easily digestible vegetable diet, without alcohol, coffee, or tea. Sleep on a hard mattress. Emptying of bladder and rectum at proper intervals. Care of the body and skin. Careful prevention of all causes which give an opportunity, and institute psychic treatment.

PSYCHOSES

The causes are the same as for nervousness. In case of idiocy, old cerebral processes and defects of the brain are also to be considered. Other causes are: Premature closing of the fontanels and degeneration of the thyroid gland, either hypoplasia or aplasia.

IMBECILITY

Imbecility is due to a lack of development of the mind, and is marked by the inability to concentrate thought and to realize mental impressions. It may be congenital or acquired and occurs in all stages, from weak mindedness to fully developed idiocy.

Chief Symptoms.—The child learns to stand and walk later than normally; uncleanliness; delayed and incomplete development of speech; limited in the ability to grasp and understand all complicated directions, whereas the simple mechanics can be learned; undeveloped disposition. Inclination to lie, to steal, and to outbursts of passion and acts of violence. Slow response to mental impressions; shows a blunt confidence in strangers; active, early analgesia (Thiemich). An idiot shows a lack of intellect from the very beginning or he reaches the intellectual height of a child from one to two years old, where a standstill mentally is reached. Idiotic expression; babbling speech with a sobbing tone; uncleanliness. Apathetic as well as active and cheerful idiots are met with. Imbeciles and idiots may live to old age, but in the severer types death occurs much earlier, due to pneumonia, eclampsia, or intestinal disease.

THE AMAUROTIC IDIOCY OF FAMILIES

Amaurotic idiocy is a family disease which appears as early as the first year of life, and is accompanied by flaccidity of the muscles and disturbances of sight. Ophthalmoscopic examination reveals white specks with red centers near the macula. Death occurs generally in two years.

Treatment of Idiocy.—Institutional treatment, which may afford considerable relief in the mild cases; courses in conversation; schools for the weak minded. In case of myxedema and idiocy administer thyroid tablets.

MORAL INSANITY

Degenerate tendencies, with defect of the intellect. It begins as "naughtiness" and "ill temper." The child is inclined to lie and is guilty of cruelty, cunning, and craftiness. He has a tendency to commit crimes, such as stealing and arson, and to do bodily harm. Every form of training is powerless.

JUVENILE INSANITY

(*Hebephrenia*)

This represents a state of weakened intellect which develops during puberty and runs a progressive course. It begins with excitement, depression, and hallucinations, and as it progresses it enters a katatonic state (negativism, stereotypy, automatism, etc.) and passes gradually, with marked mental deterioration, into weak mindedness.

PRIMARY PROGRESSIVE MYOPATHY

This is a chronic hereditary and family disease of certain muscle groups, occurring in childhood or at puberty, which is not of central, but of myogenic origin. It is accompanied by atrophy and simultaneous hypertrophy of the muscles, with intact sensibility, but without reactions of degeneration and with loss of knee-jerks.

Morbid Anatomy.—The nervous system is normal. Pale, soft, or also hard muscles; histologically there is proliferation of the connective tissue, compression of the muscle-fibers, and eventually degenerative changes and the deposition of fat in the latter.

Symptoms.—*Pseudohypertrophic Paralysis* (Duchenne).—It begins between the fifth and eighth year, generally in boys, with uncertainty in gait and in jumping; wab-

bling gait with protruded abdomen and lordotic spinal column. The child shows characteristic movements when it raises itself from the floor; the body is supported with the hands, first on the floor, then on the knees and legs, and thus climbs slowly up on its own body. The disease begins always in the muscles of the trunk and the lower extremities. The muscles, especially the gluteal and those of the calves of the legs, are thickened and shapeless. The affection progresses slowly and also involves the upper extremities, and, gradually undergoing conversion into true atrophy, renders the patient helpless. Death in the course of years from intercurrent diseases.

Erb's Form of Juvenile Muscular Atrophy.—Gradually developing weakness and emaciation of certain muscle groups of the shoulders and arms without pseudohypertrophy. The muscles uniformly involved are the pectorals, trapezius, latissimus dorsi, serratus anticus, and the rhomboidei (the shoulder-girdle type), to which are added the gluteal muscles, the quadriceps, and peroneal muscles (pelvic-girdle type). Both forms may coexist; very chronic course. Death is due to intercurrent affections.

Infantile form, with involvement of the facial musculature (Duchenne, Landouzy, Déjerine). This form begins in the muscles of the face; the eyelids are closed; whistling, laughing, and speaking become difficult or impossible. Collapse of the cheeks and hanging down of the lower lip interfere with mimicking and form the typic stupid "myopathic" facial expression.

Diagnosis.—In differentiating the myopathic from the spinal muscular atrophy, note in the former the juvenile and family character, the typic localization and the non-involvement of the sternomastoid, the deltoid muscles, and, above all, the small muscles of the hand. Muscular twitchings and the reactions of degeneration are absent.

Treatment.—Avoid overexertion; massage and galvanism; gymnastics and food rich in proteids.

ACUTE INFECTIOUS DISEASES

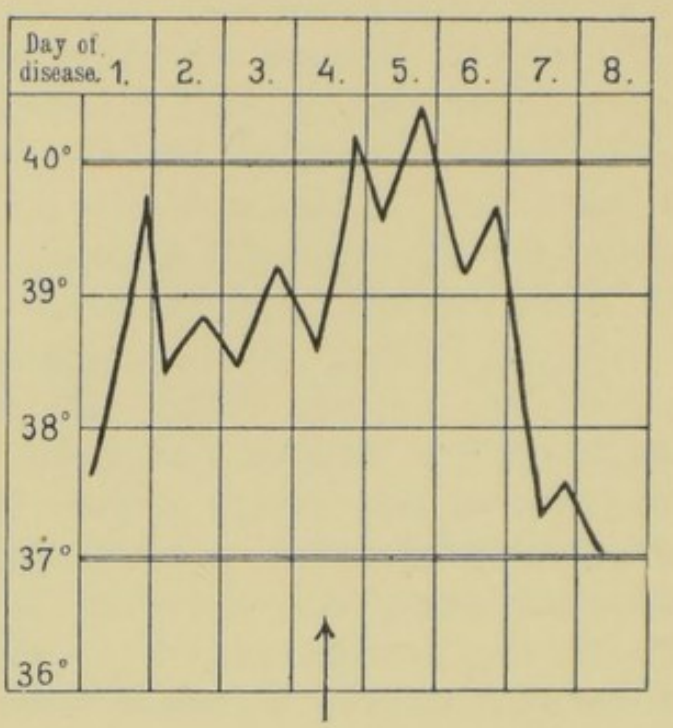
GENERAL DISCUSSION

THE acute infectious diseases which are accompanied by fever are caused by special micro-organisms which are transmitted directly from one diseased person or indirectly through a third person by means of infected articles of use or provisions. The portals of entrance for the bacteria, of which we are as yet only acquainted with a small number, are the mucous membrane of the respiratory and digestive tracts and (rarely) the skin. If the bacteria find the condition at the point of entrance favorable for growth, and if for any reason the individual's general resistance is weakened, the invading virus may call forth disease symptoms, provided no congenital or acquired specific immunity exists. These symptoms depend upon the character of the specific germ, and are caused either by the bacterial body itself, which eventually enters the blood-stream (infection in a strict sense), or through their poisonous metabolic products with which the body is supplied from the point of invasion (intoxication). The disease symptoms are not noticed so long as the micro-organisms are engaged in combat with the varying number of natural immune bodies of the organism, and so long as a sufficient multiplication of bacteria or a sufficient accumulation of the specific disease poison does not occur.

A certain period usually elapses from the time of infection until the disease makes its appearance, which is called the period of incubation. The duration of the incubation and the disease symptoms vary according to the character of the specific germ, each of which creates a symptom-complex peculiar to itself; thus, the diphtheria

bacillus causes only diphtheria and not scarlet fever or measles. If a second disease develops simultaneously with or after another one, it may be assumed that the causes of both diseases invaded the body at the same time or soon thereafter. This possibility is not so very rare, for between certain infectious diseases a closer relationship exists than that of the preparation of the soil by one disease for the other. Such a relationship exists between measles on the one hand and influenza and whooping-cough on the other. The course in each individual form of acute infection is typic. In the acute exanthems we distinguish between an eruptive, a florid, and a desquamative stage, each of which (provided complications are absent) presents a fairly definite form of development and duration. In certain diseases the eruption of the exanthem is directly preceded by more or less characteristic manifestations—the *prodromal symptoms.* The course of the disease is dependent upon the virulence of the micro-organism, the strength and the susceptibility of the patient to poisons, and, furthermore, upon the development of complications, which may be traced back to the secondary invasion of non-specific bacteria into the already weakened body. Many of the infectious diseases are complicated by nephritis, nervous and psychic disturbances, and frequently by pronounced anemia.

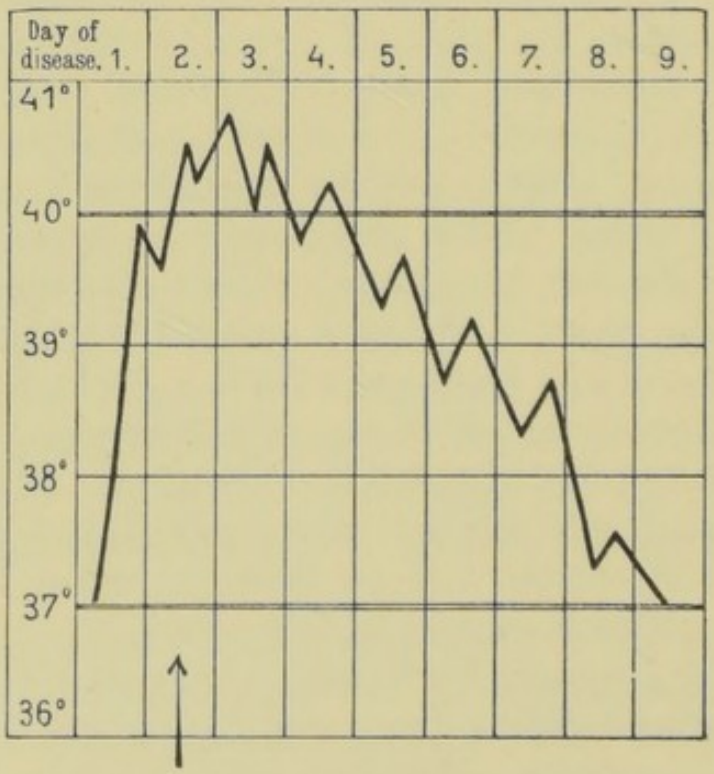
If death does not occur, the disease is overcome by the action of specific immune bodies which, during the course of the disease, have developed within the body (every specific poison in the body calls forth a protective measure to destroy it), and which creates a permanent or, at least, temporary immunity against that disease. Much may be attained prophylactically by early isolation of the patient and personal attention, also by disinfection of the articles used—the secretions and excretions and, later, of the sick room; also by keeping brothers and sisters of the patient, as well as convalescents, from visiting school (the period of isolation in measles, rötheln, varicella, and mumps is three weeks; in diphtheria and typhoid fever, five weeks; in scarlet fever, six weeks; in pertussis, eight weeks; in



Exanthem.

Initial fever. Eruptive fever.

FIG. 87.—The type of fever in measles (von Strümpell).



Exanthem.

FIG. 88.—The type of fever in scarlet fever (von Strümpell).

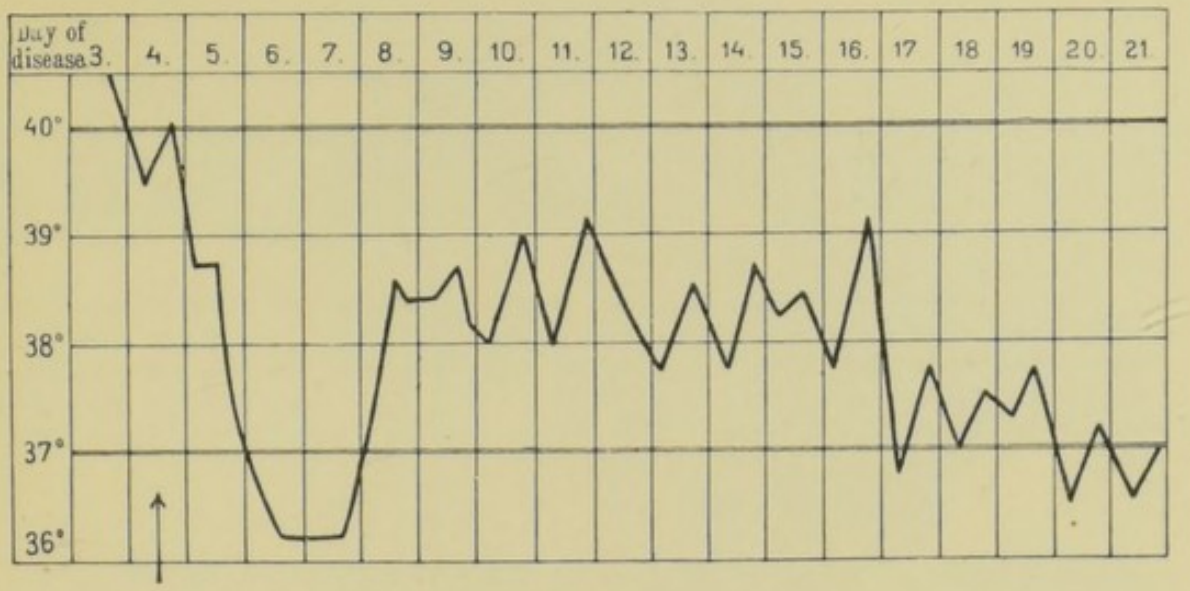


FIG. 89.—The type of fever in small-pox (Leo).

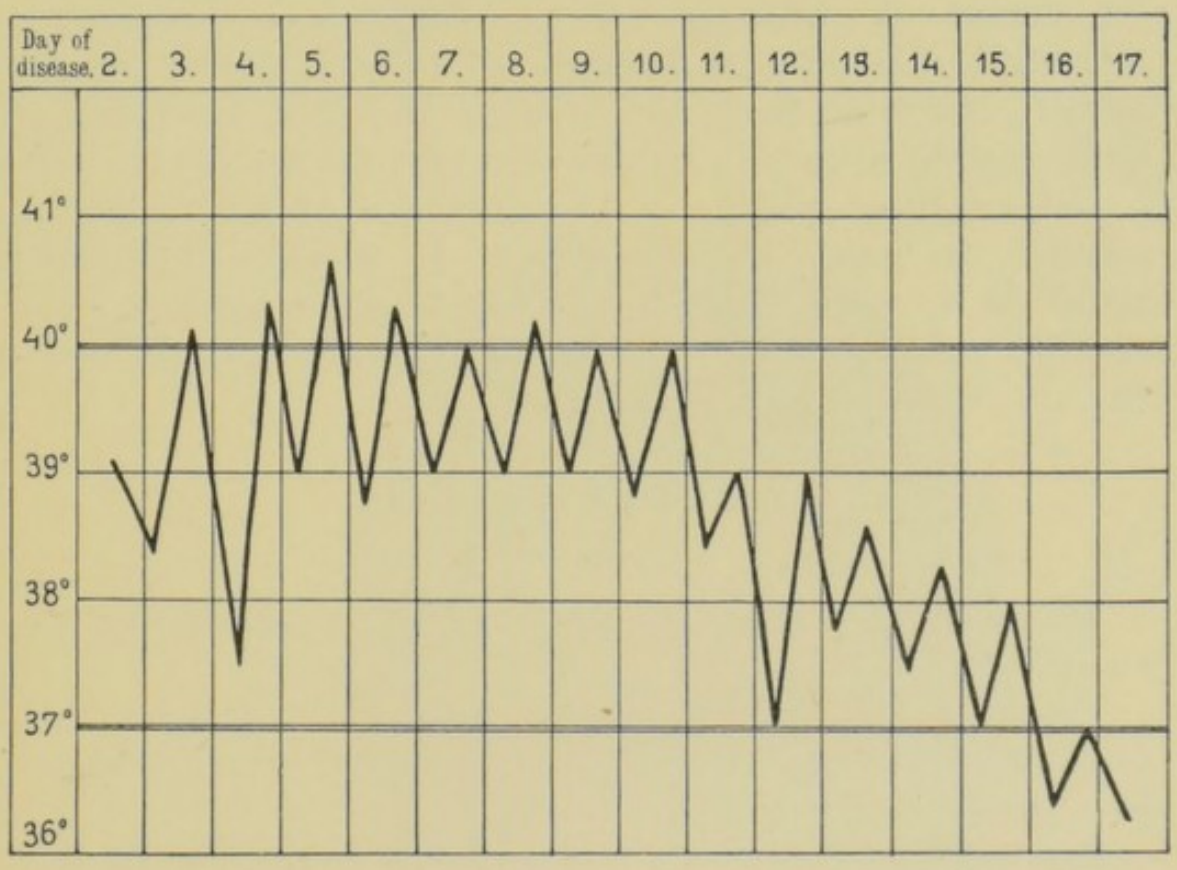


FIG. 90.—The type of fever in the typhoid fever of childhood (Gerhardt-Seiffert).

PLATE 21

Early Symptoms of Measles

FIG. 1. **Koplik Spots.**—These are seen two days before the eruption of the exanthem. The buccal mucous membrane shows reddish specks in the region of the molars, in which area are also seen somewhat elevated injected spots of varying size (fraction of a millimeter) and of a rounded or oval form.

FIG. 2. **The Eruption of Measles on the Mucous Membrane One Day Before the Skin Eruption.**—Irregularly formed, small and large pale red spots with serrated edges on the mucous membrane of the soft palate, which is still pale. The edges of the velum palati, the uvula, and the tonsils are reddened. The tongue is covered with a thick grayish-white fur.

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other diseases the child should be kept home according to the discretion of the doctor). Especial care must be observed to guard young or weak children from infection. The best protection is a hygienic life; in case of smallpox, vaccination; in diphtheria, preventive inoculation by means of antitoxic serum. The most important measures in infectious diseases are of a hygienic-dietetic nature: Provide fresh air, preserve the body heat (rest in bed), careful attention to the skin and the mouth; a non-irritating diet, which, in the presence of fever, should contain no meat. With respect to medicaments, these are of less value than hydrotherapeutic measures. Of specific remedies we possess the diphtheritic antitoxin and the antistreptococcic serum.

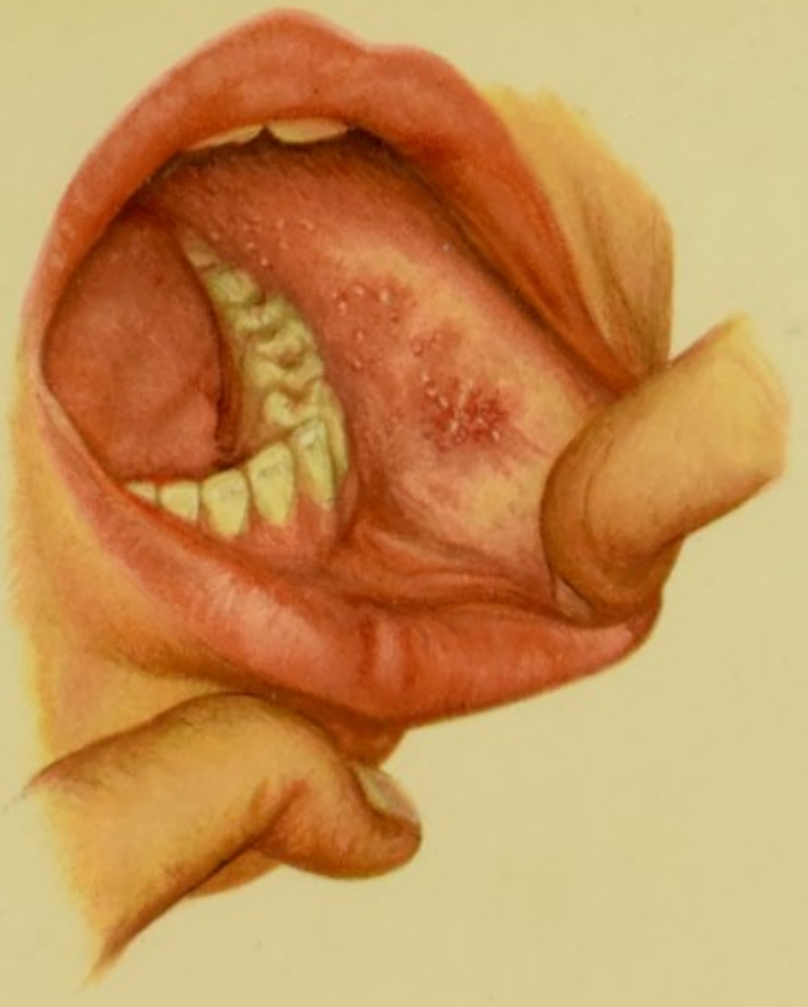
MEASLES

(*Morbilli*)

Measles is an acute febrile disease accompanied by a maculopapular rash and catarrhal phenomena.

The **incubation period** lasts eleven days and runs a symptomless course, or with the appearance of manifestations of a general character.

Symptoms.—Following the period of incubation the disease begins with catarrhal symptoms (*catarrhal stage*), a remittent or intermittent fever, catarrh of the conjunctivæ and of the upper air-passages (coryza, short, dry cough); and mucous-membrane changes of an exanthe-





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matic character, consisting of groups of bluish-white miliary injected specks (Koplik) on the reddened mucous membrane. Directly before the eruption of the rash pointed and star-shaped reddening of the palatine mucous membrane and of the conjunctiva is observed (also of the mucous membrane of the larynx and trachea)—the so-called *enanthem.*

The *eruptive stage* sets in on the third day of the disease (fourteen days after infection) with a high fever

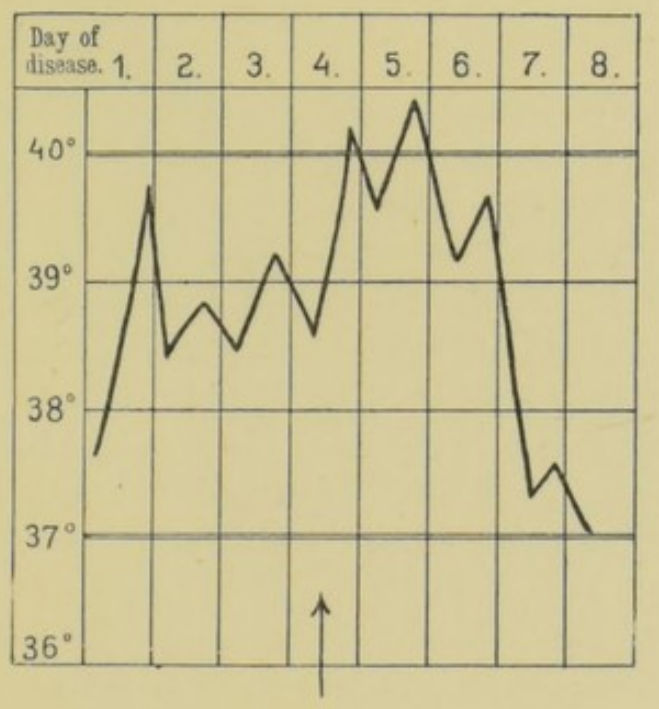


FIG. 91.—The type of fever in measles (von Strümpell).

(40° C. [104° F.] and over) and an increase in intensity of the catarrhal symptoms, especially of the laryngitis and the general constitutional symptoms; in small children it is often accompanied by convulsions. The eruption begins back of the ears, spreads to the face, and is accompanied by an increase in the fever; it extends in from one and a half to two days to the neck, trunk, and extremities. The eruption next forms little red points, which rapidly enlarge and develop into irregularly formed spots with notched edges and of a fairly definite contour;

PLATE 22

The Eruption of Measles Two Days After its First Appearance.—The skin of the whole body, with the exception of the scalp, is covered with bluish-red minute specks. The efflorescent areas have united and formed large irregularly notched figures, which in the face and in isolated patches on the body have been elevated into papules. On close inspection we note—especially on the large exanthematous areas—single elevated and reddened follicles. The skin feels hot, uneven, and greasy, and is, on the whole, somewhat swollen, especially on the face. The eyes and nose are swollen, the lids reddened and glued together, and the nares and upper lip are excoriated by the abundant purulent secretion. Temperature, 40.1° C. [104.2° F.]; troublesome cough; hoarseness. In this case the disease ran a course of eight days without complications. (Clinic of von Ranke, Munich.)

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these become confluent and form larger areas, between which, however, normal skin is seen here and there. On the face the eruption soon assumes the papular character, whereas on the body it remains, as a rule, flat; in many areas a nodular swelling of the hair-follicles and of the excretory ducts of the sebaceous glands may be felt. The color of the eruption is at first pale red, in anemic children it is paler or a dirty red, but it soon becomes darker and turns bluish red, after which it becomes brownish red and fading, passing gradually into a yellow color.

The whole skin, chiefly of the face, is made tense by congestion of the blood-vessels, and the eyelids and the nose are particularly swollen; the cervical lymph-nodes are enlarged. The urine is concentrated, gives a positive diazo-reaction, and in some cases contains albumin. About three days after the appearance of the eruption the fever disappears by crisis and the child perspires freely. The eruption begins to fade in the same order as in its development and the other phenomena recede, with the exception of the laryngitis (or bronchitis). After the sixth day a bran-like desquamation takes place, which is barely visible. From then on recovery sets in, and the manifestations of irritation of the respiratory mucous membranes gradually disappear. Uncomplicated cases last from eight to ten days after the first day of eruption. Variations in the exanthem assume the form of vesicular or hemorrhagic confluent morbilli (benign, malignant).



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Complications.—At times scarlet fever, varicella, and diphtheria develop simultaneously with measles. Measles is frequently accompanied by severe diseases of the air-passages (measly croup, capillary bronchitis, broncho-pneumonia), and is nearly always followed by inflammation of the middle ear (Nadoleczny), which may be attributed primarily or secondarily to the action of the poison of measles. In the form of measles which attacks children of poor health the involvement of the skin is of less importance than the process affecting the mucous membrane. If the virus is particularly active in the mucous membrane of the bronchial tree we may meet with necrotic destruction of the inflamed pulmonary tissue. In many cases tuberculosis sets in at the climax of the disease.

Therefore the prognosis, which may be perfectly favorable as regards the disease itself, is decidedly unfavorable when complicated by serious pulmonary disease. It is bad from the very beginning in malignant hemorrhagic morbilli, which is marked by severe cerebral symptoms, the early development of skin and intestinal hemorrhages, the presence of gangrenous processes in the mucous membrane (*Noma facialis et vulvæ*), and its rapid termination in collapse and death.

The diagnosis is usually made without difficulty from the characteristic symptoms. The differential diagnosis includes chiefly rubella, scarlet fever, infectious erythema, and small-pox during the stage of invasion. In rubella Koplik's sign and the diazo-reaction are absent, and the accompanying symptoms are of a mild grade. The efflorescence of the skin arises in groups, which show no tendency to become confluent; secondary swelling of the skin does not develop. In scarlet fever the eruption begins in the throat (in measles, in the face) and spreads much more rapidly and uniformly, and not by stages, as in measles; the region of the mouth and nose remains uninvolved. The exanthem consists of minute points which are not crowded together. Initial vomiting is almost constant in scarlet fever (in measles, exceptionally);

PLATE 23

The Eruption of Rubella One-half Day After its Appearance.—Innumerable round, flat, bluish-red papules are noted on the face, which form into irregular groups or into crescentic figures; these do not coalesce, but arise in numerous places on a common erythematous area. Mild conjunctivitis; the swollen nose is somewhat obstructed. No Koplik spots, yet a very fine exanthem was observed on the soft palate. Isolated rose-red spots on the skin of the trunk, the upper arms, and the thighs. The general health was undisturbed. The exanthem disappeared on the evening of the third day. The patient, Emily Gr., seven and a half years of age, developed mumps, March 9, 1904 (see Fig. 104); returned to school, March 16, 1904; developed rubella, April 4, 1904; returned to school, April 11, 1904; developed whooping-cough, April 16, 1904; returned to school, June 20, 1904; developed measles, July 1, 1904. The whooping-cough, which at the time of the eruption of measles had reached the final catarrhal stage, underwent a relapse, with fresh paroxysms of cough. Duration three weeks. Mild cervical adenopathy persisted.

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angina always present (in measles, catarrh) and the fever disappears by lysis (in measles, by crisis).

Erythema infectiosum (Sticker, A. Schmid) is not infrequently mistaken for measles, rubella, and scarlet fever. This is an infectious epidemic polymorphous eruption, the course of which is unaccompanied by constitutional symptoms and which resembles erythema exudativum multiforme in form, color, and development. It is distinguished from this condition, however, by beginning in the face, whereas the extremities are not attacked until later and the trunk remains free. Erythema infrequently may be mistaken for measles by the simultaneous occurrence of catarrh; for scarlet fever, by the extensive confluence of the dark red erythematous spots. It is distinguished from both by the absence of all associated symptoms and the changes in the mucous membranes, the long duration (about eight days), and the characteristic symptoms of involution of the eruption (see Erythema Multiforme). The latter peculiarity also serves, together with the confluence of the erythematous spots, to differentiate from rubella, with which erythema infrequently shares in common the absence of constitutional symptoms, and, as a rule, febrile and uncomplicated course and absence of resulting disease processes.

Treatment.—(General management as discussed in the



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Introduction.) When the eruption is delayed or partially developed resort to wet or dry pack. For itching, anoint with grease. After the eruption is completed, daily baths (35° C. [95° F.]) and soaping of the skin. For the conjunctivitis, boric acid compresses or, if necessary, unguentum hydrargyri oxidi flavus, 0.1 to 10.0 gm. In troublesome laryngitis, infusion of ipecacuanha, 0.3 gm. : 150.0, with aquæ laurocerasus, 1.5 cc.; or extract of belladonna, 0.1 gm. to 10.0 cc.; aquæ laurocerasus also with codein phosphate, 0.2 gm., of which mixture give 10 drops three times a day. (For treatment of pulmonary complications, see Diseases of the Lungs.) In beginning otitis media, warm applications and instillations of lukewarm thymol-glycerin, 0.1 : 50.0 (Nadoleczny).

RUBELLA

Rubella is an acute maculopapular exanthematous disease, accompanied by an ephemeral fever and a mild catarrh of the nose. The period of incubation is not as definite as in measles, and lasts up to three weeks.

Symptoms.—The early symptoms (*Koplik's spots*) are absent. The exanthem, which appears in the same order and to the same extent as in measles, is usually milder in intensity and consists of rounded specks about the size of a lentil; also smaller, and sometimes, though rarely so, larger, which occur singly on the trunk and the extremities; whereas on the face and the neck they develop in groups, forming semicircles, rapidly causing nodular infiltration. The color, which is originally dark red, passes after a few hours into a bluish-red, and after the second or third day into a light brownish-yellow. The skin of the face is not swollen as in measles, but, on the contrary, the papules appear more elevated than in measles and give the face an uneven appearance. The papules do not become confluent as in measles, yet several papules may be united by a simultaneous erythematous reddening of the skin. The posterior pharyngeal wall appears diffusely hyperemic, and at times a fine enanthem

PLATE 24

The Exanthem of Scarlet Fever (Third Day).—Universal extension of the fine punctiform scarlet-red eruption, which is thickest on the neck, axilla, on the back, and inner side of the thighs, while on the chest and upper arms it assumes a more spotted appearance because of irregular distribution. The parts around the mouth and nose which are not involved appear markedly pale in comparison with the red, flushed cheeks. The lips are dry and dark red. Skin appears to be edematous and feels burning hot and like short-trimmed plush (due to follicular swelling). Angina; swelling of cervical lymph-nodes. Temperature, 40° C. [104° F.]. Smooth course without complications. (Clinic of von Ranke, Munich.)

of the palatal mucosa is noted, also reddened striæ of the buccal mucous membrane and of the lips (A. Schmid). The skin, as a rule, does not desquamate. The associated phenomena are, as has been stated, very slight. The occipital and submaxillary nodes are not infrequently painfully swollen.

SCARLET FEVER

(*Scarlatina*)

Scarlet fever is an acute febrile general disease, characterized by a scarlet-red, fine, punctiform exanthem and a true inflammatory or necrotic angina. The stage of incubation is symptomless and lasts from two to seven days. The disease begins suddenly with vomiting, which is soon followed by chills (in older children), a high fever, together with a disproportionately rapid pulse, disturbances of swallowing, headache, and at times convulsions and delirium. Inspection of the pharynx discloses the tongue to be heavily coated with a thick white fur, whose exposed areas present the prominent red filiform papillæ (the point of the tongue resembles later a strawberry); the mucosa of the soft palate is covered with single dark red spots; the uvula and tonsils are the site of a yellowish mucoid deposit; the pharyngeal lymphatics are all swollen, as are also the cervical nodes. The exanthem appears at the end of the first and, at the latest, at the second day of the disease, and spreads within twelve to twenty-four hours over the neck, the chest, elbows, portions of the face, and finally over the skin of the entire body. It consists of countless extremely minute spots,



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which are so close together in some areas that the skin appears to be of a uniform fiery-red color. The single spots, which usually represent swollen hair-follicles, may be seen, on close inspection, to be separated by areas of pale skin, but later become united by increasing hyperemia of the skin and sometimes by a scarlet-red erythema. The slightly edematous skin feels burning hot, "shotty," and rough where the hair-follicles are swollen. It is noteworthy that the face is rarely attacked, but that in either

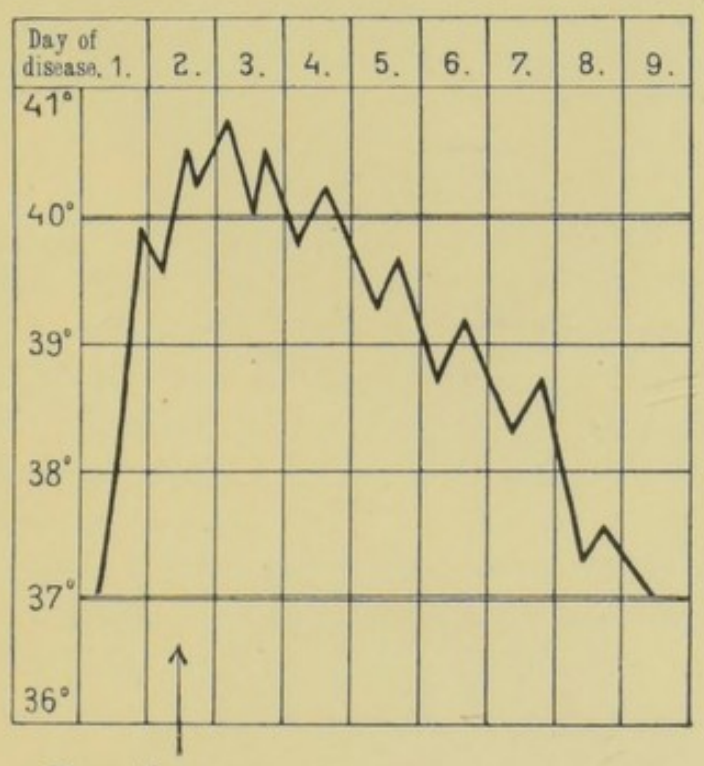


FIG. 92.—The type of fever in scarlatina (von Strümpell).

case the parts around the mouth and nose remain pale; the palms and the soles also generally escape the rash. With the increase of the fever from 39° C. [102.2° F.] on the first day to 40° C. [104° F.] on the second day, the red color of the exanthem also increases in intensity, especially on the abdomen, the inner side of the thighs, the gluteal regions, and on the back.

When the fever continues high, with only slight morning remissions, the eruption flourishes for from four to seven days. During this time the patients are very restless (many are stuporous), possess no appetite, and

PLATE 25

FIG. 1. **Scarlatinal Angina (Third Day).**—Livid discoloration of the oral and pharyngeal mucosa; the uvula and tonsils are dark red; minute hemorrhages on the soft palate and uvula; left tonsil covered with a whitish-yellow, pultaceous, and shiny lacunar deposit; whole right tonsil covered with purulent mucus; dorsum of tongue still coated with thick grayish-white fur. The clean tip of the tongue with its dark red filiform papillæ resembles a strawberry. (Clinic of von Ranke, Munich.)

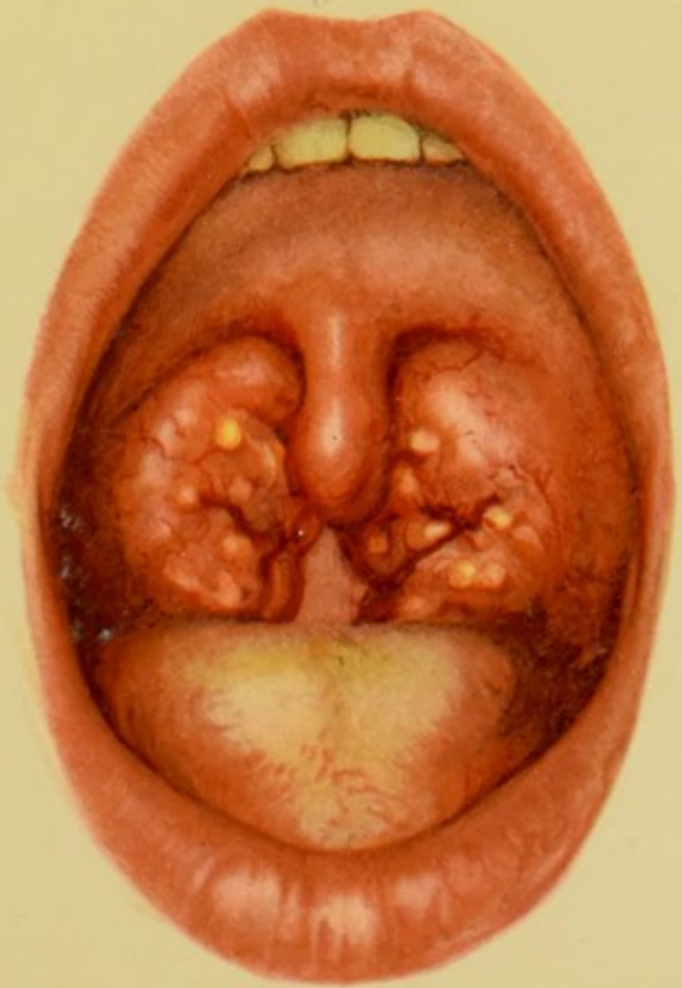
FIG. 2. **Lacunar Angina.**—Circumscribed reddening of the isthmus of the fauces; tonsils and uvula markedly edematous; the latter is decidedly elongated. Uvula and tonsils are covered with a tenacious, shiny mucus. The markedly congested tonsils show yellow coalescing lacunar deposits. The tongue is dry and coated gray. Cervical lymph-nodes markedly swollen. Temperature, 39.5° C. [102.4° F.]. Duration of disease, six days. On the fourth day several of the lacunar deposits were easily removed with a spoon and found to be smooth and caseous, similar to collections of smegma.

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complain of great thirst and pain in the neck. The activity of the heart is increased. Scanty febrile urine which contains a large amount of albumin. The bowels, after a diarrhea at the beginning, are constipated. The spleen is frequently somewhat enlarged. The fever begins to fall in from five to seven days by lysis and the eruption begins to fade. The stage of desquamation begins a few days later. The superficial layer of skin of the head, forehead, neck, and back is shed in bran-like scales, and in other areas, especially on the abdomen, the hands and the feet, in large sections or lamellæ. In some cases the skin of the whole body undergoes this bran-like desquamation. At about the eighth to the fourteenth day of the desquamative period the temperature falls to normal and remains there, provided no complications arise. Simultaneously with the disappearance of the fever the remaining symptoms disappear. The duration of the disease in favorable cases is from three to four weeks. Particular forms of this exanthem are miliary, vesicular, variegated (appearance of isolated spots of varying size), papular, and hemorrhagic scarlatina. The pharyngeal lesion may also vary in different cases—it may consist of scarlatina with angina or it may be scarlatinodiphtheroid. In the latter form the initial simple angina may be converted into a diphtheritic affection



Fig. 1.



with a strong tendency to tissue necrosis. It may run a violent or a more prolonged course and greatly endanger the patient's life. In differentiating from diphtheria note: The pultaceous (smeary) character of the deposit; the marked swelling of the lymph-nodes and tissue necrosis, which is rarely so severe in diphtheria; the slight tendency to spread to the larynx and trachea and the absence of paralysis.

Aside from the above types of this disease, we also meet with a number of other severe forms; in scarlatina gravissima the virus has a rapid paralyzing action upon the brain and heart; the typhoidal form of scarlet fever is characterized by a grave infection accompanied by typhoidal symptoms; variable hemorrhagic and septic types, which are also observed, run a course whose character is dependent upon that of the secondary septic infection.

Complications and Sequelæ.—The most frequent complications and resulting conditions following scarlet fever include otitis, nephritis, inflammatory diseases of the lungs, pleura, endocardium, and joints. The scarlatinal nephritis begins usually at the commencement of the third week. The scanty amount of urine excreted contains albumin, casts, and blood. The general health is decidedly disturbed. Partial edema, also anasarca; hypertrophy and dilatation of the left ventricle result. The affection lasts from three to four weeks (usually glomerulonephritis). In unfavorable cases the course is prolonged and passes into chronic nephritis, or it progresses rapidly with all the symptoms of a severe intoxication—uremia.

The **prognosis** of scarlet fever, because of the constant danger of serious complications, before the end of three or four weeks should be doubtful. The average mortality rate is about 12 per cent.

The **diagnosis**, when the cardinal symptoms exist, is easy.

Treatment and Prophylaxis (see Introduction).—Rest in bed for from three to six weeks; fever diet without meat for three weeks. To relieve the heart, which is threatened

by the scarlatinal virus, give daily hot baths (40° C. [104° F.]); anoint the body with soap. When nervous symptoms are prominent resort to neutral soap-baths (35° C. [95° F.]), with cold rubbing while in bath. When pronounced weakness exists, substitute the baths for cold washing or the wet pack.

Special Therapeutic Measures.—In case of delayed eruption of the exanthem, resort to packing. To lessen itching of the skin rub with thymol, 0.5 gm., carbolic acid, 2.0 gm., and vaselin, 50.0 gm., after bathing. In scarlatinal diphtheria: Priessnitz's compresses, gargle with carbolic acid solution (1 teaspoonful of 5 per cent. carbolic acid solution to $\frac{1}{2}$ pint of water); cautious swabbing of the tonsillar deposit with 5 per cent. carbolic acid solution or tincture of ferric chlorid. Heubner recommends injecting a hypodermic syringeful of 3 per cent. carbolic acid solution into the tonsils and palate twice daily (for injection it is necessary to attach a Taube cannula to a hypodermic syringe), spraying the oral cavity every two hours with katharol (3 per cent. solution of hydrogen peroxid), and bathing the nose with salt water or boric acid solution. To lessen the swelling of the lymph-nodes rub them with 10 per cent. iodovasogen or ichthyol-vasogen; in threatening abscess formation apply cataplasms. For scarlatinal otitis, ice suppositories or an ice-bag; inject 1 to 2 drops of 10 per cent. carbolic acid and glycerin; when perforation is delayed resort to paracentesis, followed every one or two hours by injections of katharol. Treat scarlatinal rheumatism by immobilizing the joint with cotton dressings (cardboard splints) and administer salicylates. In the nephritis of scarlet fever absolute rest in bed and milk-diet; only in case of distaste for the latter, or long duration of the nephritis and the development of weakness, is it permissible to cautiously add vegetable food; sour lemonade, a wineglassful two or three times daily. To stimulate diaphoresis, hot baths followed by dry packs; diuretin; caffein. Control hematuria by adrenalin or gelatin, internally or subcutaneously. Prolonged and marked albuminuria

indicates a coffeespoonful of infusion of digitalis (0.5 gm. : 100.0) every two hours. If uremia is threatened resort to warm baths and dry pack ; venesection ; enemata of chloral ; ice-bag to the head ; stimulants. The Moser antistreptococcic serum is recommended in severe cases as specific treatment (not yet sold on the market).

SMALL-POX. VARIOLA

Small-pox is a febrile contagious disease accompanied by a pustular eruption and a course which is divided into several stages.

The disease begins after an **incubation period** of nine days with a high continuous fever, severe nervous and dyspeptic manifestations, pains in the back, weakness, and occasionally a scarlatinal or a measles-like rash (initial exanthem).

The **eruption** appears on the third or fourth day of the disease, at first on the throat and face, later, on the whole body (it being thickest on the face and hands), as well as in isolated patches upon the mucosa of the digestive, respiratory, and genito-urinary tracts, and not rarely on the conjunctivæ. The virus of small-pox causes extensive vascular changes in circumscribed areas of the skin or mucous membranes, which lead to hyperemia and edematous swelling, and later a marked inflammatory exudation and infiltration of the involved parts. At first roseola-like spots are seen to develop, which are rapidly converted into flat papules, and within two or three days after partial liquefaction into variously formed vesicles ; this is the *stage of eruption*.

Following the inflammatory exudation, further conversion occurs (from the end of the first week on) of the approximately lentil-sized umbilicated vesicles—which are of mother-of-pearl color—into tense pustules on an infiltrated base, which are surrounded by a red border, filled with a seropurulent fluid, and occasionally tend to coalesce ; this is the *stage of suppuration*.

Toward the end of the second week the pustules dry

up upon the formation of new umbilications and are gradually altered into crusts, which generally (from the thirtieth to the thirty-sixth day—Fischl) loosen and fall off, leaving behind a red scar; this is the *stage of desiccation.*

The fever of invasion takes a sudden drop after the development of the vesicles. With the beginning formation of the pustules the temperature again rises—*suppuration fever*—which continues about one week, with evening exacerbations, and is followed by a decline by lysis.

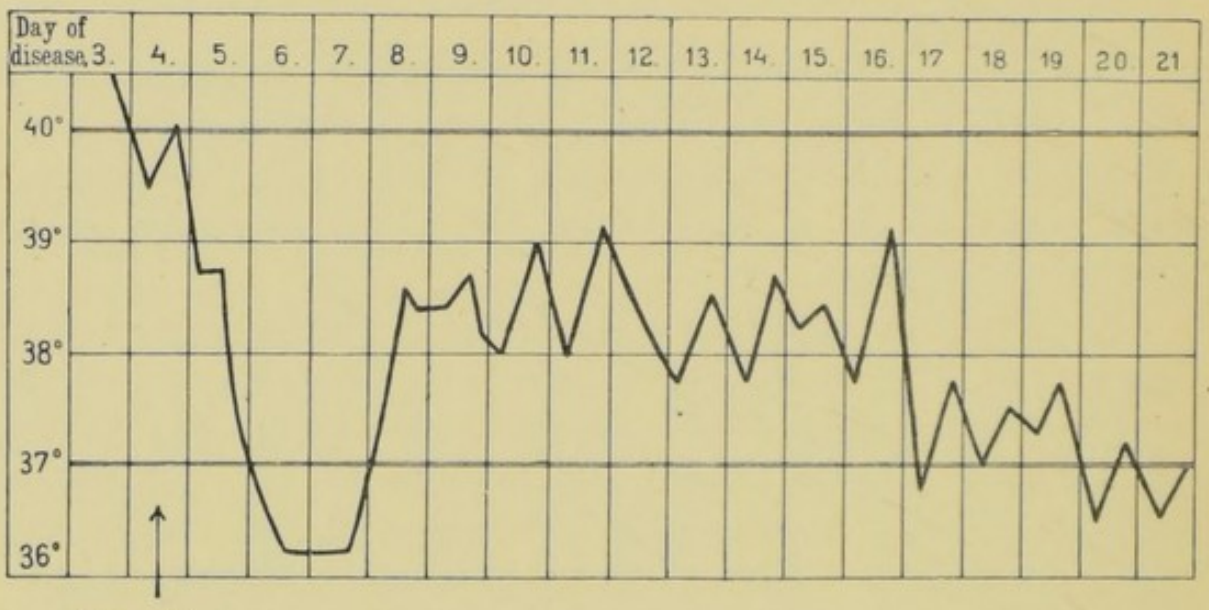


FIG. 95.—The type of fever in small-pox (Leo).

During the first days of the stage of desiccation we occasionally note an ephemeral high fever, the so-called *desiccation fever.* The general health suffers greatly from the influence of the fever, the loss of sleep on account of the itching, and the manifold disturbances associated with the affections of the mucous membrane. The urine contains albumin and blood. The conjunctival eruption endangers the eyesight, and the development of it in the pharynx leads to pseudomembranous and phlegmonous processes.

The **symptom-complex** is nearly always severe and in nursing infants death frequently occurs, even at the time

of the appearance of the suppuration fever; in older children, from secondary septic infections which originate in ruptured or scratched pustules.

Aside from the above-described moderate form of small-pox, we also meet with other severer and even milder types. To the former belongs the so-called *black small-pox* (Purpura variolosa), which is accompanied by pronounced cerebral symptoms, marked cardiac depression, hemorrhages into the skin and mucous membranes, as well as from the mouth, nose, ear, stomach, intestines, and kidneys. Death sets in before the true eruption of small-pox has had time to appear.

Another severe form is the *variola hæmorrhagica pustulosa*, in which the hemorrhagic diathesis does not appear until the stage of development.

Still another variety is the *confluent variola*, in which collapse and death occur as early as the ninth to eleventh day from extensive suppuration and marked general infection. The milder forms of small-pox include variola sine exanthemata, variola apyretica, variola abortiva, and varioloid. In the latter variety the symptoms of the invasion appear, but the suppuration fever fails to develop (attacks chiefly vaccinated individuals in whom the immunity due to a former vaccination has worn off in the course of years).

The **diagnosis** before the appearance of the vesicles may cause much difficulty. The prodromal erythema and the beginning skin eruption are distinguished from scarlet fever and measles by the absence of the enanthem, the typic scarlatinal angina, and the Koplik spots (the latter is also only present in measles in 80 per cent. of all cases). As the disease progresses the serious nervous symptoms may also be mistaken for meningitis.

The **prognosis** is dependent upon the character of the epidemic, the age of the patient (most fatal in nursing children), and vaccination.

Prophylaxis and Treatment.—All persons who have come in contact with a patient suffering from small-pox should be immediately vaccinated or revaccinated. Infected

PLATE 26

Normally Developed Vaccine Pustules on the Eighth Day after Vaccination.

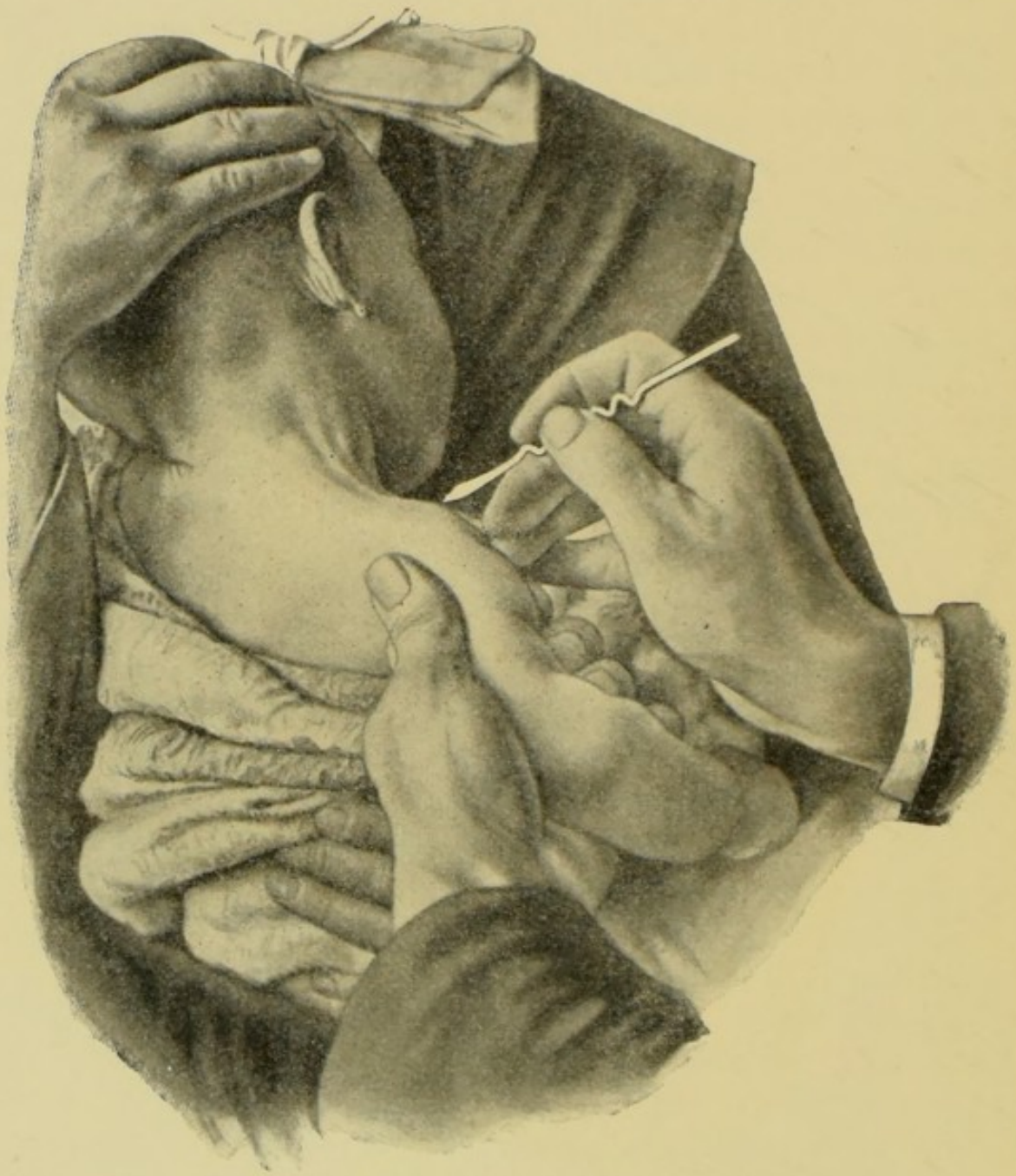
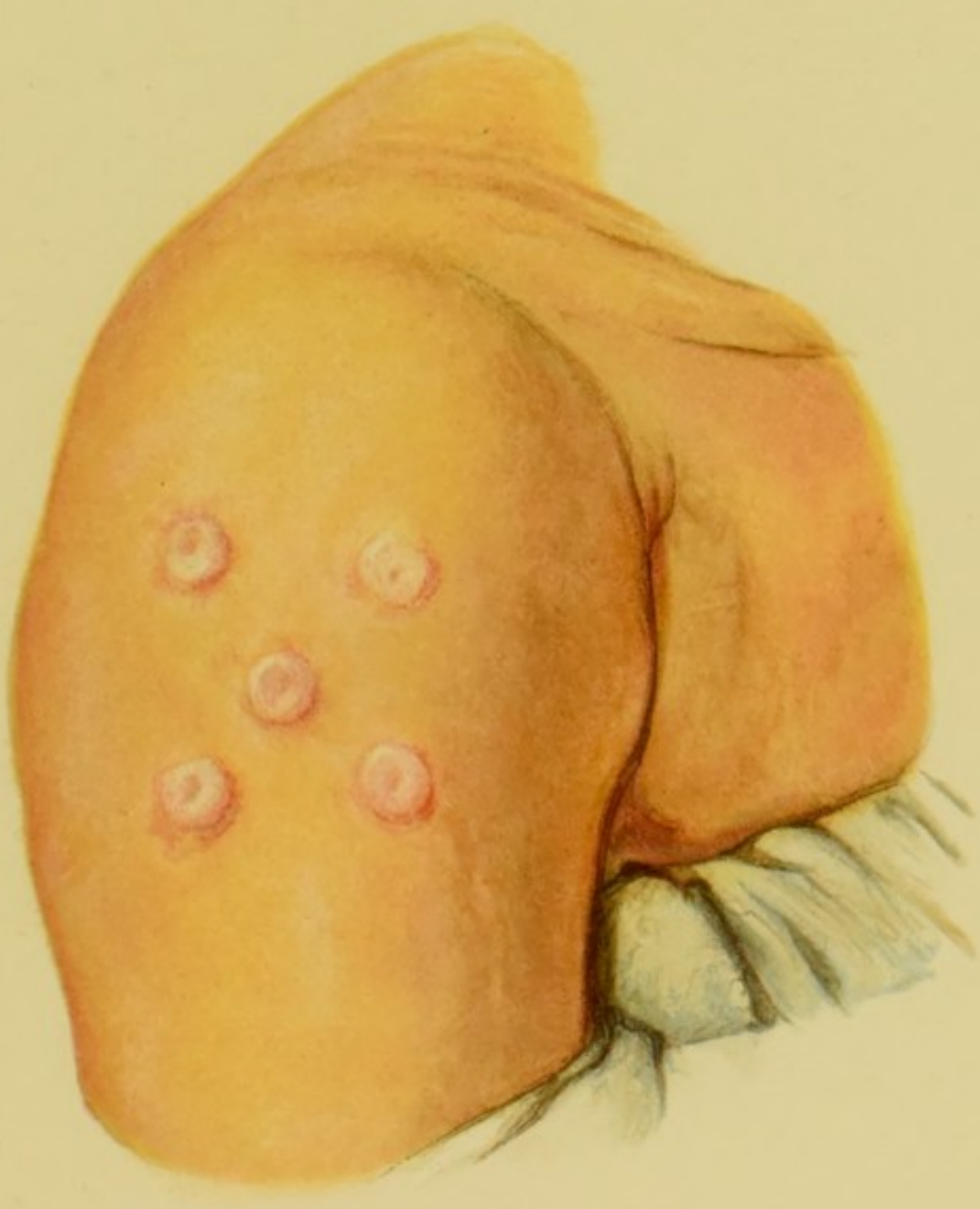


FIG. 96.—The first vaccination.

articles should be burned. Strict isolation of the patient and careful nursing. The therapy is eminently symptomatic. Lessen the inflammatory process by the continuous action of red light (red window-curtains or panes of red glass); tepid baths with cold sprays and—as recom-



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mended by Hebra—the use of a water-bed, scrupulous cleanliness of all accessible mucous membranes. Treat the skin by painting it with a 2 to 3 per cent. silver solution or apply nitrate of silver or ichthyol ointment (5 to 10 per cent.).

Vaccination.—Immunity is obtained against true small-pox by inoculating a human being with the infectious material after its virulence has been weakened by passage through the body of a lower animal; this is known as vaccination. In Germany vaccination is a hygienic measure which is required by law, and every healthy child between the ages of one to twelve years must be vaccinated. (The first vaccination may be postponed in children suffering from febrile and weakening diseases, anemia, rachitis, scrofula, and skin diseases). Animal glycerin-lymph is alone used as the inoculating material; it is obtainable at the Central Vaccine Institutions or from druggists, enclosed in little capillary tubes. The first vaccination is performed on the right, and the re-vaccination on the left, upper arm. With a vaccination lance from four to six incisions at intervals of about 2 cm. [.8 in.] are made over the deltoid muscle, care being observed to only incise the superficial layer of the skin, so as not to draw blood. [In this country glycerinated lymph is furnished in capillary tubes or on ivory points. A vaccination abrasion $\frac{1}{4}$ inch in diameter is all that is necessary. Care should be taken to merely remove the epidermis, not to cause bleeding.—Ed.] As the upper arm is grasped and held tense by the left hand of the physician the incisions stand open and the lymph can be easily introduced. Normally, the skin at the site of the inoculation turns slightly red in two days, becomes infiltrated on the third; from the fifth day on it is accompanied by a mildly remittent fever—up to 20 per cent. of first vaccinations are associated with albuminuria (Falkenheim)—and at times annoying itching; we observe the glossy and mother-of-pearl-colored pustules, which reach their highest degree of development on the seventh or eighth day. At this time the pustules already show an oval central umbil-

ication which is darkly colored. Occasionally the pustules are only surrounded by a narrow inflammatory zone, in other cases the skin of the whole inoculated area is reddened, swollen, and infiltrated. In the second week the pustular contents become turbid, then purulent, and turn yellow. The desiccation begins at the center, and a yellowish-, later, blackish-brown scab forms, which falls off in about twenty-three days after vaccination. White net-like or radial scars remain behind. Occasionally the eruptive stage is complicated by a measles-like, scarlatinal, or a vesicular vaccination rash, or, on account of scratching, pustules appear on various parts of the body; general vaccinia is, comparatively speaking, extremely rare. Worthy of note is the possibility of transmission to an individual suffering from some skin affection (eczema) who has not as yet been vaccinated.

The normal course may be considered disturbed if the vaccine has become infected with a pathogenic microorganism at the time of its manufacture or before its use, or if during the vaccination the wound becomes infected. The most frequent complications are erysipelas (which spreads from the site of inoculation soon after vaccination) and impetigo contagiosa. An infection of the pustules may also follow rupture or scratching of them (Heubner). To avoid infection, vaccination must be performed under strict aseptic and antiseptic principles (cleanse area to be inoculated with soap and alcohol), and the site of inoculation and the developing pustules must be protected as much as possible against mechanical injuries and the advent of bacteria. Worthy of recommendation are sterile vaccine points which are enclosed in glass tubes. A bath may be taken after the vaccination is manifest, provided the area is carefully protected from wetting. If inflammation becomes excessive, dust with lycopodium and make applications of moist boric acid compresses. In case of vaccination-erysipelas, resort to sublimate compresses immediately.

VARICELLA. CHICKEN-POX

Varicella is an acute febrile, vesicular exanthem, which runs a mild course.

The **period of incubation,** lasting from two to two and one-half weeks, is, as a rule, symptomless.

The **eruption** begins usually on the face and head, sometimes also on the trunk or the upper arm, with the appearance of small red nodules, which rapidly increase to the size of a lentil; the centers of these nodules form small water-colored vesicles after a few hours. The vesicles rapidly enlarge and soon involve the whole papule, but retain, as a rule, a light red border. They contain but one chamber, at first fairly well filled, which, as it increases in size, becomes umbilicated. The contents consist of a clear serous fluid which becomes turbid later, on account of which the originally gray vesicles assume a yellow color. After one or two days the vesicular contents dry up and honey-yellow, transparent, thin crusts are formed, which later turn brown. These scabs, when they fall off, leave a red spot over which the skin rapidly grows; in extremely rare cases of exceptional severity white contracted scars are left behind.

The number of vesicles is very variable, as a rule, only a few dozen. They are found on all parts of the body, but are thickest on the back, breast, and the scalp. Vesicles also develop in about one-third of the cases upon the conjunctival, oral, and pharyngeal mucous membranes; more rarely upon the genital mucosa. They soon lose their covering and are more likely to resemble aphthous ulcers. The eruptions of varicella never arise at one time, nor are all ever converted into vesicles. They occur more often individually within the course of several days, being separated by intervals of time; and it will be observed that one portion is papular in form and undergoes resolution without entering the vesicular stage. Thus we may note the various stages in development of the eruption side by side at the same time; small red papules with or without miliary vesicles; also vesicles of

PLATE 27

The Eruption of Varicella on the Fourth Day.—The second crop of the eruption consists of a few dozen vesicles, some of which are in process of development, while others are already beginning to suppurate. Of the first eruption nothing remains but little brownish scales and red spots. Mucous membranes not involved. (On the first day a number of pin-head-sized, grayish-yellow vesicles, surrounded by a red zone, appeared on the anemic palate.) Afebrile course. General health good; excessive itching. No albuminuria. Duration of disease was eight days.

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varying size on an infiltrated or slightly altered base containing light or turbid contents; finally, the various grades of desiccation and the remaining red areas of the skin.

The **duration** of the disease varies from five to ten days, depending upon the number of relapses, while full restoration of the skin occurs in about three weeks. During the first few days and at the appearance of each new crop of vesicles a high fever develops. The general health is otherwise disturbed only by the itching of the skin. The scratching which is indulged in may lead to secondary infection, suppuration of the vesicular contents, or to the subsequent formation of furuncles and deep-seated skin ulcers.

Variations in the eruption are designated as *varicella confluens*, *bullosa vel hæmorrhagica*. A mild form of nephritis develops in rare cases.

The **differential diagnosis** consists in distinguishing varicella from lichen urticatus and true small-pox. The eruption of lichen urticatus selects by preference the lower half of the body, feels very dense, and never undergoes a vesicular change. Of significance in true small-pox are the severe prodromata, high fever, erysipelatous swelling and reddening of the face and scalp, extensive coalescence of the vesicles and pustules, and the absence of the various stages of development of the eruption at one time, which is so characteristic of chicken-pox (Heubner).

Treatment.—Rest in bed for several days; non-irritating diet (nephritis). Protect against secondary infection



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(in case of excessive itching use thymol ointment or dust with talcum powder or cornstarch).

DIPHTHERIA

Diphtheria is an acute infectious disease characterized by the formation of membranous deposits and toxic constitutional symptoms.

The **cause** of diphtheria, the Klebs-Löffler bacillus, is deposited on the mucous membrane, preferably of the tonsils, of the nose, or of the larynx and trachea. In these regions it multiplies rapidly and after a variable period of incubation causes necrosis of the epithelium and marked alterations in the blood-vessels of the mucous membranes. These vessels are congested and at high tension and permit the blood-serum to leak out rapidly and in large quantities (*fibrinous exudate*).

Coagulation of the exudate causes the formation of the fibrinous diphtheritic pseudomembrane (see Plate 10, Fig. 1). The latter is sometimes loosely attached to the mucous membrane which has been deprived of its epithelium (*croupous*), but at other times it is adherent and extends deeply into the mucosa (*diphtheritic*). The diphtheria bacillus requires much oxygen and therefore spreads, as a rule, only on the surface of the mucous membranes, especially of the respiratory tract, and but rarely extends to the deeper tissues, as into the circulation and the internal organs. From this local focus the whole organism is supplied with the poisonous metabolic products, the diphtheria toxin. The latter rapidly enters the circulation and travels to the viscera, where, attacking the living cells, it causes the development of degenerative manifestations, especially of the heart muscles (fatty, and at times, waxy degeneration or secondary interstitial processes), the kidneys (parenchymatous nephritis), and the peripheral nerves (peripheral neuritis with inflammatory changes in the spinal cord).

The activity of the bacillus of diphtheria may be influenced to a certain extent by a mixed infection with other pathogenic bacteria, especially the streptococcus.

PLATE 28

FIG. 1. **Diphtheria of the Lips following Measles in a Child Two and a half Years Old.**—The upper and lower lips are greatly swollen and covered by thick greenish-yellow deposits (joined at the ends), which have spread inward to the oral mucosa. The pseudomembrane is firmly adherent and cannot be drawn off without causing hemorrhage and the loss of tissue (microscopic examination showed the presence of large numbers of the diphtheria bacillus). Fetor of the breath. The fauces are dark red, but free of deposit. The deposit disappeared in six days after local and specific treatment. (Clinic of von Ranke, Munich.)

FIG. 2. **Diphtheria of the Pharynx One Day After Serum Injection.**—Uvula, tonsils, and posterior pharyngeal wall are reddened; the median surfaces of the palatine tonsils present symmetric, whitish-yellow, sharply outlined fibrinous deposits, which are surrounded by a fairly broad blood-red zone (demarcation of serum action). Two days later the deposits had undergone softening, became smaller and smaller, and finally disappeared. The fever disappeared in three days.

The **symptom-complex** is very variable and depends upon: The *localization* of the primary disease focus; the reaction of the mucous membrane to the invasion of the bacilli; the quantity and quality of the bacterial poison on the one hand, the susceptibility of the individual to the poison on the other. The mucous membrane reacts in one case only with catarrhal manifestations, in another case with the formation of fibrinous exudates and necrosis. The organism reacts with a high ephemeral fever and mild albuminuria and at other times with grave phenomena: High fever, marked albuminuria, disease of the cardiac muscle, and paralysis.

The *local* and *general symptoms* need not necessarily correspond to each other in severity, for insignificant pathologic changes in the mucous membranes may be accompanied by the gravest manifestations of intoxication and the reverse (Escherich).

The **beginning** and **course** of the diphtheria may be fulminant or insidious, and at one time the local, at another the general, symptoms predominate. (A characteristic type of fever does not exist.) There is constant danger of the local process extending to the deeper air-passages with the sudden appearance of toxic symptoms. The duration of the disease is fairly indefinite, depend-



Fig. 1.

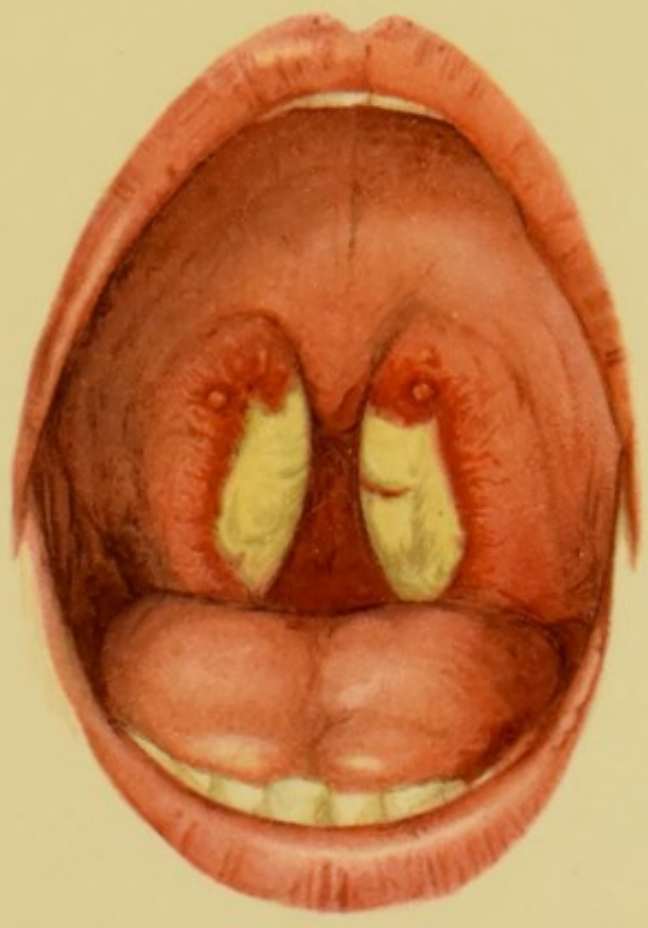


Fig. 2.

ing upon the severity of the case and the onset of complications.

According to the localization of the process we distinguish between pharyngeal, nasal, laryngeal, conjunctival, vulvar, and wound diphtheria.

The most common is the **pharyngeal diphtheria**. The mucous membrane of the pharynx is reddened and swollen and the tonsils and uvula are considerably enlarged. The tongue is heavily coated and the odor of the breath is fetid. Several small white fibrinous plaques are seen on one tonsil, more rarely on the uvula or posterior pharyngeal wall, which rapidly coalesce into an irregular continuous deposit. The latter may remain stationary or spread by contiguity or by bounds to symmetric parts of the opposite side. In the progressive form the isthmus of the fauces and, later, the posterior wall of the pharynx are soon covered with a thick layer of fibrinous exudate, and by ascending and descending processes the mucous membrane of the mouth, of the pharyngeal cavity, and of the larynx and trachea are also attacked. The deposit, which is originally white, soon becomes yellowish or yellowish gray in color; it is sharply outlined, elevated, tenaciously elastic, and may be fairly easily removed from the reddened infiltrated mucous membrane in large sections, accompanied by the loss of blood and tissue substance.

The lymph-nodes are always infiltrated and hard. In severe cases the lips and nares are excoriated by a serosanguinolent secretion; the speech, because of the immobilization of the velum palati, is nasal and the respiration is rasping and snorting. The toxic manifestations are variable in nature; when the process is very extensive they are usually quite pronounced, and consist of high fever, considerable albuminuria, and swelling of the spleen and liver; paralyses develop during the convalescence.

Aside from these typic forms of diphtheria, we also observe especially mild and particularly severe varieties. The mild type of diphtheria consists only of a severe

PLATE 29

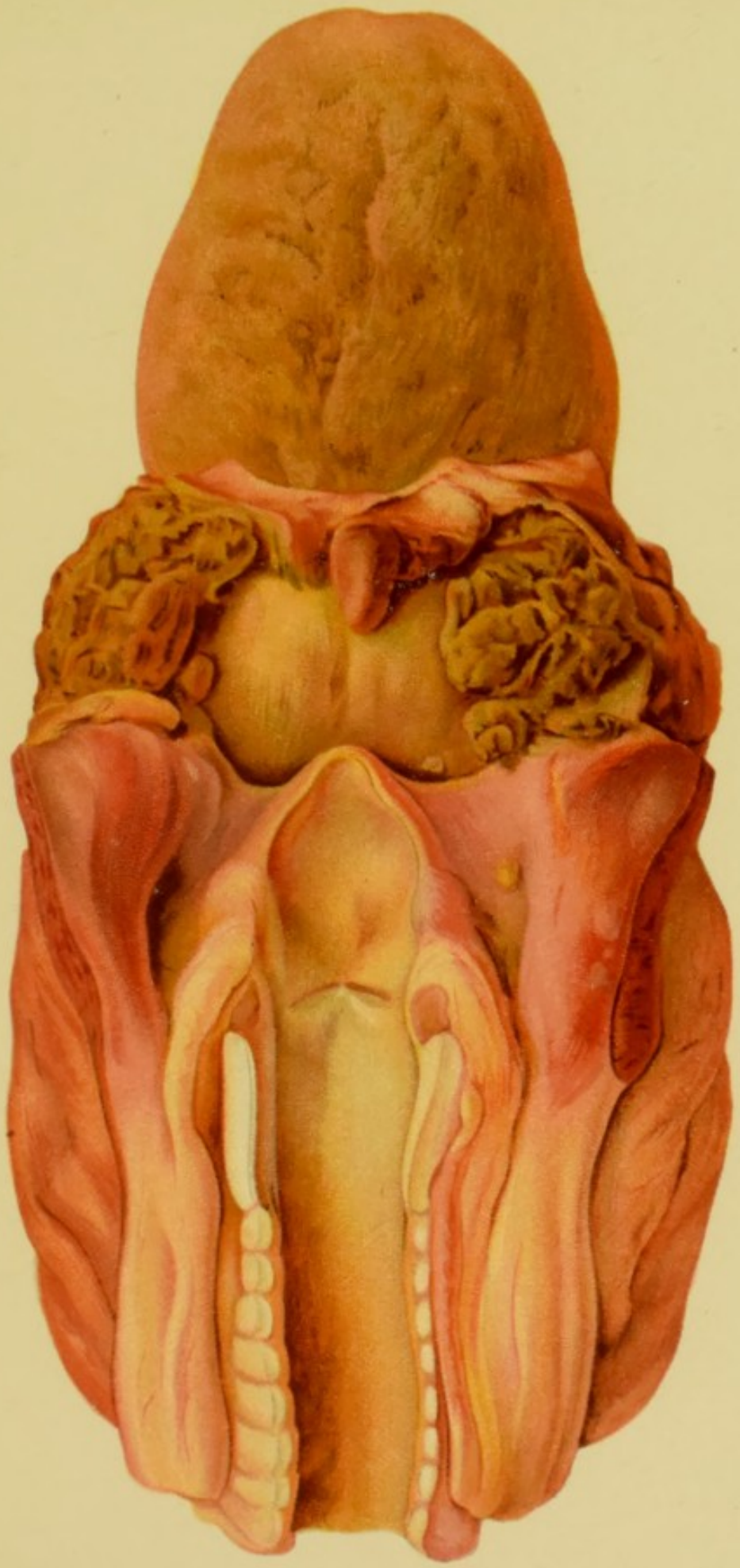
Diphtheria Gravis (Gangrenous, "Septic" Diphtheria).—"The tonsils on both sides are swollen to the circumference of a hazel-nut, their surfaces are irregularly fissured, have a foul odor, and a dirty, yellowish-brown color. On incision the parenchyma of the tonsils is found to have become gangrenous. The surrounding mucous membrane is decidedly reddened and swollen, the uvula is considerably thickened and glossy. The surface of the tongue has a dirty brown color. The larynx is not involved." (From von Bollinger, *Atlas of Pathologic Anatomy.*)

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and obstinate inflammatory catarrh of the pharyngeal mucous membrane; as regards the presence of membranous deposits, these are confined to single disseminated, mostly lacunar deposits (angina diphtheritica, diphtheria punctata). The toxic symptoms are likewise mostly mild, but may exceptionally be severe and lead to death before typic local changes are noticeable (hypertoxic form).

Diphtheria gravis (Heubner), formerly called "septic diphtheria," consists of extensive mucous-membrane involvement, accompanied by putrefactive processes and the gravest manifestations of intoxication. It is caused by extraordinary virulence of the micro-organism or by a high degree of individual susceptibility to the virus. The patients usually die soon from the double action of absorbed bacterial virus and the products of putrefaction or from pneumonia or pyemia.

Laryngeal Diphtheria (Croup).—This condition is usually an accompaniment to or a result of pharyngeal diphtheria; the latter has a marked influence upon the disease picture. With the primary localization in the larynx we note at first the symptoms of a laryngotracheitis, which, however, grows steadily worse; hoarse and finally toneless voice; a dry, irritating cough; difficulty in breathing. In beginning stenosis (inflammatory swelling—diphtheritic deposit) the auxiliary muscles of respiration become active and an inspiratory retraction of all yielding parts of the thorax occurs. Inspiration and expiration become slower, labored, and accompanied by crackling râles, especially on inspiration. Occasionally attacks of asphyxiation follow the collection of mucus and obstruction



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by loosened sections of membrane. If a severe form of increasing stenosis is not checked by an operation the process may even spread to the bronchial tubes, and the patients die from the double action of carbonic acid and diphtheritic intoxication.

Nasal Diphtheria.—This also is generally an accompaniment of pharyngeal diphtheria. In nursing infants the nose is often the primary seat of the diphtheria, and in this case there is a constant tendency to septic complications. We note the following symptoms: Swelling of the nose, obstructed, noisy nasal breathing, serosanguinolent, flaky, and (later) purulent discharge.

Rhinoscopic Picture.—Reddening and swelling of the mucosa; white fibrinous deposits, which are mostly confined to the posterior portions of the nose. Toxic symptoms also arise, as in other forms of diphtheria. A febrile purulent coryza is suggestive of diphtheria. The term "rhinitis (pseudo)membranacea" is employed to designate a benign form of nasal diphtheria with extensive membrane formation, but without disturbance of the general health.

Diphtheritic Conjunctivitis.—Diphtheria attacks the conjunctivæ rather rarely as a primary or a secondary condition. It arises gradually either in the croupous or the diphtheritic form (from an anatomic point of view), that is, deposits are formed which are easily pulled off, or a pseudomembrane, varying in color from bluish white to that of amber, develops on the conjunctiva, which can be loosened only at the expense of hemorrhage and loss of substance. The diphtheritic form may also involve the bulb and not rarely the cornea also. The serosanguinolent secretion is converted into a blennorrhea during the healing stage. Toxic symptoms and resulting conditions may occur, as in any other form of diphtheria.

Diphtheria of the Vulva.—This is a rare localization of diphtheria which is associated with marked manifestations of a severe intoxication. The mons veneris, the inner surface of the thighs, and the labiæ majores are considerably swollen and reddened and the neighboring

PLATE 30

FIG. 1. **Diphtheria of the Conjunctiva in a Young Boy.**—"The inflammatory swelling and reddening of the upper lid is more pronounced than in blennorrhea neonatorum; the skin of the lower lid and in the region of the inner canthus has undergone purulent infiltration, and is partially eroded by the discharge.

FIG. 2.—"The lower lid of the same case inverted, to show the depth to which the diphtheritic infiltration of the conjunctiva has extended; it is discolored yellowish gray." (From Haab, *Atlas of the External Diseases of the Eye.*)

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lymph-nodes are markedly infiltrated. Multiple disseminated—and sometimes coalescing—ulcers, varying in size from a lentil to a bean, are seen on the vaginal margins; these ulcerations are covered by a grayish-white, closely adherent deposit. In some cases the whole vulva is covered by a connected dirty gray deposit, beneath which a deep-seated necrosis exists.

Complications and Sequelæ of Diphtheria.—The commonest complications are nephritis, bronchitis, and pneumonia. The sequelæ consist of cardiac weakness and paralyses. The danger of paralysis of the heart is ever present during the acute stage and in convalescence, and requires the greatest care in treatment. The heart loses its strength either gradually or death due to heart failure may arise suddenly. Both conditions are caused by alterations in the cardiac muscular fibrillæ, which, according to Eppinger, are a direct sequel of a toxic edema due to the diphtheritic virus. The postdiphtheritic paralyses are a manifestation of peripheral neuritis whose course is unaccompanied by fever, pain, or paresthesiæ. Recovery occurs almost without exception in from four to six weeks. The velum palati and certain ocular muscles are especially prone to become paralyzed; more rarely the muscles of the trunk and the extremities. Threatening life are paralysis of the larynx and pharynx, the abdominal musculature, and that of the diaphragm.

The **prognosis** of diphtheria is dependent upon the age and strength of the patient (the older the patient the more favorable the prognosis), upon the character of the epidemic, and the time at which skilful scientific treat-



Fig. 1.

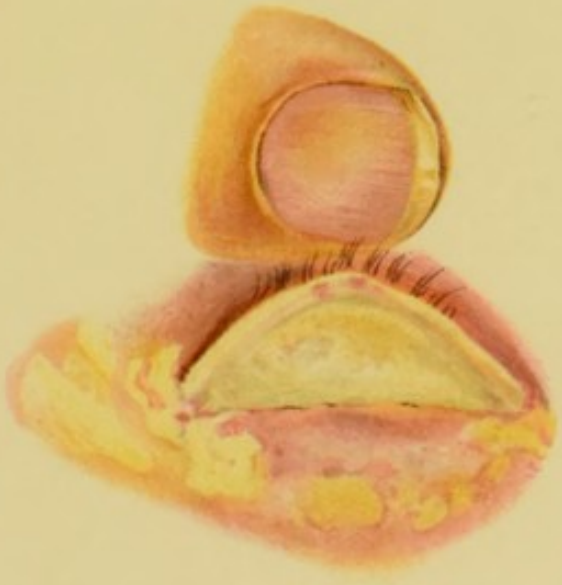


Fig. 2.

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ment is obtained. The mortality rate, when serum therapy has been employed, is only a small percentage (von Rauchfuss, Bayeaux); in operative cases it equals about 36 per cent. (Siegert).

Diagnosis.—The greatest difficulty is met with in distinguishing diphtheritic from non-diphtheritic angina, and laryngeal diphtheria from pseudocroup. In the microscopic and bacteriologic examination, which should be resorted to in every doubtful case, we must bear in mind that the discovery of single bacillus on the inflamed mucous membrane, when the diphtheria bacilli are spread widely over the mucous membrane of a healthy person, is not indicative of the diphtheritic character of the disease, for the latter only holds true when diphtheria bacilli are found in colonies. On the other hand, the diagnosis of diphtheria in a case which appears clinically to be such should not be rejected because the specific bacillus has been displaced by other bacilli or because it was not found by chance in the examined material. For microscopic examination remove a bit of the deposit with a pair of forceps, wash in distilled water, and spread between two cover-glasses. Fix over a flame. Stain with Löffler's methylene-blue. The diphtheria bacilli are slender, slightly curved rods, about as long but twice as wide as the tubercle bacillus; the ends are often clubbed and assume a characteristic angular position. They stain intensely with methylene-blue and are peculiarly nucleated.

Treatment.—*Specific Treatment: Serum Treatment.*—The antitoxin should be injected as early as possible in the progressive form of diphtheria when signs of laryngeal involvement and toxic symptoms occur, even in the mild types. The serum has a local as well as a constitutional action. It prevents further progress of the local process and hastens the dissolution of the fibrin. It also neutralizes any diphtheritic toxin which may be in the circulation at the time of injection. Failure to relieve is due to either a severe and irreparable toxic action before the time of serum injection or to a mixed infection, in

FIGURE 93

Microscopic findings in diphtheritic angina. Colonies of diphtheria bacilli, isolated cocci, and thready fibrin. Enlarged 510 times.

FIGURE 94

Microscopic findings in lacunar angina which is non-diphtheritic in character. Diffused bacterial growth. Of the numerous varieties none seem to predominate. Spare fibrin threads. Enlarged 510 times.

which case we can only expect the antitoxin to influence the specific and not the foreign virus. Irrespective of age the following injections should be made: In localized diphtheria, 1000 I. U. (Behring II.); in progressive diphtheria or with involvement of the larynx, 1500 I. U. (B. III.); in laryngeal stenosis or severe intoxication, 2000 to 3000 I. U. (B. D. IV.–VI.); as a prophylactic inject 600 to 1000 I. U. (B. I. or II.).[1] [The above doses may be considered small. In a case of clinical diphtheria 3000 units at least should be given at the onset. This dose may be repeated at intervals of six hours until some effect is produced on the membrane.—Ed.]

The serum may be injected with any syringe containing 5 cc. and which can be easily sterilized. For the injection, a portion of the skin should be selected beneath which there is loose subcutaneous tissue, as, for instance, the side of the chest. [The gluteal region is easily accessible when the child is held on the lap of mother or nurse, and is a favorite site for these injections.—Ed.] The site is thoroughly cleansed; a fold of the skin is seized in the fingers and the cannula introduced parallel to it to such a depth that the latter is freely movable in

[1] The curative serum is usually obtained from horses which, after careful preparation (repeated injections of gradually increasing doses of the diphtheritic virus), are immunized to a high degree against diphtheria. The value of the antitoxin thus obtained is determined by its action against tested solutions of the diphtheritic virus. That amount of serum which in strength equals 100 times the amount required to neutralize a dose of virus which is fatal to guinea-pigs is spoken of as an immunization unit = I. U. If this action is obtained in 1 cc. of the serum, we speak of a simple serum; if it already exists in $\frac{1}{100}$ part of a cc., we speak of 100-fold serum, etc. At the present time 250-, 400-, and 500-fold serum may be obtained on the market.

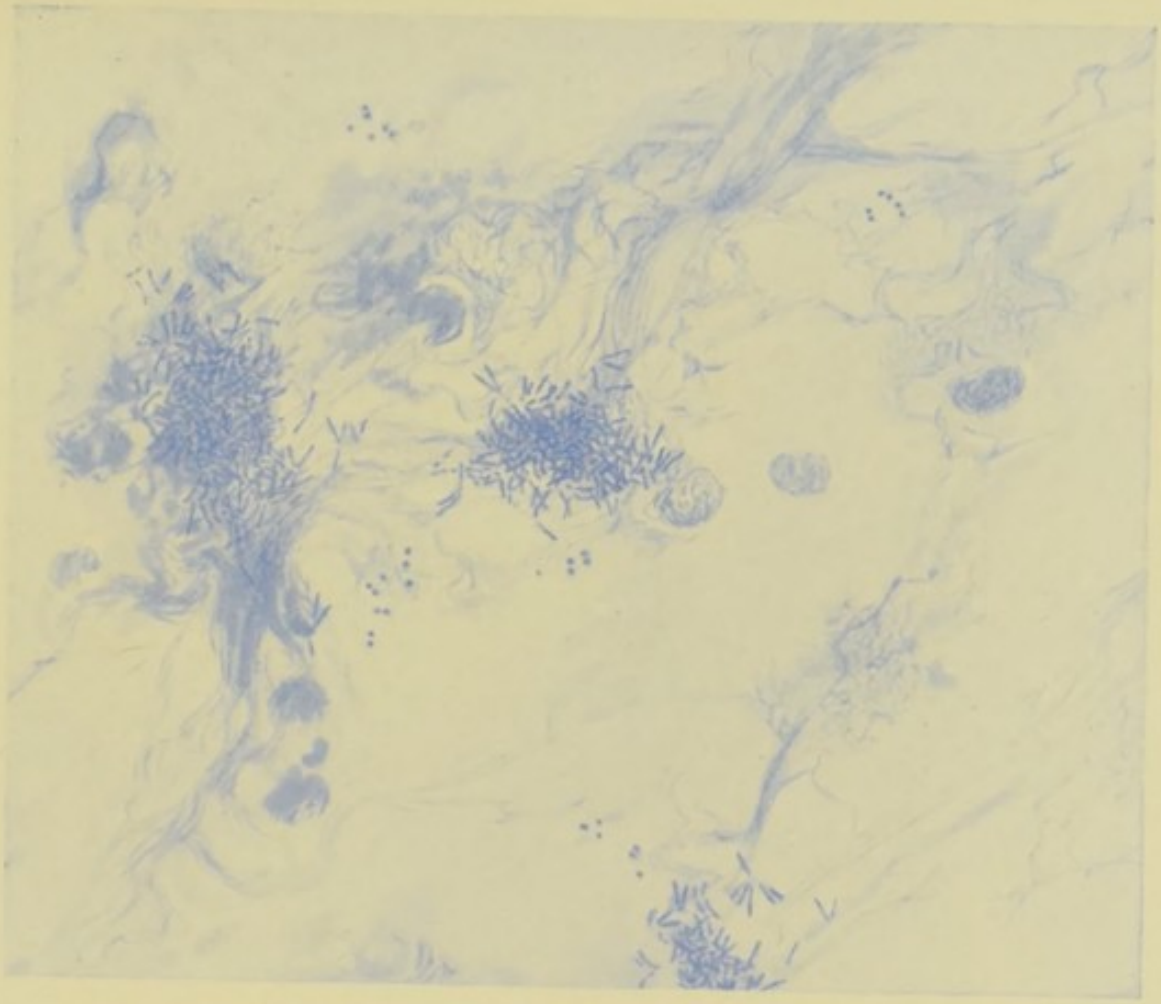


Fig. 93.

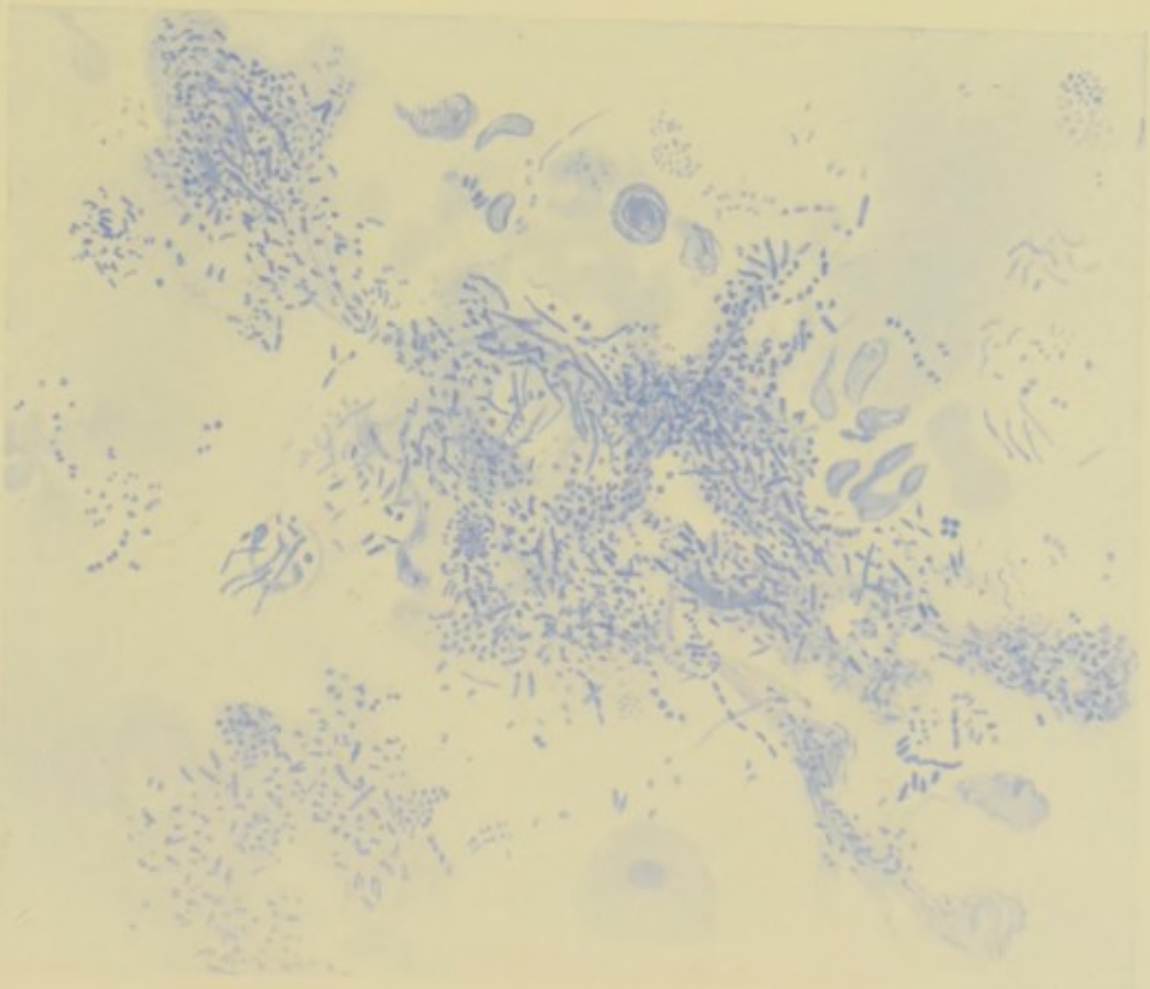


Fig. 94.

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the subcutaneous cellular tissue. Before withdrawing the cannula a piece of adhesive plaster should be applied to the point of injection to prevent the escape of the serum and the infection of the wound. Massage of the swollen area which results is unnecessary. Pains in the wound disappear within twenty-four hours. Occasionally within the first fourteen days after the injection we may note an increase of fever, constitutional disturbances, and the appearance of morbilliform, scarlatina- or urticaria-like rashes, and, in rare cases, articular pains. These ill effects of the serum injection disappear without leaving any traces behind within several hours or, at the most, within one or two days. The serum has no other untoward effect.

Local and Constitutional Treatment.—Cleanliness of the mouth; nasal douches; hydrotherapeutic measures; neutral soap-baths (35° C. [95° F.]), with cold rubbing. Light, stimulating diet. The nephritis and paralyses usually require no special treatment.

Special Treatment.—In nasal diphtheria douche the nose with weak antiseptic solutions or insufflations of powdered boric acid or sodium sozoiodol. For diphtheria of the conjunctiva make warm

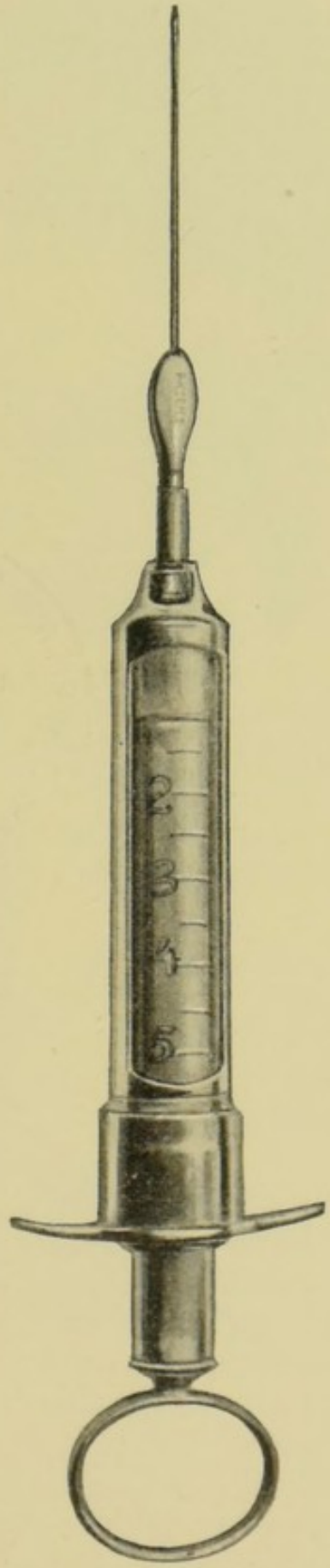


FIG. 97.—An easily sterilizable serum syringe with a metallic piston (modified by Walcher).

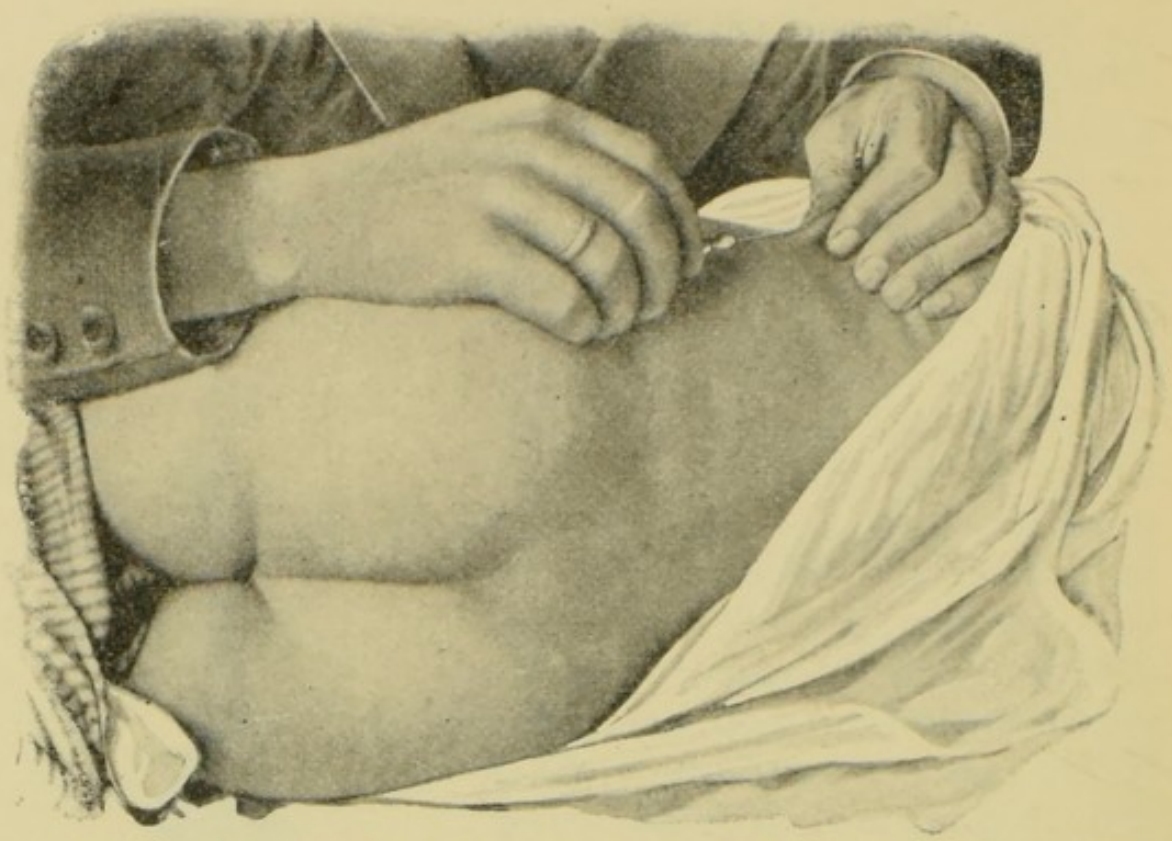


FIG. 98.—The injection of serum in the left axilla, the arms being tightly supported on both sides, so that the patient cannot interfere with the operation. The cannula is introduced, parallel to the surface of the body, into the elevated fold of skin.

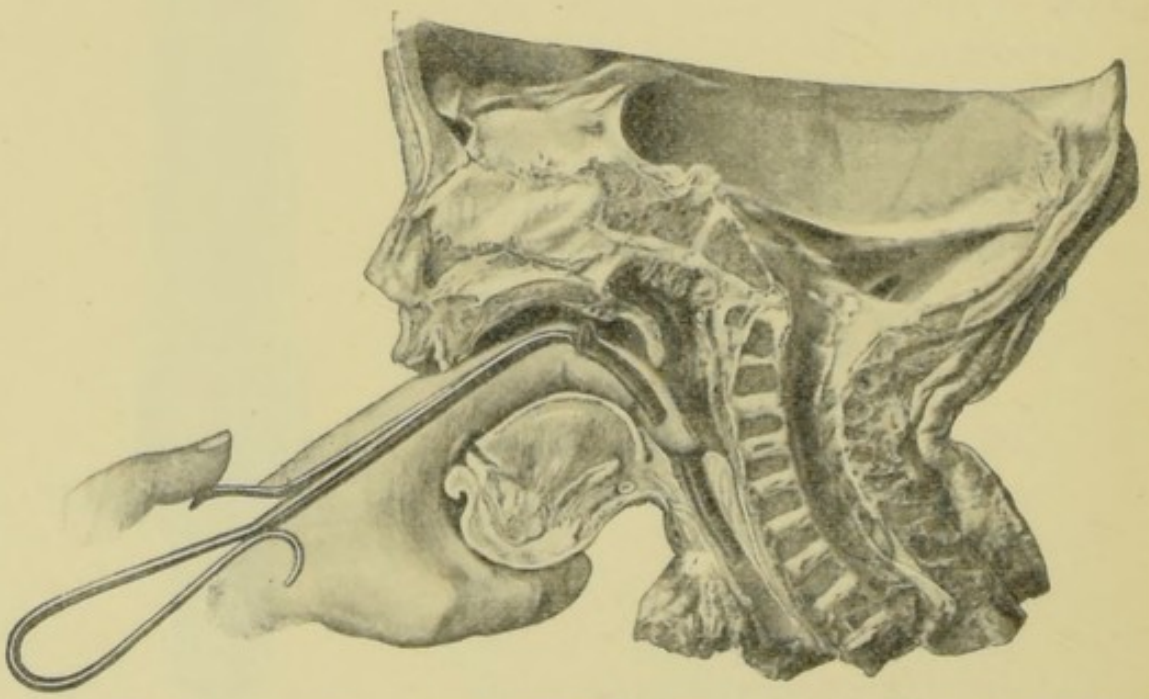


FIG. 99.—Intubation with elastic tubes. As the tube is being put into place it assumes the curvature of the introducer, which conforms in shape to the curvature of the tongue. After it has been introduced it follows the curvature of the laryngotracheal tube. (Note how the left index-finger, which lies at the entrance to the larynx, draws the epiglottis and root of the tongue forward and upward in order to expose the entrance into the larynx as much as possible.)

applications; apply disinfectants for diphtheria of the vulva; after cleansing, apply boric-iodoform powder. At the beginning of laryngeal stenosis order a hot bath, followed by sweat-stimulating packs; energetic vapor treatment in order to reduce the inflammatory swelling and to hasten softening of the membrane. When the stenosis threatens life, resort to intubation (O'Dwyer) or tracheotomy.

Intubation consists in introducing into the larynx by way of the mouth a small tube constructed of metal, hard rubber, or some elastic material, and allowed to remain in place until the local process has undergone resolution, which is about three days. The patient is wrapped from neck to feet in a sheet, and intubated while lying in bed or sitting on the lap of an assistant, who fixes with his thighs the child's legs, holds the mouth open with one hand and the head in a median position with the other. The tube is inserted by first introducing the left index-finger far into the pharynx to hold the entrance of the pharynx open by pressing the tongue as far forward and upward as possible and the epiglottis against the root of the tongue. During this operation the following precautions are necessary:

The instrument must be introduced in the middle line to avoid entering one of the different lateral mucous fossæ.

The handle of the introducer should be raised after the epiglottis is passed, in order that the tube will not glide over the entrance of the larynx, which is half covered by the tongue, into the esophagus.

After the tube is inserted into the larynx the handle should again be lowered to avoid injuring the anterior wall of the larynx by the tube. The operation should last only a few seconds. Accidents during the operation itself are rare, but, on the other hand, difficulty in swallowing, coughing the tube out, or obstruction of the tube, and the more extensive formation of pressure ulcers are more or less serious accompaniments. [In the hands of experienced operators this procedure is not difficult, hence accidents are rare. In the inexperienced,

however, injuries to the mucosa of the larynx are more common than would be expected from the literature.—Ed.]

The *extubation* is performed either by means of a pharyngeal forceps-like instrument or, better, by the use of a silk thread, one end of which is fastened to the head of the tube and the other end passing out of the

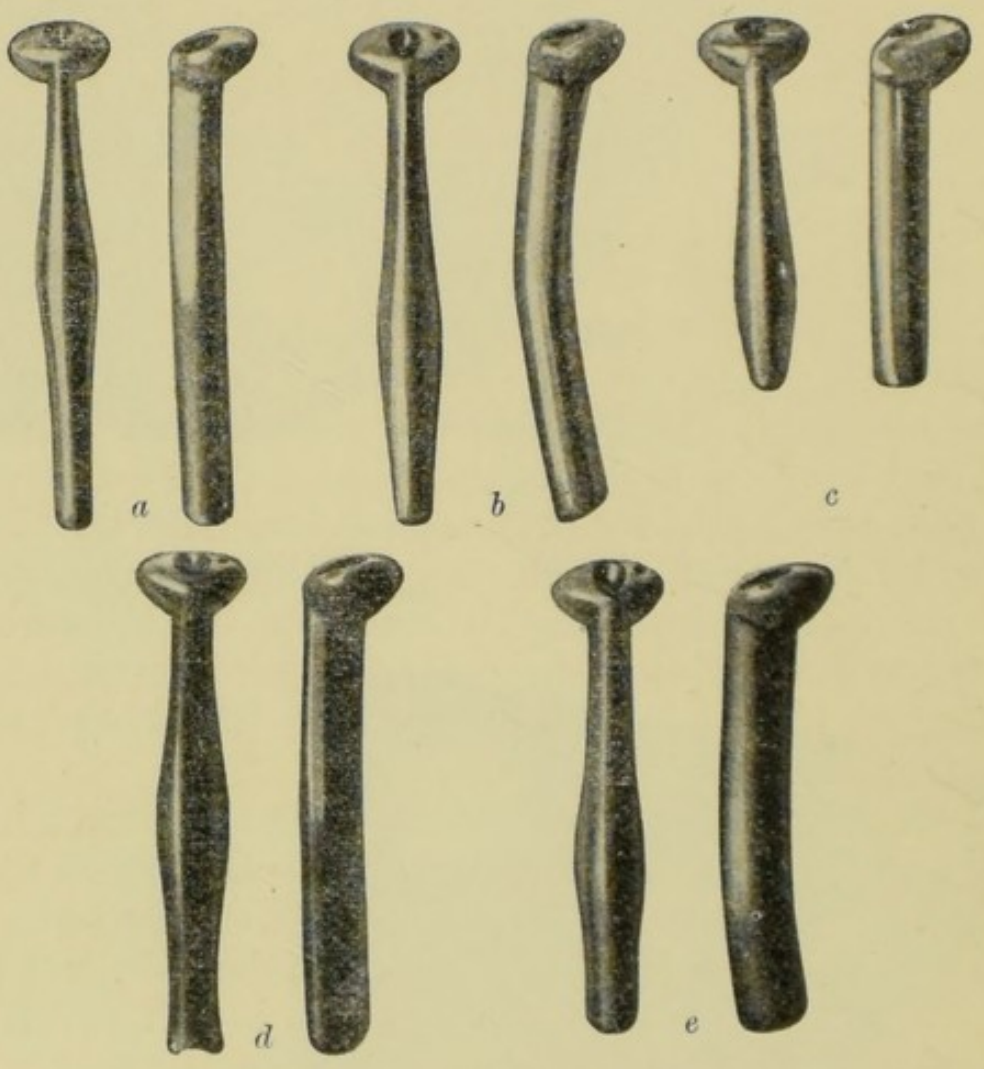


Fig. 100.—Intubation tubes. Metallic tubes: *a*. O'Dwyer's original tubes. *b*. Bauer's curved tubes. *c*. Bayeux' short tubes. Rubber tubes: *d*. O'Dwyer's ebony tubes. *e*. Trumpp's elastic tubes.

mouth, which is fastened to the cheek by means of a piece of adhesive plaster.

If the existing conditions prevent free respiration through the tube or if, for any reason, it is impossible to introduce the tube, the **bloody operation** must be substituted, and the trachea opened above or below the isthmus of the thyroid.



FIG. 101.—Intubation (O'Dwyer's instrument, ebony tube). First operation: Introduction into the mouth. The handle of the introducer is depressed.



FIG. 102.—Intubation. Second operation: Insertion into the larynx. Handle of the introducer is elevated.

PLATES 31-33

Tracheotomy

The plates show the various tissues of the neck which must be severed in tracheotomy. The incisions are so presented as to show simultaneously the important anatomic relationships in high and low tracheotomy. In practise the skin incision in high tracheotomy is made 1 cm. [.4 in.] higher and in low tracheotomy 1 cm. [.4 in.] lower than is shown in the figure.

PLATE 31.—The skin has been incised and the subcutaneous cellular tissue exposed. The hyoid bone, the thyroid and cricoid cartilages, the trachea, the thyroid. and thymus glands are traced in dotted lines for purposes of demonstration. In palpation remember that in small children only the hyoid bone and the cricoid cartilage can be plainly felt, and that the latter cartilage (not as in the case of an adult, the thyroid) represents the most prominent portion.

PLATE 32, FIG. 1.—The adipose tissue has been severed and the superficial cervical fascia with the branches of the inferior thyroid vein brought into view. The musculature with the linea alba is seen to shine through the fascia.

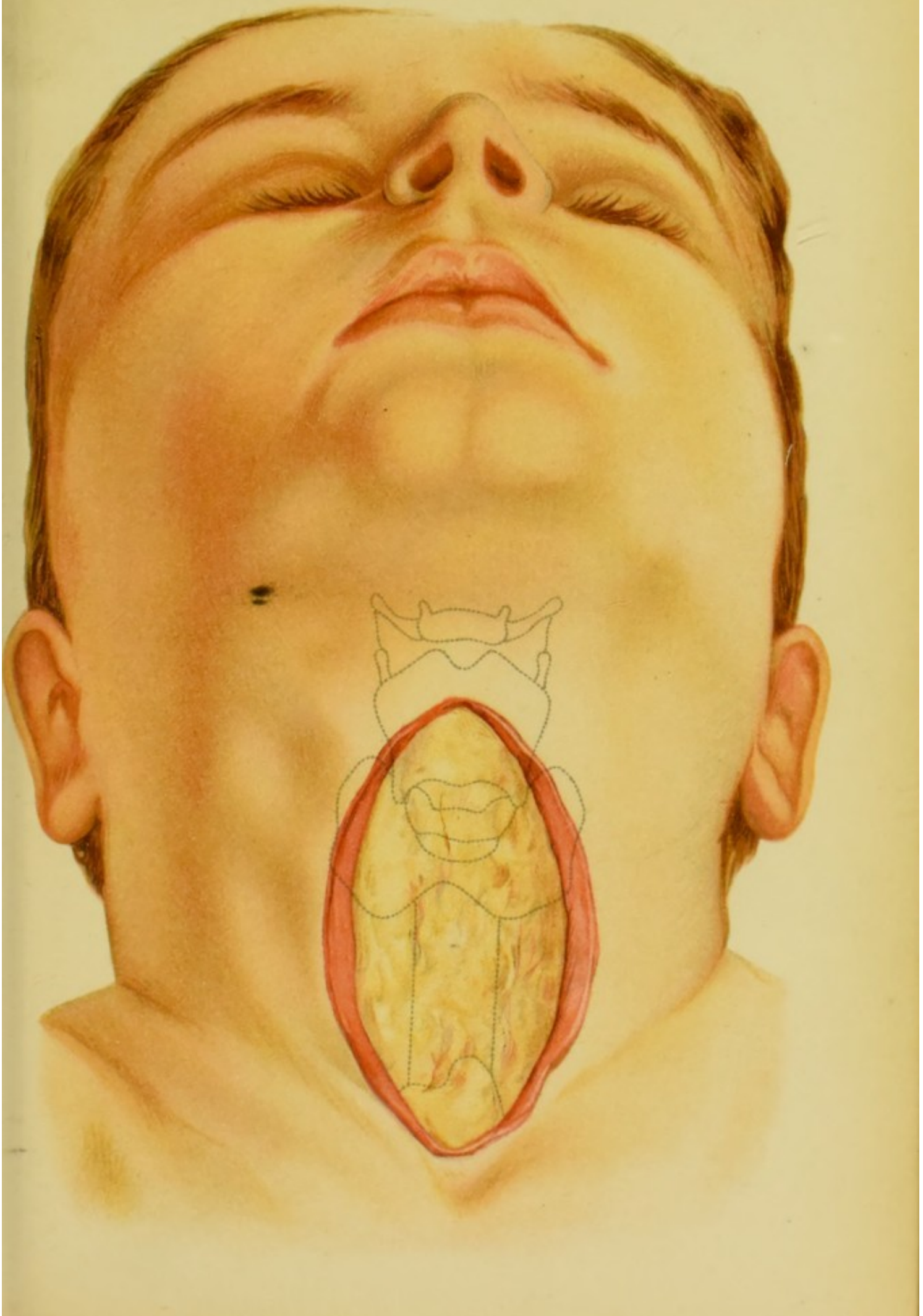
FIG. 2.—The superior cervical fascia has been incised and the sternohyoid muscles, which are joined in the median line by the linea alba, exposed.

FIG. 3.—The musculature has been cut and the deep cervical fascia exposed.

FIG. 4.—The superficial layers of the deep cervical fascia are incised, and we see exposed at the upper portion of the wound the isthmus, which is about 1 cm. [.4 in.] wide, and, to a certain extent, the lateral lobes of the thyroid gland, also the anastomosis of the inferior and superior thyroid veins. The thymus gland, which protrudes markedly upward, is seen at the lower end of the wound. Loose cellular tissue and the deep layers of the cervical fascia lie between the thyroid and thymus gland, also the anastomosis of the inferior thyroid with the anterior jugular vein.

PLATE 33. **Low Tracheotomy.**—Cellular tissue and the deep layers of the cervical fascia are severed and the trachea exposed between the thyroid and thymus glands. To the left of the lower portion of the wound is the innominate artery, which is in a high position. (The innominate, as a rule, occupies a high position in children during the first year of life, also—as is not rarely the case—during the second and third years. This must be remembered to avoid injuring it.)

The patient is wrapped in a sheet (as in case of intubation), placed on a table, and—in order to obtain full extension of the neck—a bottle wrapped in cloth is placed underneath it. An assistant attends to the anesthetization (which in profound carbonic-acid intoxication is unnecessary) and watches the neck of the child during the operation, in order to prevent lateral movement or



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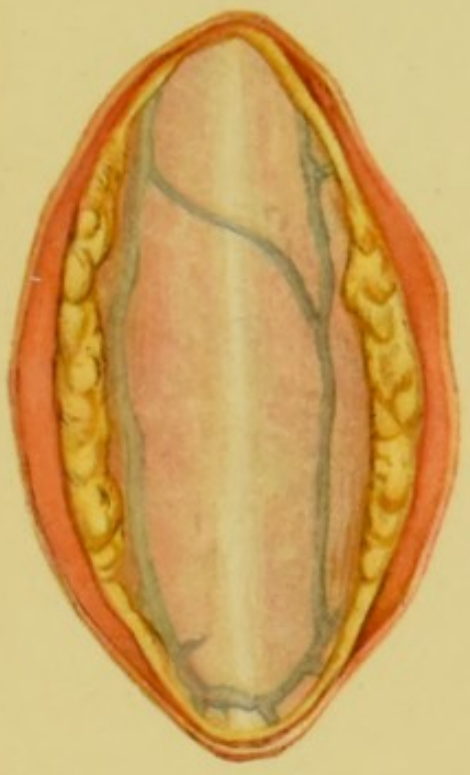


Fig. 1.



Fig. 2.



Fig. 3.

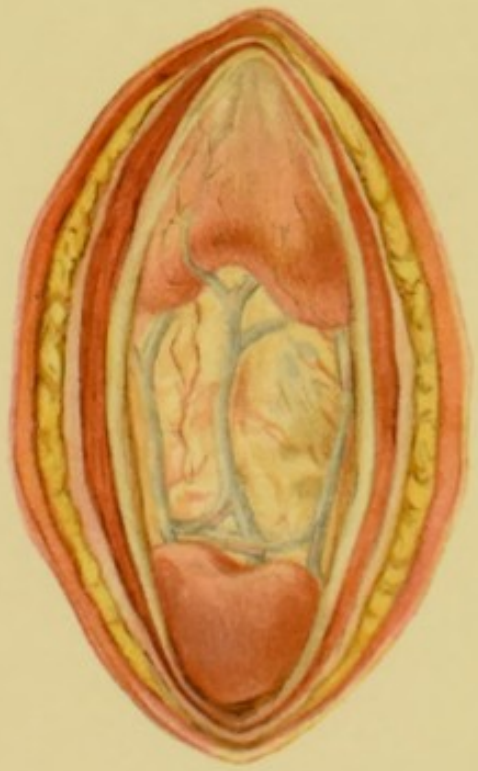
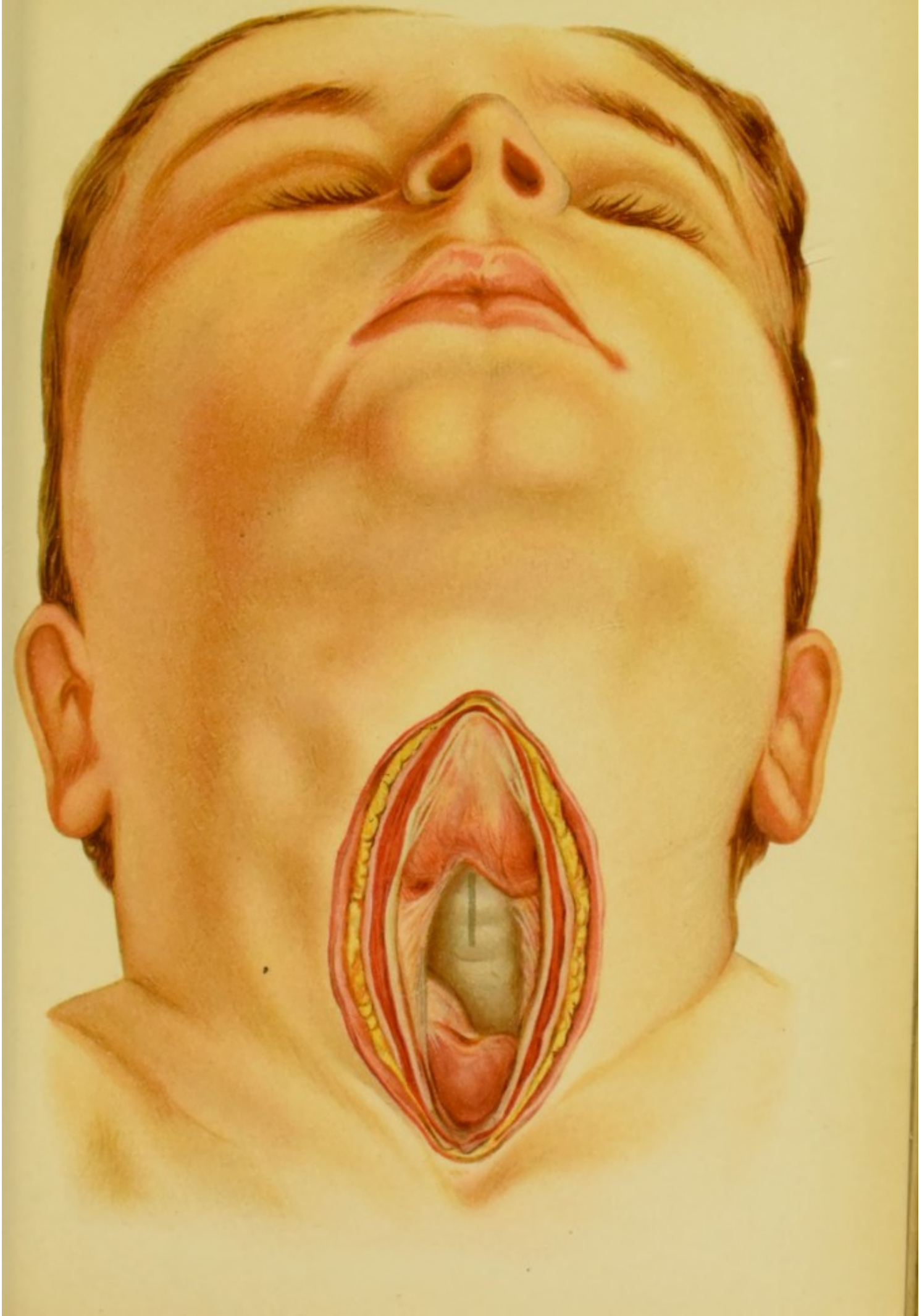


Fig. 4.

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displacement. The same asepsis is necessary as in any other bloody operation. The incision of the skin as well as that of the other tissues should be exactly in the median line. The opening in the skin should be at least 5 cm. [2 in.] long and reach, in case of superior tracheotomy, to the chin; in the case of inferior tracheotomy, to the sternum. The subcutaneous tissue is retracted by means of two artery forceps; next the superficial fascia and beneath it the glistening linea alba of the sternohyoid muscles are severed on a grooved director. The next step differs in high and low tracheotomy. In the former, a transverse incision is made through the deep cervical fascia, which lies exposed beneath the muscles, to the lower edge of the cricoid cartilage. It is then loosened by blunt dissection and drawn upward together with the enclosed thyroid gland to expose the trachea. In low tracheotomy the cervical fascia is incised in a longitudinal direction, layer by layer, on a grooved director until the thyroid gland is reached. After the deepest layer has been severed, the trachea, which lies partially exposed, is caught by two sharp hooks and drawn upward and freed by blunt dissection of any loose cellular tissue which may still adhere. A sharp-pointed knife is now forced through the trachea until the hissing sound of the escaping air informs us that the tracheal lumen has been opened; the incision is then sufficiently enlarged with a probe-pointed bistoury to permit the entrance of the cannula, that is, from 1 to 1.5 cm. [.4–.6 in.]. Not until the breathing has become absolutely free is the cannula (with a movable shield, as recommended by Lüer or Hagedorn) introduced and fastened to the neck with a simple band. The wound is well dusted with iodoform and covered with a piece of lint or gauze to receive the expelled tracheal secretion, and also with a piece of gutta-percha or a piece of cambric.

Difficulty may be encountered in performing this operation by the presence of an abnormally large thyroid gland, or its close union with the trachea, a large thymus, numerous congested venous branches, and (in rare cases)

arterial anomalies. The after-treatment is complicated by the presence of post-operative hemorrhages, dysphagia, obstruction of the cannula, and decubitus.

For the sake of cleanliness the cannula must be changed on the third day. To do so, retract the soft parts which are still ununited with hooks and pass a catheter, with a large opening, through the cannula into the trachea and employ it as a conductor for the removal of the old and introduction of the new cannula. In one or two days a speech-cannula is inserted, and by closing the same the permeability of the larynx may be tested. If the child passes through a night well and sleeps undisturbed the closed speech-cannula may be removed and the wound allowed to heal.

TYPHOID FEVER

Typhoid fever is an infectious disease which is primarily localized in the intestines and accompanied by swelling of Peyer's patches and of the spleen. It occurs in children, especially after five years of age, almost as frequently as in adults.

The **morbid anatomy** as well as the **symptom-complex** is, on the whole, the same in older children as in adults, but in younger children there is a decided difference. In the latter the upper portions of the intestine are chiefly involved and the morbid process is not as deeply seated, accordingly necrotic eschars, extensive typhoidal ulcers, and intestinal perforation are rare in children.

Clinically, we frequently note in place of the typic diarrhea (*pea-soup stools*) an obstinate constipation, with partly pappy and partly hard nodular stools. In other respects the disease picture represents the mild typhoid of adults (*gastric fever*): Dyspepsia, headache, remittent fever, slight swelling of the spleen, and sometimes roseola. The manifestations are frequently so slightly characteristic of typhoid that the diagnosis remains doubtful, and is only made possible by severe relapses or by etiologic relationship with other undoubted cases of typhoid fever. Sometimes the condition passes into

a form of moderate severity which is accompanied by an initial pseudomembranous angina, epistaxis, cerebral irritation, diarrhea, dry bronchitis, and marked loss of strength. The severe type of typhoid in children does not differ much either in its course or complications from that disease in adults; children, however, complain more frequently of abdominal pain, the nervous symptoms are more prominent, and the whole duration of the disease is, in general, shorter.

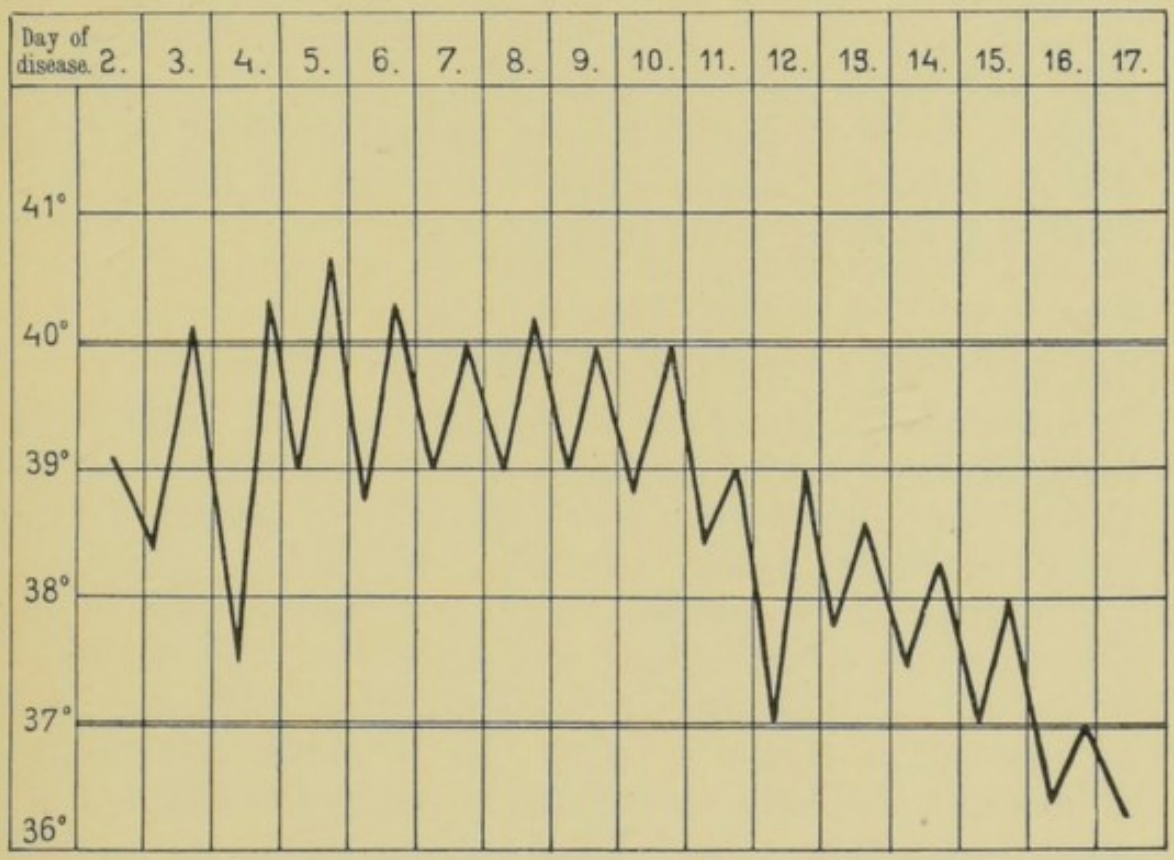


FIG. 103.—The type of fever in typhoid fever of childhood (Gerhardt-Seiffert).

The **diagnosis** is made from the characteristic step-ladder-like ascent of the fever, the roseolar rash, enlargement of the spleen, absence of leukocytosis (Baginsky) (leukocytosis in pneumonia), diazo-reaction of the urine, Gruber-Widal reaction (agglutinating action of diluted blood-serum from a typhoid patient upon the typhoid bacillus).

The **prognosis** in children is, on the whole, more favorable than in adults.

Treatment.—The strictest observation of all hygienic measures, especially as regards cleanliness of the mouth. Baths from three to six times daily at a temperature of from 30° to 35° C. [86°–95° F.] and of five to ten minutes' duration, during which the child is energetically rubbed and finally douched with water which has been cooled off by means of ice. The number and temperature of the baths depend less upon the degree of fever than upon the severity of the attack (Heubner). In case of very high fever and severe diarrhea make cold applications to the chest and abdomen, which are changed every fifteen minutes. Give large quantities of liquids and limit to a milk or carbohydrate diet.

INFLUENZA

Influenza in children is characterized by attacks of high fever, an initial retropharyngitis, and toxic constitutional symptoms which are especially referable to the gastro-intestinal canal, the nervous system, and, to a less extent, the respiratory organs. The bacillus of influenza seems to attack by preference the mucous membrane of the postpharyngeal wall, from whence, after an incubation period of from one to eight days, it distributes its poisonous metabolic products throughout the whole organism. Prodromal manifestations are usually absent.

Symptoms.—The disease begins with the development of pronounced weakness, headache, sometimes chills, and a high remittent fever which often lasts but two or three days. Inspection of the pharynx discloses a diffuse redness of the dry mucosa, a *retropharyngitis* (Soltmann). Pain develops in the neck, back, joints, and head, where it is very severe. The appetite is lost, the pulse is small, rapid, and at times arhythmic. Not rarely symptoms of cardiac weakness and mild cyanosis are met with. The remaining symptoms depend largely upon the age of the

child. In older children as well as in adults the phenomena of a descending catarrh of the respiratory tract exist; frequently also an influenzal croup (descending croup with exceptionally tenacious expectoration). On the other hand, in younger children the dyspeptic or enteritic (Baginsky, Schlossmann) and cerebral symptoms predominate (diarrheic, mucous stools of a foul odor, sometimes the typhoidal state, slight enlargement of the spleen, coma, delirium, meningitic symptoms or true primary influenzal meningitis, due to infection of the blood by the bacillus of influenza).

The *bronchial* and *pulmonary phenomena* in influenza show a remarkable and characteristic resistance toward the ordinary therapeutic measures. They are, in general, not of a serious character; however, cases of bronchopneumonia are met in which the confluence of lobular foci lead to consolidation of whole lobes, and death may even occur, due to abscess or necrosis. Influenza frequently involves the tympanic cavity, in which case hemorrhagic inflammation of the tympanum and suppuration of the middle ear almost always develop (Hartman, Heubner). Conjunctivitis and extreme photophobia are not rare complications (Spiegelberg, Comby). In about 12 per cent. we note the development of a measles-like, roseolar, or scarlatinal eruption (Schlossmann), and, more rarely, nephritis.

The **duration** of the disease is from three days to as many weeks and, rarely, longer.

The **prognosis** is, on the whole, more favorable than in adults.

The **diagnosis** may be made in questionable cases from catarrh, bronchitis, pneumonia, and meningitis by the detection of the bacilli of influenza. The latter are minute rods, usually occurring in pairs, which lie in large groups between the pus-corpuscles and frequently also within the cells. Doubt as to the influenzal nature of gastro-intestinal symptoms may be settled by the associated joint and muscle pain and headache.

Treatment. — Symptomatic (at the beginning of the

disease procedures which increase perspiration may be indicated). Give as many decigrams of quinin twice daily as the patient is old in years.

WHOOPING-COUGH. PERTUSSIS

Whooping-cough is a catarrhal affection of the upper air-passages occurring in epidemics, which is characterized by marked irritation of the respiratory mucous membrane, and especially by attacks of coughing, accompanied by a prolonged crowing inspiration which occurs frequently at night.

The **laryngoscopic** and **pathologic findings** consist of a catarrh of the upper air-passages extending into the large bronchi, accompanied by redness, swelling, and softening of the mucous membrane, with the excretion of an extremely tenacious discharge, which is rich in mucin and contains varying quantities of pus-cells. The greatest degree of reddening is noted in the interarytenoid space and at the bifurcation of the trachea. Laryngoscopic examination shows these areas to be particularly irritated, and that the passage over them of the tenacious mucus flakes causes the typic spasmodic attacks of coughing. The characteristic changes in the lungs consist of ecchymoses of the pulmonary cortex, acute distention of the apices, emphysema, and distention of the bronchioles. The latter contain a thick, creamy pus, which at times finds its way into the alveoli, these, through violent inspiration of the secretion (into the formerly collapsed alveoli), become dilated to the size of a pin head or pea (Fauvel, Ziemssen). Dilatation and hypertrophy of the right heart is nearly always present (due to increased pressure in the pulmonary circulation). The clinical manifestations at the beginning and during the stage of decline show little that is characteristic. We distinguish between an initial catarrhal stage, a catarrhal convalescent stage, and the interval between them, or the convulsive stage.

The period of **incubation** lasts from three to ten days and is symptomless.

Symptoms.—The *initial catarrhal stage* is marked by the symptoms of a febrile laryngo-tracheo-bronchitis, which resists the treatment of an ordinary catarrh. Toward the end of the initial stage the catarrhal symptoms gradually disappear, and the cough, which is at times loose and at other times dry, assumes a peculiar metallic tone. It occurs more frequently at night and becomes more spasmodic. The initial and frequently high fever sinks after a few days, as a rule, to normal, and the constitutional symptoms lessen in severity.

In about two weeks after the commencement of the first symptoms of the disease the attacks of cough develop less often, but are of a convulsive character—*convulsive stage.* The convulsions are preceded for several seconds or a minute by an aura in the form of a tickling sensation or burning of the throat, a feeling of oppression, great restlessness, nausea, and tracheal rattling. The cough, which has been vainly held back, then breaks forth; numerous expiratory coughs follow each other, interrupted only now and then by a laborious, sighing, and crowing inspiration, which follows at times a short period of rest. This is continued until the main attack and two or three after-attacks (Réprise, Baginsky) have forced out a tenacious plug of mucus, which frequently fails to occur until one to five minutes after a vomiting spell. During the attack, in which the child is frequently close to asphyxiation, the venous stasis causes the lips and eyelids (the latter is often present even after the attack) to swell and the face to become red and finally cyanosed. The pulse is very rapid, in many cases hemorrhages are noted from the nose and ear or into the conjunctivæ and, in rarer cases, into the brain, accompanied by the symptoms of cerebral pressure and even death. (A series of venous complications of whooping-cough is attributed by Neurath to toxic inflammation of the meninges.) The child soon recovers after the attack, and in uncomplicated cases feels perfectly well during the interval. Examination of the lungs is negative or discloses a few dry râles.

The duration of the interval is most variable. In

mild cases only about a dozen—and in severe cases several dozen—attacks occur within twenty-four hours. In the latter case the child, especially when raised under unfavorable circumstances of life, fails in general health, its sleep is disturbed by the frequent attacks, and the repeated vomiting interferes with nutrition.

After the convulsive stage has lasted two or three and sometimes eight or ten weeks, the attacks begin to become less frequent and lessen in severity. The cough gradually loses its spasmodic nature and becomes looser, and the disease passes into the terminal catarrhal stage, the duration of which depends upon external hygienic and climatic conditions. Relapses during convalescence because of neglect are quite frequent.

The **prognosis** of pertussis in small, weakly, especially rachitic children, is very dubious, because of the frequency of severe complications, such as eclampsia, capillary bronchitis, bronchopneumonia, and sometimes purulent meningitis. Danger of asphyxiation during an attack is especially likely to threaten nurslings, for in them the seizures are less noisy and frequently the crowing inspiration is replaced by a sneezing sound (at times also in older children [Hagenbach]), and not rarely decided air hunger already exists before attention is attracted to the child's condition. Older children present an obstinate catarrh of the respiratory organs, bronchiectasis, and, as a frequent sequel, tuberculosis; less rarely, otitis and nephritis. A doubtful prognosis is always made in mixed infection, as in the occurrence of measles, rötheln, scarlet fever, and diphtheria.

The **diagnosis** is easily made if a typic coughing spell is heard; in some cases it is possible to artificially excite such an attack by pressure upon the root of the tongue or on the larynx (pressure upon the vocal cords by means of the index-finger introduced through the mouth [Variot]). Of diagnostic significance is a bloated face with the presence of a doubtful cough, also the existence of ulcerations upon the frenum of the tongue, due to the wedging of that organ between the teeth during an attack.

The urine is of high specific gravity and the amount of uric acid is increased (Hippius-Blumenthal). Finally, the anamnesis of the patient is of assistance in reaching a diagnosis.

Treatment.—Provide fresh, not too cold or dry, and, above all, dust-free air. For this purpose the child should live alternately in two rooms during the febrile initial stage when rest in bed is necessary, as well as later throughout the course of the disease when the weather is unfavorable. These rooms must be constantly well ventilated and properly heated (the two-room treatment of Wertheimber). To supply the air with the proper degree of moisture employ cloth hangings which have been immersed in a carbolic acid solution; and the floor should be wiped repeatedly each day with the same solution. If the out-door air presents the necessary requirements (later a change of climate) the warmly dressed child should spend as much time outside of the house as possible. Aromatic baths (hayseed, camomile). Hardy children should receive daily baths at a temperature of 35° C. [95° F.], followed while in the bath by cold rubbing. An easily digestible diet with prohibition of dry and strongly sweetened food. In case of frequent vomiting give small portions of food in the form of gruel every half hour or every hour.

Medicaments.—Quinin, euquinin (expensive), antitussin, pertussin, bromoform, and for older children use extract of belladonna together with codein or morphin. Inhalations of oil of cypress (Soltmann) or of a 2.5 per cent. solution of carbolic acid. Administer alkaline waters. The bromids in large doses and enemata of chloral are indicated for eclampsia.

MUMPS. EPIDEMIC PAROTITIS

Mumps is an acute febrile and contagious swelling of the parotid glands and surrounding structures.

The **incubation period** lasts from one to three weeks and is symptomless.

FIG. 104.—Epidemic parotitis. Second day. The picture shows the uniform swelling in the region of the left ear, which has spread to the face and the submaxillary areas; also the characteristic elevation of the auricular lobule. The filling of the fossa between the mastoid process and the ramus of the lower jaw is unfortunately not visible. (See Fig. 107, cervical lymphadenitis.)

The **prodromal phenomena** consist of general uneasiness which in a few days leads to a local disturbance. The child experiences a painful drawing sensation in the region of the ear, and finds that chewing and swallowing are somewhat interfered with. Simultaneously with these symptoms a swelling is noted below the lobule of the ear, which rapidly spreads forward to the region of the parotid gland. Sometimes collateral edema involves the neighborhood, including the whole side of the face as far as the nose and orbits, and the neck as far as the distal end of the clavicle. The swelling causes a characteristic elevation of the auricular lobule and distortion of the face, which increases decidedly in width. In from two to four days the affection also spreads frequently to the other parotid gland, in which case the extensive swelling of both sides meet below the jaws and give the face a comical, pear-shaped appearance. The skin over the swollen portions of the gland remains pale, although occasionally it may be slightly reddened. The parotid, the sublingual, and submaxillary glands, as well as those at the angle of the jaw (which may likewise be involved), are sensitive to pressure and may be plainly felt through the swelling (which is elsewhere fairly soft and doughy) as dense nodules.

The painfulness of the inflamed glands and the pressure of the swelling upon the deep-lying soft parts represent the chief disturbances. Movement of the head is limited; troublesome swallowing and ear-ache are present. As a rule, an initial fever of 38.5° C. [100.9° F.] occurs; only rarely is the temperature higher; frequently lacunar angina and fetor of the breath coexist. The swelling begins to diminish after two or three days and disappears in about eight days. Recovery

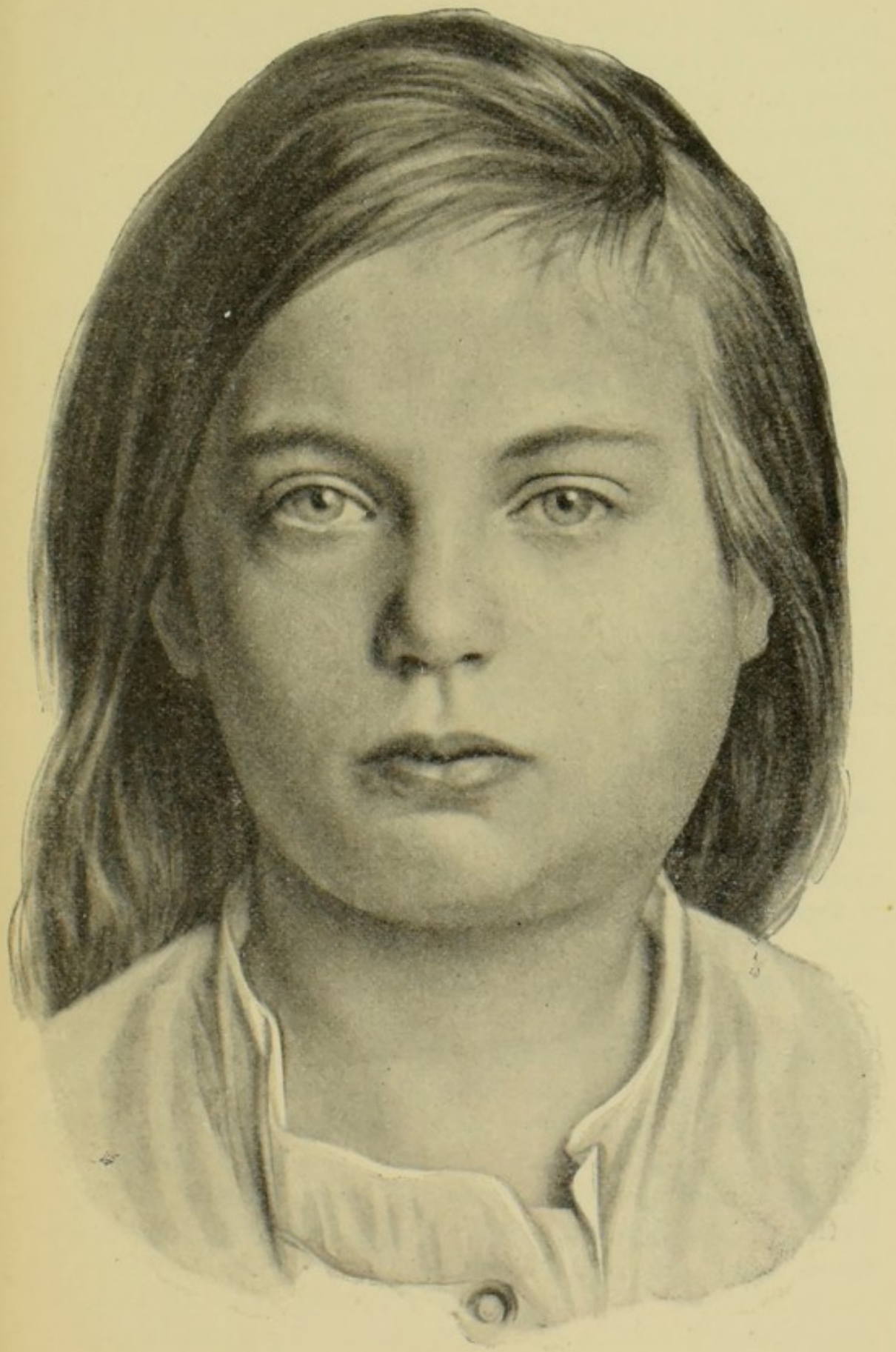


FIG. 104.

is decidedly delayed in involvement of both the parotid glands. It is worthy of note that in exceptional cases in place of involvement of the parotid the submaxillary glands are alone involved—*submaxillary mumps.*

Mumps is not rarely complicated by middle-ear disease, which may result in complete and incurable deafness; also by nephritis. Metastasis to the testes and ovaries occurs only exceptionally in children.

Diagnosis.—Parotitis must be differentiated from lymphadenitis when it exists in the region of that gland. The swelling is similar in both conditions at the beginning, excepting that in case of lymphadenitis it is not localized so exactly between the mastoid process and the angle of the lower jaw, it grows more slowly and presents, as it becomes more tense, reddening of the skin, and, finally, fluctuation if an abscess forms (suppuration is rare in epidemic parotitis). The displacement of the auricular lobule, which is so characteristic of parotitis, is also absent in lymphadenitis. Inspection and digital examination of the oral and pharyngeal cavities precludes the possibility of mistaking parotitis for the secondary edematous swelling of stomatitis, alveolar periostitis, and retropharyngeal abscess.

The **prognosis** is favorable provided no complications arise.

Treatment.—Rest in bed during the fever and confinement to the room until the swelling has completely disappeared. Cleanliness of the mouth is important, as is also a liquid or semiliquid, non-irritating diet, because of difficulties in swallowing and of the danger of nephritis. Depletion by way of the intestines. The swelling is covered with zinc powder or rice flour and protected with cotton. In case of pain resort to rubbing with heated oil of hyoscyamus, and with potassium iodid ointment or 6 per cent. iodovasogen if resorption is delayed.

DISEASES OF THE CIRCULATORY APPARATUS

GENERAL CONSIDERATIONS

DISEASES of the arteries in childhood are very rare, but, on the contrary, pathologic changes in the *heart*, especially after the fifth year of life, are quite frequent. Such alterations are mainly due to infections or to toxic influences (atheromatous processes do not occur in children). [Atheroma is found in congenital syphilis.] As causal conditions we have the acute infectious diseases and, above all, acute articular rheumatism (which is known to attack children even during the nursing period), also rheumatic conditions, which are apparently of a mild type, such as angina, for which reason the heart should always be examined in those diseases.

The *congenital anomalies* of the heart, which are not very numerous, are attributed to disturbances in development which alone affect the circulatory apparatus, or more frequently but simultaneously, also other organs (deformities of all varieties, hare-lip, situs inversus, etc.). Such anomalies may also follow fetal endocarditis (the transmission of the infectious micro-organisms from the maternal blood to the fetal circulation), which frequently causes permanent changes in the heart valves, and may be associated by a certain genetic relationship with malformations of the heart due to arrested development.

The *symptomatology* of cardiac diseases in children presents fewer characteristics than in adults. On account of the more efficient supply of blood to the heart musculature and the physiologic tachycardia we note that:

Disturbances of compensation occur more rarely and later, and that, therefore, dropsy and secondary changes

in the liver, kidneys, spleen, and lungs are only rarely observed.

Congenital as well as acquired affections of the heart may exist for a time without influencing in any way the heart dulness, so far as percussion can detect. These reasons, together with the better nourishment of the heart, probably contribute in making the prognosis of acquired heart disease during childhood more favorable than in later life (Hochsinger).

The knowledge of certain peculiarities of the infantile heart is requisite in establishing a *diagnosis* of cardiac disease in children. The apex-beat during the first or second year of life lies about 2 cm. [.8 in.] outside of the left mammillary line in the fourth interspace, and moves during the course of years to the right and downward, so that after the fourth year it is located in the fifth interspace, at first in the mammillary line, but later within that line.

The *absolute cardiac dulness* in the first year of life reaches above to the lower border of the third rib, to the left mammillary line, and to the left border of the sternum. While the outer and inner borders of the heart remain stationary, the upper edge extends at the age of four years to the upper edge of the fourth rib, and in the twelfth year to the lower edge of the fourth rib.

The *relative cardiac dulness* reaches in the first year above to the second rib, to the left somewhat beyond the apex-beat, to the right as far as the right parasternal line. The upper border moves gradually downward until the twelfth year to the third rib, and its inner border moves during the same time to the right sternal border.

In *auscultating* note that, first, up to the second year the first sound is normally accentuated everywhere; second, in easily excitable children the first 15 to 20 heart-beats at the beginning of the examination are accompanied by the so-called "cardiac-pulmonary murmur" (jerky exaggeration and weakening of the inspiratory murmur in the medial pulmonary cortex caused by

the rhythmic movements of the heart, with which they are synchronous; most evident when the heart's action is vigorous and rapid and the respiration rate is increased in frequency); and third, the so-called *accidental, inorganic cardiac murmurs*, heard in children during the first three years, are very rare; and a systolic cardiac murmur is almost a positive indication of the existence of organic heart disease, even when it represents the only demonstrable clinical heart symptom (Hochsinger).

CONGENITAL HEART DISEASE

Cardiac monstrosities, like acardia, ectopia cordis, etc., are of no interest to clinicians, and only those anomalies in which the children continue to live for a longer or shorter period of time need be considered. As a rule, these cases represent a combination of disease forms; thus, anomalies of circulatory communications show a causal relationship to narrowing of the large arterial trunks, for example, congenital pulmonic stenosis, a persisting ductus Botalli, and a defect of the septum; typic cases of single malformations are much less common. (The fetal heart offers the current of blood, which seeks to overcome an obstruction in circulation, various other paths of exit.) Indeed, depending upon the extent and nature of the lesion, it may remain unnoticed for a long time (frequently so during the first half year of life), and the patient may pass through life without any symptoms of heart disease, or, on the other hand, decided disturbances of circulation may already exist at birth.

Diagnosis.—The diagnosis between congenital and acquired cardiac defects is not always easy. A congenital anomaly is detected by—

(1) A loud systolic murmur which is heard over the whole heart with no demonstrable points of maximum intensity. (Localization by auscultation in endocarditis is probably possible.)

(2) Cyanosis in association with heart murmurs. This

does not arise in all cases, but only in those where obstruction to circulation in the left or right heart causes a damming back of the venous blood. In these affections cyanosis does not progress with the development of edema, as in acquired heart disease, nor is it always present at birth, but fails to arise until months and years

FIG. 105.

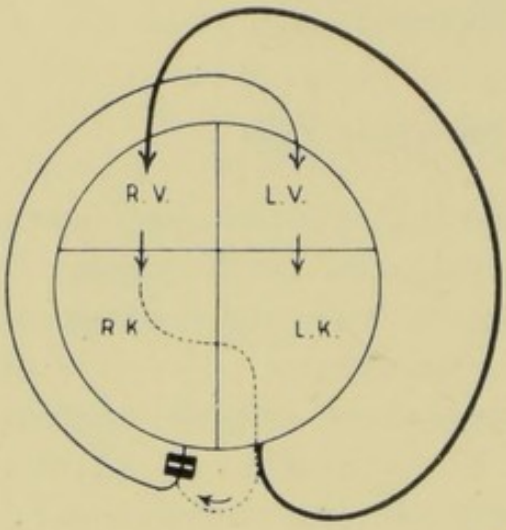


FIG. 106.

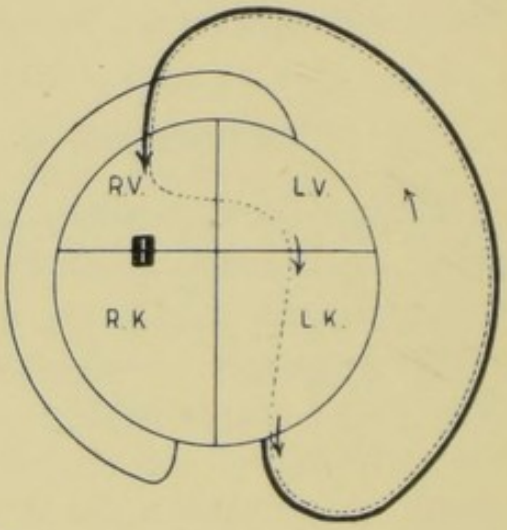


FIG. 105.

Pulmonary stenosis + Persistent ductus Botalli + Defect in septum.

Systolic murmur over pulmonic area.

Systolic murmur at the apex.

Second sound not accentuated.

Second sound is accentuated because of distention of heart with blood.

The pulmonic circulation is chiefly involved.

FIG. 106.

Insufficiency and stenosis of the tricuspid valve + Open foramen ovale.

Systolic and diastolic murmur over tricuspid area. Second pulmonic sound not loud on account of changes in pulmonic valves, stasis, cyanosis, and venous pulse.

Open foramen ovale uncombined with another lesion produces no murmur.

The systemic circulation is chiefly involved.

have elapsed, combined with attacks of asphyxiation and asthmatic disturbances. In not a few cases the cyanosis is replaced by a marked pallor of the skin.

(3) Apparent lack of relationship between the shape of the heart and the other cardiac symptoms, for example, normal area of dulness with loud, rough musical murmurs, or increased cardiac dulness with heart murmurs,

but a weak apex-beat. Furthermore, in making the diagnosis of a congenital anomaly in children, we should note the age of the child, the duration of the manifest heart symptoms, and all diseases which predispose to endocarditis (Hochsinger).

As simple as the diagnosis of congenital heart disease may be, the diagnosis of special changes is difficult and, indeed, impossible, for the manifold combinations of an anomaly may mask the action of a simultaneously existing second anomaly. Bear in mind that an abnormally weak second pulmonic sound in the presence of a clear systolic murmur indicates a congenital pulmonic stenosis; a loud systolic thrill in the region of the manubrium of the sternum and a noticeable and palpable closing of the pulmonic valves speak in favor of a patulous ductus Botalli. A defect of the septum is characterized by a very loud systolic murmur heard over the whole heart and unaccompanied by a palpable thrill (Hochsinger, de la Camp).

Treatment.—This is symptomatic. Avoid all injurious factors, bodily and mental exertion, exposure to cold and infection, and forbid alcoholic drinks. Later, cardiac remedies may be necessary.

PERICARDITIS

The most frequent forms of heart disease in children are pericarditis and endocarditis, which, not rarely, occur simultaneously and are caused by the same injurious factors. As a rule they occur secondary to other diseases, either due to direct extension from a neighboring diseased organ (lungs, pleura, peritoneum) or due to transmission through the blood of the disease factor from some other part of the body. In newborn infants endocarditis and pericarditis are frequently the partial manifestations of septic processes. In older children, tuberculosis, rheumatic affections, certain forms of angina, scarlet fever, diphtheria, and measles represent the commonest etiologic conditions.

In pericarditis a circumscribed or general inflammation of both coverings of the heart exist, accompanied by a fibrinous, serofibrinous, purulent or sanious (in septic processes), or bloody (in hemorrhagic diatheses) exudate. The clinical phenomena often develop masked by the symptoms of the causal disease. In other cases the disease picture is fulminant from the very beginning and accompanied by chills, fever (atypic), sense of fear, pain over the cardia, marked cardiac arhythmia, and dyspnea. In dry pericarditis (fibrinosa) a friction-murmur, which is exaggerated on inspiration, may be heard or felt. In exudative pericarditis the anterior thoracic wall bulges, the heart dulness is increased, and assumes the shape of a triangle with the base downward (reaching to the left always beyond the mammillary line, to the right frequently as far as the parasternal line). The friction-rub and the apex-beat which lie within the area of dulness gradually disappear, and the heart sounds become faint. The patient may recover in from two to three weeks without any sequelæ, whereas in other cases the occurrence of relapses and complications (endocarditis, pleuritis, pneumonia, peritonitis) may prolong the course over many weeks. Not rarely induration and adhesion to the thoracic wall results in dilatation and hypertrophy of the heart. At times pericarditis leads to paralysis of the heart and death.

The **diagnosis** is made from the friction-rub, the characteristic shape of the heart, displacement, and finally, disappearance of the apex-beat and the increase of the heart sounds in intensity upon rising and in bowing forward. A hectic fever and rapid decline point to a purulent exudate. Intercostal systolic retraction indicates adhesion to the thoracic wall. In hydropericardium the friction-rub is absent and dropsies exist elsewhere in the body.

Treatment.—To quiet the action of the heart absolute rest is needed (comfortable and almost sitting posture); ice-bag to the heart and infusion of digitalis internally (0.15 to 0.3 gm. : 120.0) and a mild and nutritious meat-

free diet. Later, to stimulate the secretion of sweat and urine, give caffein, sodium benzoate, or sodium salicylate (0.05 to 0.1 gm., twice daily). When the exudate becomes so great as to endanger life, resort to puncture and aspiration; in case of a purulent or serous effusion, incise and drain. [Great caution should be observed in attempts to puncture the pericardial sac. It has been shown—and verified by necropsy—that in the majority of cases of cardiac enlargement the increase in size is due to dilatation, the two layers of the pericardium are adherent. On this account the needle is easily thrust through the wall of the ventricle.—Ed.]

ENDOCARDITIS

Infantile endocarditis arises idiopathically or associated with rheumatic affections and, less rarely, in the course of scarlet fever, scarlatinal nephritis, and chorea. It usually assumes the verrucose form with the formation of fibrinous growths on the valvular apparatus of the left heart (in fetal endocarditis, the right heart). The ulcerative form, with ulceration of the endothelium and of the fibrous layer together with severe constitutional toxic symptoms, is very rare in children.

Symptoms.—Endocarditis sets in, as a rule, with a high atypic fever. The general health is considerably disturbed, the heart much excited and arhythmic, and the pulse rate may reach 180. The respiration is markedly dyspneic. In rare cases the disease begins and runs a course almost symptomless, and a systolic murmur heard loudest at the apex of the heart may represent the only clinical symptom. The changes in the heart are most variable. No murmurs are heard in endocarditis of the cardiac wall, but if deposits have formed on the valves the heart sounds lose their clearness, and blowing, jerky, and even whistling murmurs are heard. If the mitral valve is affected a loud systolic murmur best heard at the apex exists, together with a palpable systolic thrill, and also occasionally a weakened apex-beat. The heart is in-

creased in size (dilatation of the left auricle and consecutive excentric hypertrophy of the right ventricle) and the second pulmonic sound accentuated. Affection of the semilunar valves of the aorta—which occurs very rarely—is sometimes characterized by a diastolic murmur, together with a systolic murmur at the aortic area. Endocarditis in children is frequently less serious and is more likely to undergo complete and permanent cure than in the case of adults. Thus, in favorable cases, recovery may follow in two or three weeks. In a type of endocarditis, especially in young children, the process may, however, at the close of the acute stage run a more incipient and subacute course, and under the picture of essential anemia escape detection, or the disease becomes chronic, and presents a persisting systolic murmur, slight cardiac enlargement, and marked exaggeration of the second pulmonic sound (Hochsinger). In the majority of older children valvular defects, together with their consequent phenomena, remain behind, yet recovery in case of fully developed heart disease is possible in after years. The rare malignant and ulcerative forms of endocarditis run a typhoid-like course.

The **prognosis** is dubious in all cases, especially on account of the danger of fatal cerebral embolism. [The extent of inflammatory changes in the pericardium, myocardium, and endocardium, the degree of cardiac dilatation, and the subsequent effects of a ruptured compensation decide the fate of the little patients.—Ed.]

Diagnosis.—In the presence of typic local signs the diagnosis is easy. In other cases a probable diagnosis of heart disease is made from the cardiac palpitation, arhythmia, dyspnea, fever, and increased heart dulness, in the absence of other organic disease. (For differential diagnosis between this condition and Congenital Heart Disease, see the latter—of importance is the history of previous articular pain.)

Treatment.—Rest in bed, a non-irritating diet, ice-bag, and infusion of digitalis for the tension and arhythmia of the pulse. In case of rheumatic endocarditis administer

sodium salicylate, 3.0 gm. : 100.0. During convalescence pay strict attention to careful nursing and prescribe arsenic and iron. All exertion and excitement must be avoided for a long period of time.

MYOCARDITIS

Inflammation of the heart muscle is either diffuse or localized in foci due to the deposit of inflammatory products in the muscular tissue (Steffen). Diffuse myocarditis with dilatation and weakened but clear heart sounds represent occasional complications or sequelæ of diphtheria. The second form sets in at the termination of prolonged infections, fevers, in tuberculosis, and syphilis ; suppuration of the inflammatory foci predominates in septic processes. The infection occurs by contiguity from endocarditis or pericarditis or by way of the blood current. The anatomic relations are the same as in adults.

Symptoms.—The symptoms are usually severe and indefinite. Frequently we meet initial cerebral symptoms, fever, weakness, dyspnea, cardiac palpitation, rapid, irregular, and weak action of the heart, pallor, and cyanosis. Locally are observed : Dilatation of the ventricle, muffled heart sounds, and a systolic blowing murmur at the apex. If healing does not occur the gradually increasing cardiac weakness leads to stupor and death, but sometimes the latter may be sudden and unexpected.

Diagnosis.—The severe symptoms of dilatation of the heart and the weak arhythmic cardiac action make the diagnosis probable but not certain during life.

Treatment.—Absolute rest, a strengthening diet, stimulation, caffein, or sodium benzoate.

FATTY DEGENERATION OF THE HEART MUSCLE

Fatty degeneration of the infantile heart occurs only partially in the musculature of the right ventricle, runs an acute course in infectious diseases and septicemia, and a chronic course in valvular defects, protracted pneumo-

nia, and pertussis. The muscles are relaxed, slightly glossy, covered with yellowish specks, and frequently with extravasated blood.

Symptoms.—Weakness, dyspnea, cold extremities, weak, arhythmic pulse, and lessened heart sounds. With increasing dilatation and insufficiency of the valves weak blowing murmurs are heard. Death occurs while the patient is in a stuporous state or it may follow sudden collapse. Recovery is possible if the disease develops only to a mild degree.

Treatment.—The same as in Myocarditis.

DISEASES OF THE BLOOD-VESSELS

Diseases of the arteries in children are very rare. (For congenital regional (capillary) anomalies of the blood-vessels, or such as develop during the early period of life, refer to Vascular Nevi.) Dilatation of the veins in the skin are often of diagnostic importance as symptoms of obstruction in cerebral, intrathoracic, or intra-abdominal affections (hydrocephalus, enlarged bronchial or mediastinal nodes, etc.).

LYMPHADENITIS

Inflammation of the lymph-nodes is due to infection and usually follows regional disease of the skin and mucous membranes or constitutional conditions, especially tuberculosis. An acute and a chronic form are distinguished. Acute lymphadenitis consists of an inflammatory hyperemia, while chronic lymphadenitis represents a cellular hyperplasia or tuberculous infiltration of the lymph-nodes.

Clinical Symptoms of the Acute Form.—Enlargement of the lymph-nodes, which are sensitive to pressure, swelling of the surrounding tissues, and slight disturbance of the general health. Complete resolution, also suppuration, caseation, and induration may set in. Occasionally acute lymphadenitis may assume the picture of



FIG. 107.—Chronic cervical lymphadenitis. Ten year-old girl. Indolent swelling of the nodes of the left side of the neck for three and a half years. No symptoms of tuberculosis demonstrable. Iodin treatment had no noticeable effect. Ten nodes, which were mostly caseated and varying in size from a hazel-nut to a pigeon's egg, were removed by operation. The wound healed in seventeen days. In the course of two months glandular swelling returned in the region of the scar. Spontaneous rupture; healing followed painting with iodin and boric acid ointment.

an acute infectious disease with high fever, yet it runs a rapid and favorable course ("glandular fever").

In **chronic lymphadenitis** a gradual swelling of single lymph-nodes sets in unaccompanied by pain or disturbance of the general health. The resolution is equally

slow and frequently accompanied by suppuration, caseation, and induration. Chronic lymphadenitis is usually a sign of syphilis and tuberculosis, and the node is not rarely the point of origin of tuberculosis in other organs. Particularly suspicious of tuberculosis is the development of numerous small hard lymph-nodes in the occipital region (micropolyadenitis).

Treatment.—This is symptomatic. Employ iodin preparations. When tuberculosis is suspected the glands should be extirpated as soon as possible. [Favorable results are reported in the treatment of tuberculous lymph-nodes by the use of the *x*-rays. A full diet and out-door life is advisable in these cases.—ED.] (For further discussion, see Scrofula.)

DISEASES OF THE RESPIRATORY ORGANS

GENERAL DISCUSSION

THE upper air-passages—the *nose*, *mouth*, and *pharynx*—possess certain protective agencies in order to preserve the extremely sensitive mucous membrane of the true respiratory organs from injurious influences. The mucous membrane of the respiratory portion of the nose is supplied with ciliated epithelium which retains bacteria and dust and provides a bacteria-destroying secretion. The gland-like structures at the isthmus of the fauces and roof of the pharynx, the palatine and pharyngeal tonsils, may likewise be looked upon as a sort of bacteria-filter. Thus, the inhaled air is filtered before its entrance into the larynx and moistened and warmed by means of the rich blood supply to the nasal mucous membrane. Disturbances of these protective agencies lead, as a rule, to disease of the air-passages. Even breathing through the mouth, which offers but little protection when the nose is obstructed, results in irritation and inflammatory catarrh. Most marked, however, are the disturbances noted when a patient breathes through a tracheal cannula, in which case the air, without any prophylactic measures, passes with all its injurious elements directly into the trachea and bronchial tubes. Hence we must attach more importance to diseases of the upper air-passages, especially in the case of sensitive children, than we are wont.

ACUTE RHINITIS

(*Coryza; Snuffles*)

A catarrhal condition of the nasal mucous membrane, accompanied by swelling, redness, and increased secretion, is quite frequent in children and even in infants. It is always due to an infection; primarily through

various forms of bacteria and secondarily in various infectious diseases through the specific etiologic factor. In primary rhinitis, thermic, mechanical, and chemic irritants act as predisposing factors.

The affection, which is at first accompanied by a watery mucoid and, later, thick yellowish-green secretion in large quantities, causes in young children, especially in nurslings, decided constitutional disturbances, interference with nourishment and respiration (orthopnea, Henoch), and finally a high fever. There is danger of involvement of the Eustachian tube when the process extends backward to the nasopharynx. (For Acute Pharyngeal Angina, see that condition.) It is of diagnostic significance that the course of the dangerous primary nasal diphtheria is also accompanied by the symptoms of a febrile coryza.

Treatment.—Apply wet compresses, over which are drawn thick woolen compresses; hot pack to the head; increase perspiration in order to rapidly abort the condition. Insufflation of boric acid powder by means of a paper cylindric tube or a powder insufflator. (Do not blow upward.) Nasal douche: By means of a coffee-spoon or individual nasal glass pour into each nasal orifice a little cleansing fluid while the patient's head is held slightly backward. As a cleansing fluid employ: Boric acid, sodium chlorid, and glycerin, of each, 2.5 gm.: 250.0 of water. Later, paint with lukewarm almond oil. In case of orthopnea instil 1 drop of a 1 per cent. cocain solution, followed by a douche of physiologic salt solution, or the application of a menthol ointment, consisting of menthol 0.2 gm., unguentum of boric acid 35.0 gm., liquid paraffin 10.0 cc. If necessary, administer nourishment with a spoon.

CHRONIC RHINITIS AND OZENA

Chronic rhinitis develops gradually as a result of frequent recurrences of acute catarrh or on account of continued sojourn in a dusty, damp, and foul atmosphere. It may also occur as an accompaniment to hereditary

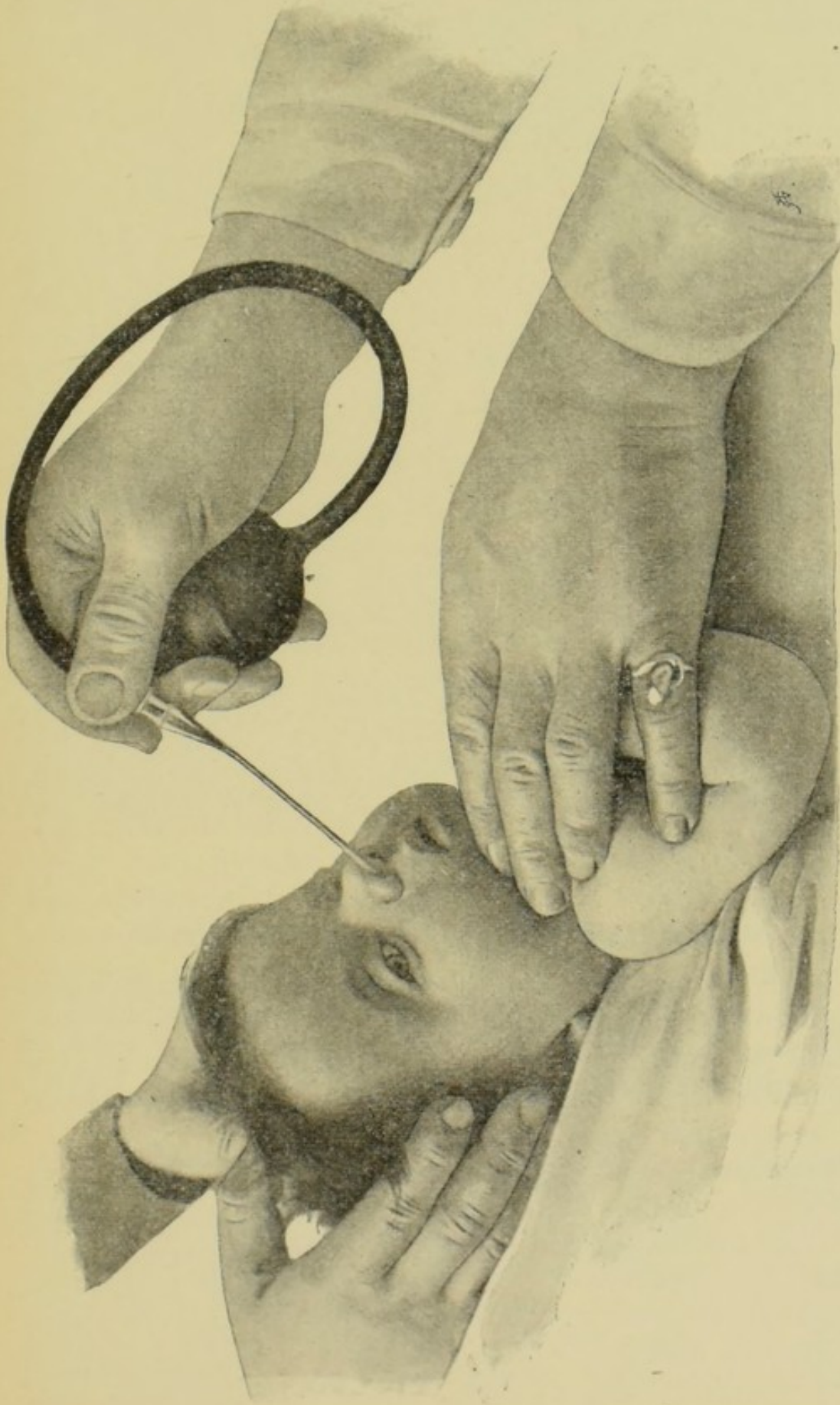


FIG. 108.—Insufflation of boric acid into the nose by means of a powder insufflator.

syphilis or tuberculosis. Diseases of the pharyngeal lymphatics, adenoid vegetations, and nasal polypi are frequently concomitant conditions.

The nasal mucous membrane is considerably reddened, swollen, and elevated in a cushion-like manner; the secretion is greenish yellow and purulent. Nasal breathing is decidedly interfered with and involvement of the Eustachian tube frequently causes difficulty in hearing. The stage of inflammatory hyperplasia is followed after a longer or shorter time by atrophy of the mucous membrane and of the nasal stroma. The pale mucous membrane of the dilated nostrils is seen to be coated with grayish-green scabs. In ozena—the origin of which, aside from association with syphilis and tuberculosis, is unknown—the atrophied mucosa presents a decidedly thickened epithelial layer (pavement instead of ciliated epithelium), the desquamated cells of which undergoing putrefaction impart to the nose the characteristic extremely foul odor.

Treatment.—When possible remove the causal condition. Nasal douches; introduction of boric acid and zinc ointment tampons. In ozena, regular and conscientious spraying with katharol and, finally, employ the yeast-cure.

ACUTE LARYNGITIS AND PSEUDOCROUP

Catarrhal inflammation of the laryngeal mucous membrane arises from the same causes as acute rhinitis (exposure to cold, specific or non-specific infection), but frequently occurs only as a sequel to a catarrhal condition of the nasopharyngeal space.

Symptoms.—The disease begins with manifestations of catarrhal irritation of the nose, conjunctiva, and with a tickling, burning sensation in the throat and a great increase in temperature. A short, dry cough and slight hoarseness soon set in. Laryngoscopic examination shows the mucous membrane of the larynx and adjacent trachea to be reddened and swollen. In severe cases the sub-

mucosa of the upper portion of the larynx may also be involved (genuine croup of measles), likewise the subchordal region (pseudocroup). Narrowing of the lumen of the larynx and the collection of secretion causes stenotic disturbances. The latter occur in *pseudocroup* in attacks and only during sleep. The child, which formerly showed only mild catarrhal symptoms, awakens suddenly during the night with a high fever, a barking cough, and hoarseness (which is never so severe as to result in aphonia), and presents all of the signs of asphyxiation, which for several minutes appear to threaten life. The attack, however, soon passes over and the child rapidly recovers; the attack may repeat itself during the same night or during the following night. Only rarely does death follow asphyxiation during an attack.

The explanation of the rapid and frequent development of threatening *laryngeal stenosis* in children is found in the peculiar anatomic construction of the child's larynx. The latter is of a very delicate and yielding structure, not only absolutely—but also relatively—smaller than in the adult, especially in the sagittal diameter. The glottis is short, the interarytenoid space is especially small, and is furthermore lined with a mucous membrane rich in blood-vessels and glands, the swelling of which may easily cause closure of the respiratory glottis. Such a swelling, because of the sensitiveness to irritation of the infantile laryngeal mucosa, occurs very frequently. It may be assumed, especially in pseudocroup, that the collection of the large quantities of tough secretion during sleep excites a reflex glottic spasm with the sudden development of stenosis.

The **differential diagnosis** offers difficulty not only during the interval, but also in an attack (see the following subject), particularly because in highly excitable children it is usually impossible to make a laryngoscopic examination. Such an examination tends only to increase the stenosis. Inspection of the pharynx offers the same picture in pseudocroup as in laryngeal diphtheria (without

FIG. 109.—Multiple papilloma of the larynx. Girl two and a half years old. Since the first year of life increasing hoarseness, otherwise healthy. Treatment for the suspected laryngitis was ineffectual. At the beginning of the second year adenotomy was performed, but without influence upon the condition. In a short time the hoarseness increased to complete aphonia. Attacks of asphyxiation occurred at times. Laryngoscopic examination was frustrated by the patient's restlessness and by the rapid development of stenosis which the operation excited. Intubation was then performed. The tube was easily introduced, but was soon coughed out. This was followed by considerable relief, the cyanosis disappeared, and a certain amount of phonation was possible. The child recovered and treatment ceased. Four weeks later death occurred suddenly at night in an attack of suffocation. Necropsy: The superior portion of the larynx was filled with a white papillomatous mass. A narrow central canal, which could have been occluded by a floating portion of a growth which was attached to the median portion of the larynx.

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pharyngeal deposits), namely, redness and swelling of the mucous membrane and thickening of the ulcerated epiglottis. We are, therefore, dependent upon the history in making this distinction which is of so much importance therapeutically. In favor of diphtheria are gradually but steadily developing catarrhal symptoms and stenosis, together with increase of hoarseness, until complete aphonia sets in. The patient recovers only partially after the attack, continues to be dyspneic, and its restless sleep is repeatedly disturbed by fresh attacks of asphyxiation. In favor of pseudocroup are the sudden and unexpected development of severe symptoms, a clearer tone to the cough, and the absence of aphonia. After the attack the patient usually falls asleep and only the symptoms of a severe laryngotracheal catarrh persist.

Treatment.—In simple laryngitis, rest in bed, Priessnitz's compresses, and hot drinks to stimulate sweating. For phlegmonous swelling, mustard and water and hot pack to encourage perspiration. During the attack of pseudocroup give hot drinks, apply a hot sponge to the neck, administer an emetic, and in case of a high-grade stenosis, resort to intubation. [During an attack of acute laryngitis, sedatives, such as sodium bromid or camphorated tincture of opium, are of great service in quieting the child, diminishing the paroxysms of cough, and lessening the local congestion.—ED.]

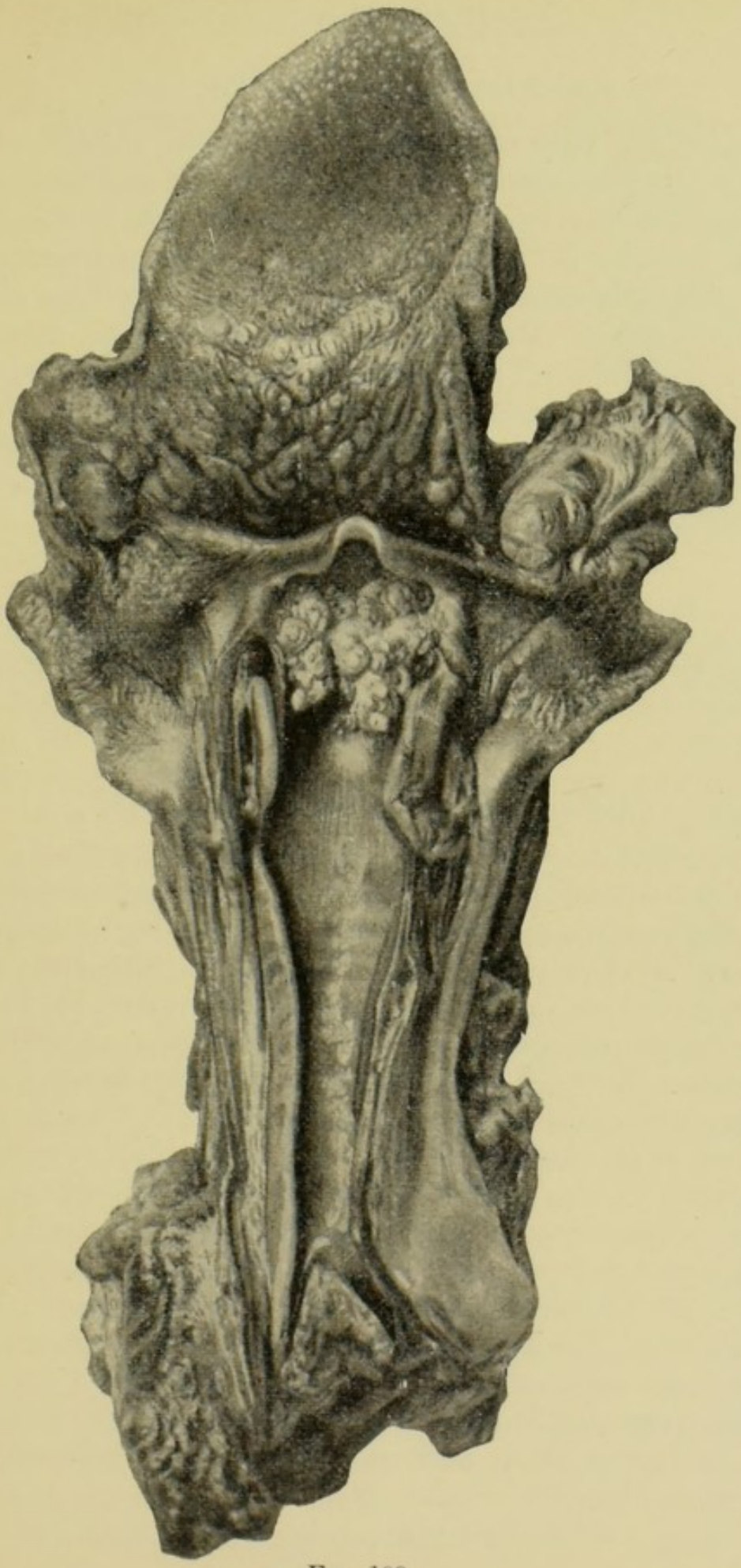


FIG. 109.

PAPILLOMA OF THE LARYNX

Papilloma represents the commonest tumor of the larynx in childhood (von Rauchfuss). It may be congenital or follow continued inflammatory affections of the larynx. According to its location, size, and number it may gradually produce a stenosis or a valve-like closure of the larynx. Permanent hoarseness arises unaccompanied by fever. The prognosis is, as a rule, unfavorable. As a therapeutic measure resort may be had to intubation with a heavy metallic tube (or curetment with O'Dwyer's fenestrated tube). A laryngotomy may be performed, the growth excised, and a tracheotomy tube inserted.

FOREIGN BODIES IN THE AIR-PASSAGES

The entrance of a foreign body into the air-passages of children is a very frequent and nearly always fatal occurrence. It may be aspirated by way of the mouth through an involuntary violent inspiration in laughing, coughing, and fright. The body then causes a mechanical obstruction to breathing, either on account of its size or on account of its sharp edges ; a stenosis indirectly, by piercing the mucous membrane and exciting a laryngeal edema. If the body enters a bronchial tube and cannot be removed by means of the bronchoscope, it leads to consecutive complications like bronchitis, pneumonia, atelectasis, abscess, gangrene, and, finally, death.

Diagnosis.—Of diagnostic significance is the sudden development of choking and dyspnea in a child previously healthy. In the course of time frequently repeated attacks of pneumonia occur at the same site, and soon symptoms arise of cavity formation, empyema, and pneumothorax. Characteristic is the rapid change of the phenomena (Fronz, Hecker). Secure a skiagram of the larynx and bronchus.

The **treatment** is operative. After removal of the foreign body the most serious pulmonary processes, even gangrene, undergo resolution and recovery.

STRUMA (see page 138).

HYPERPLASIA OF THE THYMUS GLAND

Extraordinary increase in size of the thymus gland, especially in case of true tumors of that organ (leukemia and lymphosarcoma), may lead to chronic stenosis of the trachea or bronchus (*stridor thymicus*, Hochsinger). Furthermore, hyperplasia of the thymus tends to cause laryngospasm, and is an important concomitant symptom of the constitutional anomaly called "status lymphaticus" (Paltauf, Escherich), which may lead to sudden death. Percussion reveals at both sides of the manubrium—especially to the left—an increased amount of dulness, which passes downward into the cardiac dulness.

The **diagnosis** is verified by skiagraphy.

The **treatment** consists in trying organotherapy (tablets of thymus-gland substance) or displacement of the gland by operation.

NERVOUS OR BRONCHIAL ASTHMA

A pure essential bronchial asthma, which is a reflex neurosis accompanied by spasmodic attacks of dyspnea (spastic contracture of the bronchial musculature), occurs with the well-known symptoms in children of all ages. Hereditary influence plays an important rôle. It is frequently also caused by nasopharyngeal disease (Baginsky), especially adenoid growths (nasal asthma, the usual exciting cause is an acute catarrh) or indigestion (dyspeptic asthma). During the first two years of life asthma (cardiac) is frequently at first superimposed upon a congenital defect of the heart. Eczematous patients are not rarely subject in later life to asthma (Feer).

Treatment.—As prophylaxis avoid exposure to cold and judicious hardening of the body. Change of scene; sojourn at the sea or at mountain resorts of moderate height. Operative procedures for nasal asthma; emetics for dyspeptic asthma. During and after the attack give every two hours a coffeespoonful or a child's spoonful of

sodium bicarbonate or potassium iodid, of each 2.0 gm. to 100.0 cc. (Neumann). Hot pack to stimulate sweating. Vapor inhalations; emetics. During the intervals give arsenic.

ACUTE TRACHEITIS AND BRONCHITIS

Acute tracheitis and bronchitis arise from the same causes as acute laryngitis, and frequently at the termination of a catarrhal disease of the upper air-passages.

Morbid Anatomy.—The mucous membrane of the trachea and the large bronchial tubes is reddened, swollen, relaxed, and after long duration of the disease becomes pale gray and atrophied. The secretion covering the mucous membrane is generally tenacious, glossy, and contains air-bubbles, but at a later stage it becomes thicker, mucopurulent, yellow, or greenish yellow.

Symptoms.—The disease begins with a dry, painful, and spasmodic cough, increased respiration rate, fever, loss of spirit and appetite. Young children usually swallow the excretion; the expectoration of older children shows the above-described characteristics. Palpation reveals râles over the whole thorax. Percussion is negative. On auscultation at the beginning, when the mucous membrane is simply swollen, we hear dry râles, but as the serous secretion increases, moist large and small vesicular râles are heard. The intensity of the tone of the râles depends upon the distance between the disease focus and the body surface. The respiratory murmur is vesicular, accentuated, and sometimes interrupted by the noise of the râles. In catarrh of the bronchi, both lungs, especially the lower lobes, are involved. After several days' duration the fever falls by lysis, all symptoms lessen in severity, and the cough becomes looser and disappears gradually in one or two weeks. In weak children living in an unhealthy atmosphere and in neglect of the acute symptoms the affection becomes chronic and lays the foundation for tuberculosis of the bronchial nodes. The prognosis is, therefore,

dubious under the conditions mentioned. (For differentiation from Pertussis, see that disease.)

Treatment.—Attempt to abort the disease during the development by hot packs or baths, in order to increase perspiration. Provide fresh air, keep the body warm, and rest in bed in case of fever. Priessnitz's compresses; hot drinks to excite expectoration and perspiration. Moisten the inhaled air by means of an inhaler or croup

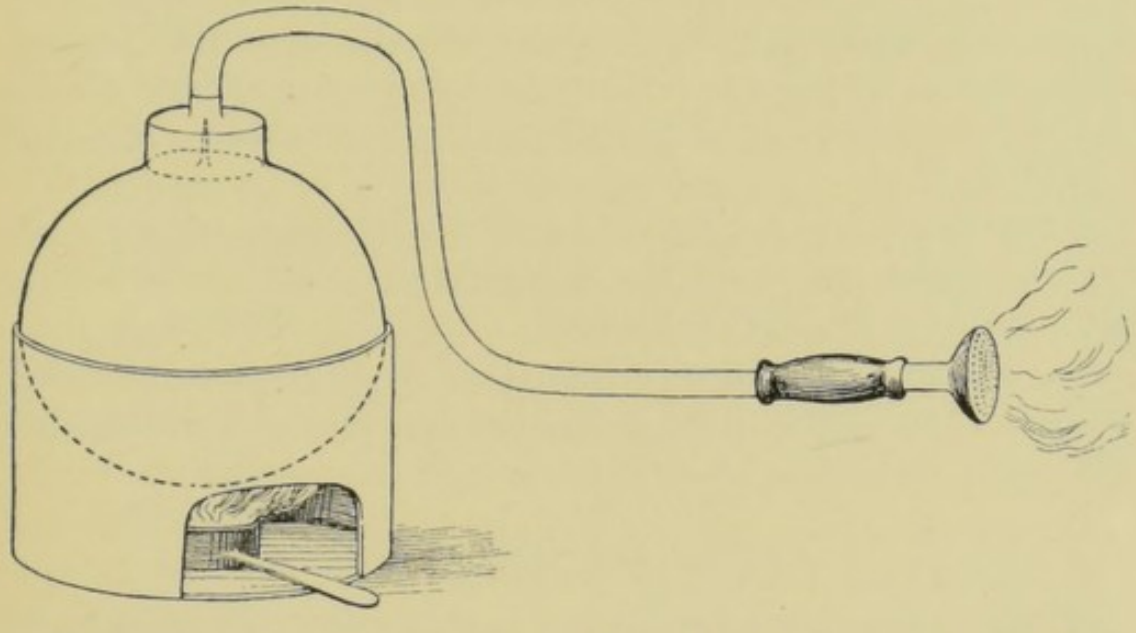


FIG. 110.—Vapor apparatus for moistening the air to be inhaled in diseases of the respiratory tract. The kettle is filled with 1 liter [1 qt.] of boiling water. Camomile and other preparations are placed in the upper portion of the apparatus. The current of steam which travels for about 1½ meters [4½ ft.] is directed against the face of the patient. To obtain a more pronounced action (especially in croup or pseudocroup) a primitive steam room may be arranged by spreading linen sheets over the bed.

kettles. Internally give a coffeespoonful of the infusion of ipecacuanha, 0.3 gm. to 120.0–150.0 cc., every two hours. To this may be added the extract of belladonna, codein, or aqua amygdalæ amaræ, in order to lessen the irritation of the cough, and liquor amm. anis. or (according to Fischl) potassium iodid, 0.3 to 1.5 gm., to stimulate expectoration. In very young children administer a child's spoonful, every two hours, of syrup of ipecacuanha with syrup of senega or althea.

CHRONIC BRONCHITIS

In chronic bronchitis the persistent cough is looser and of a catarrhal character; the secretion is sometimes more profuse, more frequently, however, it is less in quantity, while the sputum is grayish yellow and appears in lumps. On palpation mucous râles are felt, and on auscultation vesicular breathing and coarse râles are heard. The respiration rate is not increased. The affection, provided it is not of a tuberculous nature, is not accompanied by fever or marked disturbances of the general health, although in some cases asthmatic symptoms arise. (With reference to the development of Bronchiectasis, see that condition.)

Treatment.—Provide as hygienic a life as possible. Sojourn at the sea or in the mountains. A non-irritating and, preferably, vegetable diet. If the secretion is profuse, inhale oil of turpentine; if it is scanty, inhale salt water. Internally give potassium iodid and alkaline and muriatic waters.

CAPILLARY BRONCHITIS

If the inflammatory process spreads from the large and moderate-sized bronchial tubes to the smaller and minute bronchi, we have a capillary bronchitis, the most dangerous disease of the respiratory passages. When this region is once involved, the process extends rapidly to a large section of the bronchial tree.

Morbid Anatomy.—The mucous membrane of the bronchial tubes—even in the smallest branches—is intensely red, swollen, and covered with a tenacious, glossy, and (later) mucopurulent secretion. The bronchioles are in some areas completely obstructed by the swelling and secretion, and not rarely the associated alveolar portion collapses after the absorption of the air and becomes atelectatic. The atelectatic lobules are bluish red, relaxed, hyperemic, and diminished in volume. On section, slight pressure will cause a large amount of pus to ooze from the medium- and smallest-sized bronchial tubules.

The disease picture is a very severe one. The inflammatory swelling of the mucous membrane tends to cause a dangerous stenosis in the infantile bronchi, for the narrow bronchial lumen easily becomes obstructed by the tenacious secretion. The direct result is defective aëration of the lungs, that is, insufficient entrance of oxygen and discharge of carbon dioxid. The indirect results are an increased activity and a decrease of the heart's ability to work.

Symptoms.—Clinically these changes make a distinct impression. When very extensive they begin with high fever, dyspnea, and cyanosis. The respiration is superficial, irregular, and rapid; in the case of nursing infants it is increased to 60 and 100 per minute. On inspiration the ribs are retracted. The alæ nasi move with the respiration. The expiration is prolonged, accentuated, and sighing. The cough is frequent, short, painful, and therefore suppressed as much as possible. Great restlessness and symptoms of indigestion. The skin is pale and the mucous membrane slightly cyanosed. The pulse is small and has a rate of 120 to 180. The temperature averages between 39° and 39.5° C. [102.2° and 102.7° F.], it is irregular, remittent, and frequently increases with extension of the process. Examination of the lungs gives the same results in capillary bronchitis as in acute bronchitis, only we find in several areas, especially at the bases posteriorly, fine vesicular râles which frequently drown the puerile vesicular breathing, and are only distinguished from the crepitant râles of pneumonia by the fact that they are also audible on expiration. The respiratory murmur is often completely absent over portions of the lungs where the bronchi are obstructed. With gradual disappearance of all symptoms the patient may completely recover in the course of a week. It frequently causes death (especially in rachitic children) or it may lead to atelectasis and pneumonia.

The **prognosis** is always doubtful, especially in weak and rachitic children or in those predisposed to tuberculosis. It is unfavorable when the process extends sud-

denly to the whole bronchial tree. In many instances the inflammation involves the pulmonary tissue also.

Diagnosis.—The passing of a bronchitis into the capillary type is marked by the sudden development of high fever, increased irritation of the cough, shortened respirations, and a sighing expiration. After the preliminary symptoms only a few febrile catarrhal manifestations exist. Capillary bronchitis is distinguished from pneumonia by the absence of bronchial breathing, bronchophony, and dulness. The differentiation may, however, be impossible when the pneumonia is not extensive and it is impossible to determine the presence of pneumonic consolidation by physical signs.

The **treatment** is that of bronchopneumonia.

BRONCHOPNEUMONIA

(*Lobular Pneumonia. The Pneumonia of Children*)

If the inflammatory process spreads to the pulmonary tissue the alveoli become filled with inflammatory products (serous or serofibrinous exudate, pus-corpuscles, and desquamated alveolar epithelium). The involved portion of the lungs is thus congested and unable to perform its function, and we have the condition known as bronchopneumonia. The inflammation travels longitudinally along the bronchial tubes to the alveoli or it penetrates the bronchial wall and attacks the peribronchial tissue. In both cases we note circumscribed, either lobular or peribronchial foci, which coalesce and, enlarging, finally involve a whole lobe, the so-called "pseudolobar form."

This type of pulmonary disease is characterized by the fact that it originates in a disease of the bronchial tree. The direct cause of the condition may be of a specific or non-specific nature. To the first class belong the microorganisms of measles, whooping-cough, diphtheria, and influenza, and to the second class the various pneumococcic bacteria (bacillus of Friedländer, Fränkel-Weichselbaum diplococcus) and pus cocci. Occasionally the pneumonia follows the aspiration of particles of food or

mucus by children when in a stuporous state or when a tracheotomy has been performed—*foreign-body* or *aspiration pneumonia*.

Morbid Anatomy.—The diseased lung tissue is already recognizable macroscopically by its increased volume, dark discoloration, and its hard, nodular consistency. The pleura in the region of the disease process is at times covered with a thin fibrinous deposit and isolated hemorrhages. Section shows a varied picture; side by side are seen pale normal air-containing lung tissue and brownish-red, prominent, airless, and dense inflammatory foci of varying size, the centers of which are sometimes faded; aside from which we always note bluish-red, soft, airless, and somewhat retracted atelectatic areas. The inflamed foci show a smooth surface which—when considerable fibrin exists—is granulated. Pus can be squeezed out of the inflamed bronchi and a turbid yellowish fluid from the diseased portions of the lungs. The air-content is decidedly diminished. In peribronchitis the bronchial wall is thickened. The pulmonary cortex is frequently emphysematous and atelectatic. Prolongation of the disease leads to cylindric ectasia of the smallest bronchi, hyperplasia of the bronchial nodes, fatty degeneration of the heart muscle, and dilatation of the right heart. Microscopically we find round-cell infiltration and pronounced congestion of the alveolar borders, and, later, of the peribronchial tissue also. The alveoli are filled with a mass of cells which have undergone partial fatty degeneration and an inflammatory exudate, which sometimes contains but a small amount of fibrin and at other times a large quantity. This peculiarity of the bronchopneumonic exudate, which is only rarely of a purely catarrhal nature, is characteristic of the pneumonia of children, and is also noticeable in the clinical course of the disease. Noteworthy is the frequent occurrence of giant cells in the pneumonic infiltrated alveoli in diphtheria and measles. (Concerning characteristic changes in the Pneumonia of Whooping-cough, see that disease.)

PLATE 34

FIG. 1. **Confluent Bronchopneumonia in a Child Two Years Old.**—The inflammation has existed for several weeks. Enlarged 52 times. The microscopic picture of a fully consolidated pulmonary lobe offers a uniform appearance. The alveoli are filled with an exudate which consists of fibrin (retracted on account of the hardening process), degenerated alveolar epithelium which stains with difficulty, and a few leukocytes, while in some areas only pus-corpuscles are present. 1. Exudate composed of fibrin, degenerated epithelium, and a few leukocytes. 2. Purulent exudate. 3. Alveolar borders showing round-cell infiltration.

FIG. 2. **Bronchitis and Beginning Bronchopneumonia in a Child One Year Old.**—Died of enteritis. Enlarged 52 times. The picture shows the extension of the bronchopneumonia from a bronchitis. The inflammatory process, which was primarily confined to the bronchial mucous membrane, has penetrated the whole bronchial wall and caused infiltration of the peribronchial tissue. The manner in which two peribronchitic foci have become confluent may be seen. The freshly infiltrated tissue is hyperemic. 1. Lumen of a bronchus. 2. Desquamated bronchial epithelium. 3. Catarrhal bronchitic exudate. 4. Bronchitic exudate with a large amount of exfoliated bronchial epithelium. 5. Beginning bronchopneumonic exudate. 6. Confluent infiltrate. 7. Dilated blood-vessels. 8. Normal lung tissue.

Symptoms. — Bronchopneumonia can often be distinguished clinically from capillary bronchitis only by the changes in percussion. As soon as the congested foci have, through confluence, reached a certain extent, provided they are not too deeply seated, they may most frequently be demonstrated by percussion in the region of the axilla and parallel to the spine. Auscultation discloses, aside from loud catarrhal and at times bronchial murmurs, fine vesicular râles, and, if the afferent bronchial tubes are not obstructed, bronchial breathing and bronchophony. Vocal fremitus is increased when the process is extensive. The course of the disease and the general and local symptoms vary from case to case, according to the extent of the anatomic process and the character of the exudate. When large pneumonic areas become confluent and a cellular and fibrinous exudate occurs, the symptoms resemble those of fibrinous and croupous pneumonia, so that it is impossible to distinguish between these two forms. On account of frequent relapses the intensity of the symptoms may also vary considerably in individual cases.

The disease frequently runs a **course** of many weeks,



Fig. 1.

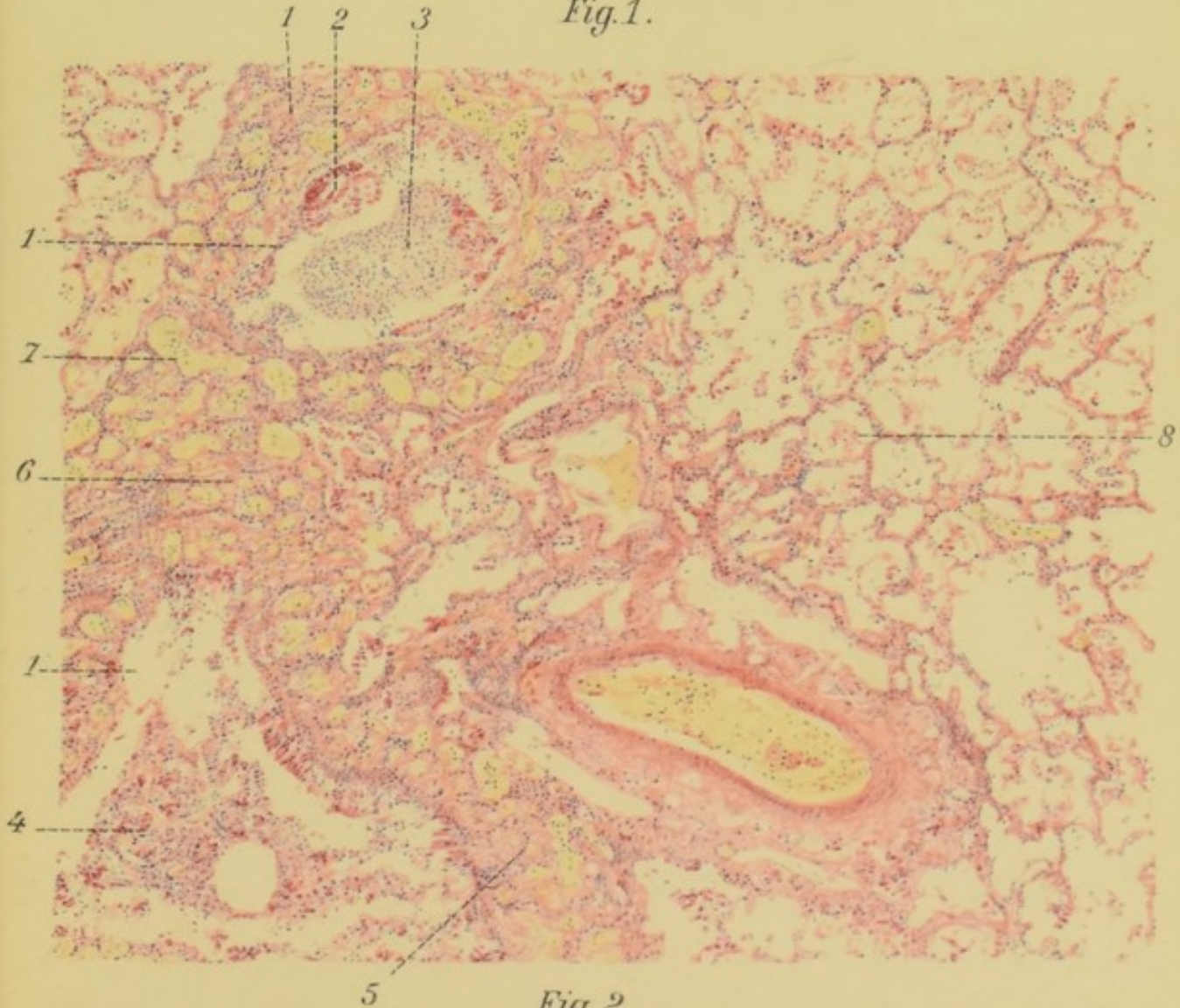


Fig. 2.

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although in favorable cases it may end in one or two. Resolution sets in, with the gradual disappearance of the dulness, fall of the fever by lysis, and improvement of all remaining symptoms and the general condition. Death may follow from weakness or from carbonic-acid-poisoning. Lobular pneumonia passes into the chronic form, not rarely by caseation of the alveolar contents or inflammation of the interstitial tissue. It is quite often primarily of a tuberculous character or it terminates in a miliary tuberculosis. Frequent complications are pleurisy, gastritis, and otitis media.

Diagnosis.—The lack of physical signs of changes in the pulmonary parenchyma at the beginning and the prevalence of some causal disease frequently leads to a mistaken diagnosis. In favor of bronchopneumonia are:

Sudden and marked rise in temperature during the course of a bronchitis or the persistence of a high fever beyond the usual febrile period of a causal disease (measles).

Lessening and painfulness of a formerly vigorous cough. Difficult and rapid breathing. Activity of the accsssory muscles of respiration. Movement of the alæ nasi in breathing.

Physical signs of pulmonary consolidation.

Occurrence on both sides. In favorable cases resolution is gradual, the fever falls by lysis, without regard to critical days and slow convalescence.

In atelectasis the percussion-note is not so dull and is usually accompanied by a tympanitic note. Bronchial breathing and bronchophony are absent, only subcrepitant inspiratory râles are audible, which disappear with deep respiration. In croupous pneumonia the disease is a primary condition which begins on one side; the catarrhal râles are absent, the consolidation involves part of a lobe, the fever is higher and ends nearly always by crisis. In pleurisy the dulness is more resistant, of a characteristic form and extent. The vocal fremitus is lessened. The anamnesis gives valuable information in caseating tuberculous pneumonia. The loss of strength

is disproportionately rapid. The course is suspiciously long. Symptoms of cavity formation and the development of tuberculous affections in other organs frequently arise. The prognosis in weak, rachitic, or scrofulous children is always doubtful. The disease is far more dangerous than croupous pneumonia and is more frequently followed by permanent changes in the respiratory tract.

Treatment.—Treat the original causal disease. Observance of general dietetic and hygienic principles as in acute bronchitis. Frequent change of position in the bed to avoid hypostasis (infants should be carried about at intervals). At the beginning of the disease relieve the bronchi by an emetic. Give for this purpose powdered ipecac, 0.5 to 2.5 gm., with syrup of althea, 40.0 cc., of which a coffeespoonful is given every ten minutes until effect is produced. Later, for thermic stimulation of the nerves, to combat the fever, to deepen the respiration, and to excite expectoration, resort to hydropathic measures: Baths at a temperature of 25° to 35° C. [77°–95° F.], varying with the fever and the patient's strength, followed by short cold douches. (If influenza, pertussis, or rachitis are simultaneously present, the body heat must not be lowered too vigorously.) In case of subnormal temperature or difficult breathing on account of carbonic-oxid intoxication, subject the occiput and neck to a stream of water as cold as possible. As many as ten streams following each other may be applied, at intervals of from ten to twenty seconds, and each stream should measure about 1 cm. [.4 in.] in diameter (Jürgensen). In place of baths employ cold and moist packing, leaving the head, arms, and legs exposed; to increase the radiation of heat renew the pack every ten minutes. If the condition becomes worse and the surface of the body cold, use mustard pack. The internal medication consists of enemata of quinin (0.3 gm. to 20.0 cc.); infusion of ipecac, 0.3 gm. to 100.0 cc.; together with liquor ammonii anisatus, 1 to 1.5 gm., or, as stimulants, triturates of camphor and benzoic acid, of each 0.015 gm.,

every one or two hours. For delayed resolution administer a child's spoonful of potassium iodid (2 to 3 gm. to 100 cc.) every two hours; oxygen for dyspnea. Champagne, camphor, or injection of ether for threatening heart failure. Venesection for threatening asphyxiation.

CROUPOUS PNEUMONIA

(*Fibrinous Pneumonia; Lobar Pneumonia*)

Aside from bronchopneumonia with its atypic exudate we also find fairly frequent in children, especially during the first five years, croupous pneumonia, which begins with a high fever, runs an acute course, and is accompanied by a purely fibrinous and slightly cellular exudate. The micro-organisms mentioned in the last disease are the etiologic factors. The irritation caused by these bacteria involves directly and at one time a large surface of the alveolar epithelium, so that, unlike bronchopneumonia, instead of the small inflammatory foci which gradually spread, a large area, usually a whole lobe, is involved at once. The disease is more prevalent in cold and wet weather, and is brought on by those factors which lessen the resisting forces of the organism, as circulatory disturbances. Croupous pneumonia of children presents no special etiologic, anatomic, or clinical differences from the same disease in adults. The previously described micro-organisms are present; anatomically the stages of inflammatory engorgement exist, that is, hepatization and purulent infiltration, also a localized fibrinous pleurisy. The clinical symptoms consist of a sudden onset, with vomiting, chilly sensations, rarely convulsions; a high, continuous fever, dyspnea with pain in the side, cough, and a rust-colored sputum. Percussion reveals slight dulness. A tympanitic sound is elicited on deeper percussion over a whole lobe or a large portion of it. On auscultation crepitant râles, exaggerated or weakened, and indefinite breathing are heard. Later there is pronounced dulness, bronchial breathing, bronchophony, and increased vocal fremitus. In favor-

able cases resolution is accompanied by disappearance of the symptoms in the same order as in their development. The fever disappears by crisis—rarely by lysis—on the fifth to the ninth day (exceptionally earlier or later), usually with continued severe perspiration. Characteristic of croupous pneumonia in childhood is the beginning in young children with vomiting, convulsions, and a slight chilliness, instead of the marked chills of adults; also a pulse and respiration rate corresponding to the height of the fever, the development of symptoms of cerebral irritation, and a more rapid convalescence than in adults. When lobar pneumonia occurs at the close of an acute infection, absence of reaction on the part of the organism interferes with the typic course, and the exudate fails to undergo the usual resolution. This type is more likely to be protracted and terminates frequently in death or in caseation or connective-tissue new growth (Ziemssen). The primary form of pneumonia in young children may also offer diagnostic difficulties. The subjective symptoms are absent, usually the characteristic sputum also, and, as mentioned above, the initial chill. Cough, shortness of breath, and pain in the side are frequently pronounced. The diagnosis is especially difficult when the pneumonia is of central origin. For example, in pneumonia of the upper lobes, in which case the physical signs usually do not develop until the fourth or fifth day, and the presence of cerebral manifestations may lead to suspicion of a cerebral or meningeal affection. The following facts are of assistance in making the diagnosis of croupous pneumonia: Leukocytosis, acetonuria, diaceturia, herpes labialis (less common in children than in adults), and disappearance of the patellar tendon reflex (not until after the third year of life—Pfaundler).

Differential Diagnosis.—In capillary bronchitis, atelectasis, and bronchopneumonia the child is pale and cyanotic, whereas in croupous pneumonia the initial stage is accompanied by marked redness of the cheeks. In the first disease the pulse is small and soft, but in croupous pneumonia, full and hard. Bronchopneumonia progresses

with the gradual development of catarrhal symptoms, the areas of dulness are smaller, and the temperature is normal or of a moderate height. (For the characteristics of Caseous Pneumonia, see that disease.) The dyspnea is less, the temperature rarely rises as high as in croupous pneumonia, and shows an irregular morning rise, or the *typhus inversus*. Croupous pneumonia accompanied by cerebral symptoms is distinguished from meningitis by the mildness and lack of constancy of the nervous manifestations, the regular and rapid pulse, the simultaneous development of symptoms of a pulmonary disease, and, finally, the loss of patellar reflex (which is increased in meningitis). The temperature of meningitis does not show the influence of critical days.

Prognosis.—Croupous pneumonia, as a rule, runs a favorable course in children previously healthy and strong and who have been brought up under favorable conditions of life. Death may follow pulmonary edema when the inflammation is very extensive, or a complicating pleurisy, pericarditis, or meningitis (otitis, nephritis). Tuberculous infection of the exudate in weak scrofulous individuals may also cause death.

Treatment.—General hygienic and dietetic measures as in bronchitis and bronchopneumonia. Of especial importance is a strengthening liquid diet. Cleanliness of the mouth. Regular bowel movements. Cold pack; baths, with cool douches and subsequent vigorous rubbing (omit all hydrotherapeutic procedures at the time of crisis). In case of heart failure and somnolence give camphor, ether, or champagne. For extreme dyspnea, inhalations of oxygen. Employ the ice-bag for severe nervous phenomena. In case of delayed resolution, moist warm pack and iodin preparations.

CHRONIC PNEUMONIA

(*Bronchiectasis*)

The passing of a pneumonia into the chronic stage (bronchiectasis) occurs more frequently in catarrhal than in croupous pneumonia. It is marked by inspissation of

the alveolar contents and proliferation of the interstitial connective tissue with consecutive contraction of the parenchyma. The child is anemic, emaciated, and sallow; every exertion is accompanied by shortness of breath and cough is constant. A remittent or intermittent fever with afebrile intervals exists; also dyspepsia and profuse sweats. *Locally:* Dulness, indefinite or bronchial breathing, and râles; mucopurulent expectoration. The affected side of the thorax is contracted when the lung shrinks.

Bronchiectasis may not only follow pulmonary contraction, but may also be due to continued increased inspiratory or expiratory pressure. The latter occurs when the inflammatory process in chronic purulent bronchitis penetrates the bronchial wall and, involving the surrounding tissue, causes the bronchial wall to become gradually thin, soft, yielding, and its elasticity damaged. This is especially likely to develop in the course of the pneumonia developing in diphtheria, whooping-cough, and measles. A severe, troublesome, and spasmodic cough, which arises chiefly in the morning and evening, brings up a thin, purulent, greenish expectoration which has a foul odor; this discharge not rarely gushes forth from the nose and mouth and forms layers in the vessel in which it is collected. Characteristic of this condition are the changes in percussion and auscultation as the cavity-like dilated bronchus becomes filled. The symptoms of a cavity are presented when a large expansion of the bronchus occurs superficially. A cure is impossible in indurated interstitial pneumonia and bronchiectasis. The resorption of an inspissated exudate may, however, occur after a period of weeks, provided caseation or tuberculosis do not develop.

The **treatment** of this chronic disease of the lungs must preferably be hygienic and dietetic. Breathe air which is free from dust; sojourn at the sea, mountains, or winter resorts. Woolen underclothing. A strengthening diet which is rich in fats. Cod-liver oil. Tepid baths. Priessnitz's compresses. Turpentine inhalations for bronchiectasis.

PLEURISY

Pleurisy occurs quite frequently in children, especially at the middle period of childhood, and, as a rule, secondary to or as an associated manifestation of disease elsewhere in the lungs or to a constitutional condition. In exceptional cases pleurisy is primary in origin, due to cold or trauma. Pleurisy develops as in adults in the dry, fibrinous, or exudative form ; the latter type is designated according to the character of the exudate.

Serous or **serofibrinous pleurisy** is accompanied by the excretion of a clear, yellowish fluid, which is poor in cellular elements and chiefly composed of serum containing more or less fibrin.

Purulent pleurisy, or **empyema**, is associated with an abundant cellular purulent exudate, which may become decomposed by the entrance of putrefactive bacteria and give forth a fetid odor (ichorous empyema).

Hemorrhagic pleurisy is characterized by a serous fluid which is colored reddish or brownish red by the admixture of red blood-cells. The development of a pleurisy is due, on the one hand, to the entrance of large masses of pathogenic bacteria or their virus, and on the other, to alterations in the pleura through disease processes of neighboring organs, trauma, exposure to cold, diseases of the blood, and disturbances of circulation. The entrance of bacteria or their toxins follows either from the blood or lymph-vessels or directly from the diseased neighboring organs. In the first case the pleurisy is an expression of a constitutional infection ; in the second, it is usually the result of pulmonary diseases. Pleurisy is also frequently associated with an acute infectious disease, in which case a specific infection is not always at fault, for it is more likely that the disease produces only a predisposition of the pleura to secondary involvement. A by no means small percentage of pleurisies are of a tuberculous nature. In pneumococcus pleurisy we find this organism most frequently, whereas in pleurisy following an acute infectious disease, streptococci and staphylococci are most likely to be found.

Symptoms.—The disease often begins very gradually, with insignificant symptoms which are barely noticeable and consist of a slight cough, mild dyspnea, and a moderate increase of fever toward evening. In other cases it has a sudden onset, with headache, vomiting, chills, high fever, short, suppressed, very rapid breathing, and pain in the side, which is made worse by respiration and motion. The affected half of the chest is usually contracted in children and shows diminished respiratory excursion. Percussion is negative; auscultation reveals weakened breath sounds and sometimes circumscribed friction-rub at the height of inspiration—*dry pleurisy.*

If an exudate develops in a few days the findings change considerably. The friction-rub gradually disappears (until the beginning of absorption), the whole diseased side bulges, shows obliterated intercostal spaces, and shares only slightly or not at all the movements of the rest of the chest. Breathing is dyspneic and painful. To relieve the pain the patient lies on the diseased side. As soon as the exudate measures 60 ccm. or more we obtain dulness over the affected area, yet the dulness is rarely as absolute as in adults, for in the infantile thorax percussion also elicits the tone of the air-containing portions. If the collection of fluid is large, dislocation of the heart or liver occurs, and if it is on the left side, the Traube's space is obliterated. Palpation shows tenderness to pressure and a sense of resistance in the dull area; vocal fremitus is diminished. On auscultation diminished bronchial breathing is heard, as well as bronchophony, which is even audible over the compressed lung, since the dimensions of the infantile thorax are too small to interfere with the conduction of consonant tones (Ziemssen). When pleurisy occurs during the course of a pneumonia or as a sequel, the high temperature of the pneumonia persists and bronchial breathing and bronchophony are louder, the dulness almost absolute, and the pectoral fremitus preserved. In empyema we note collateral edema of the soft tissues lying over the suppurative focus, a high septic fever, and decided disturbance of

the general health. If the exudate exists in large quantities the health is rapidly undermined and the symptoms of carbonic-acid-poisoning develop; the physical symptoms of a serous exudate are present.

The **course** and **result** of pleurisy is dependent upon the etiologic factors, the age, and the strength of the patient. Dry pleurisy and serofibrinous pleurisy with slight exudate may heal in one week, but if the excretion is abundant the course may be prolonged over weeks and months. Cases accompanied by the rapid development of a large amount of exudate are always doubtful as regards the prognosis, especially in young children. Empyema may rupture spontaneously—externally or into the compressed lung. Pleurisy is frequently followed by induration, contraction, deformity of the thorax, and scoliosis. Serious complications and sequelæ include consecutive inflammation of neighboring organs, tuberculosis, chronic bronchial catarrh, and bronchiectasis. The continuance of the fever after an operation for empyema should lead one to suspect other purulent foci (Vierordt).

The **diagnosis** may remain uncertain for a long time. Constant manifestations of an exudative pleurisy are a persistent tendency to lie on the same side, cough, pain in the side, dyspneic breathing without accentuation of expiration (pneumonia), a resistant dulness, and bronchial respiration. On the other hand, pectoral fremitus, which is so important in reaching a decision, is under all circumstances demonstrable with difficulty in children; sputum is rarely ever procurable; the percussion-note is absolutely dull only when a very large exudate or a pneumococcus pleurisy is present, and the respiratory murmur and the voice do not show the characteristic weakness seen in adults. Friction-rub may be absent. Thus, no conclusion may be reached until later in the course of the disease or by means of an exploratory puncture. (The fluid thus removed should always be examined microscopically, and if it is found to contain only a few bacteria and mononuclear leukocytes are present, the exudate is probably tuberculous, whereas the presence of numerous

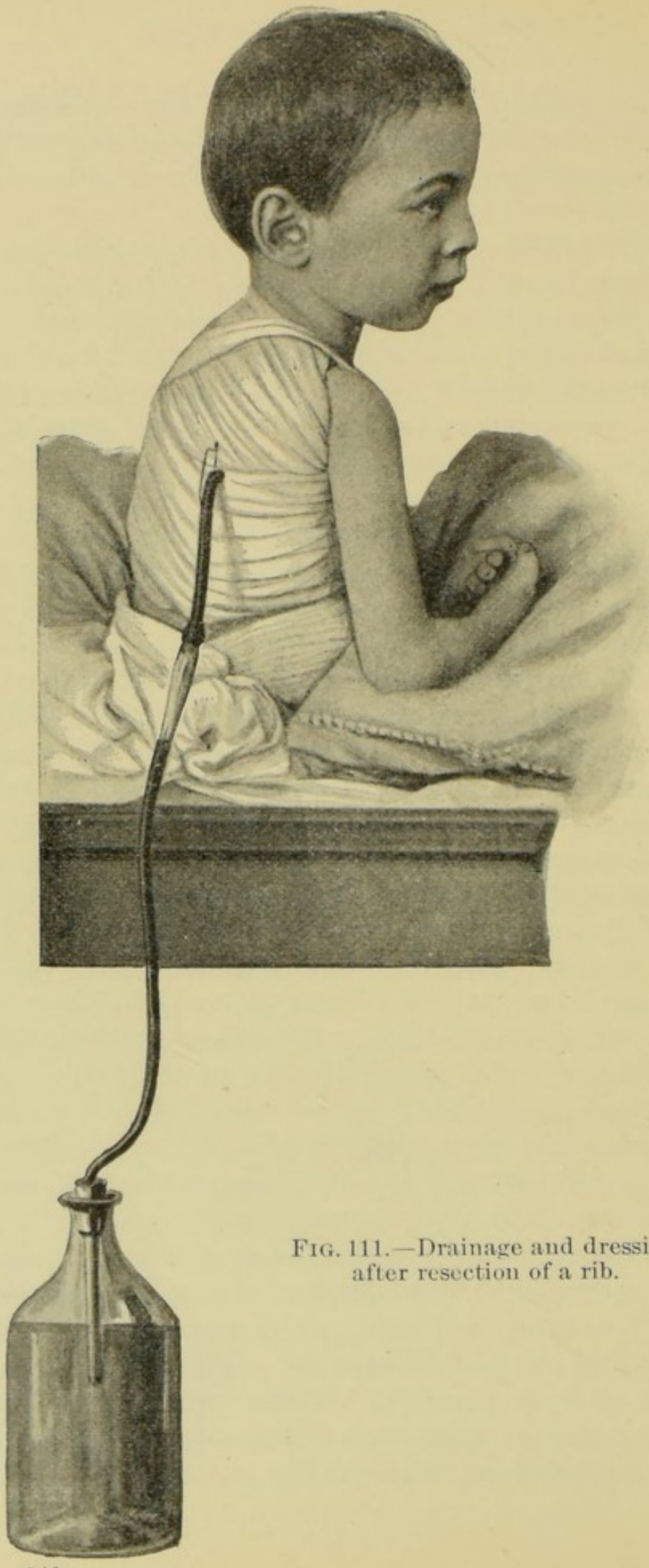


FIG. 111.—Drainage and dressing after resection of a rib.

micro-organisms together with polynuclear leukocytes, speak against tuberculosis.)

Differential Diagnosis Between Pleurisy and Pneumonia.—The fever is rarely as high in the former, it is atypic, the dulness is more resistant, and spreads uniformly over the posterior and anterior half of the chest, whereas in pneumonia the infiltration does not extend anteriorly until later. A tympanitic note is obtained above the dulness. Weak breath sounds and vocal fremitus absent. Traube's space is preserved in pneumonia.

Treatment.—If fever be present, rest in bed. A non-irritating diet with limitation of liquids. In recent cases apply ice-bags; for troublesome cough give narcotics. To excite resorption apply wet pack, also resort to rubbing in locally iodovasogen or ichthyol vasogen. If the exudate is excessive and the dyspnea is pronounced, puncture with a trocar and remove a portion of the exudate by means of an aspiration apparatus (simple piston-syringe with a double stopcock or the aspirator of Dieulafoy-Potain). Puncture the fifth or sixth interspace at the anterior axillary line. In case of empyema a rib should be resected (seventh rib at posterior axillary line) and followed by drainage; this is best performed by introducing a glass tube joined to a rubber one, which empties into a receiving vessel containing a solution of carbolic acid; the wound is dressed with a thick layer of cotton. Better drainage is obtained in children on account of the narrowness of the intercostal space by resection of a rib than by means of the Bülau method of drainage, which is so extensively practised in the case of adults. In place of the latter an attempt may be made in very young children, or in patients already debilitated, by means of the E. Müller permanent cannula, which consists in introducing by means of a trocar a curved metallic cannula furnished with a shield. After the pus has been removed apply a tight cotton and gauze dressing. During convalescence give a strengthening diet, cod-liver oil, extract of malt, the iodid of iron, respiratory exercises, and sojourn in the country.

DISEASES OF THE DIGESTIVE ORGANS

DISEASES OF THE MOUTH AND PHARYNX

GENERAL DISCUSSION

DISEASES of the mouth and pharynx represent, next to gastro-intestinal diseases, the commonest affections of the early years of childhood. Certain physiologic peculiarities of the infantile oral cavity, and especially the sensitiveness of the mucous membrane to injurious external influences of all varieties, favor a predisposition to various diseases. The true etiologic factors of this disease include mechanical, thermic, and, above all, bacterial irritants. The disease process may, however, develop to a certain extent as a sequal to diseases of the gastro-intestinal tract or constitutional anomalies.

BEDNAR'S APHTHÆ

Bednar's aphthæ are small superficial ulcers at both sides of the palatine raphé, where the palatine mucous membrane is tightly drawn over the hamulus of the pterygoid bone. The entrance of bacteria through sucking or swabbing out of the mouth converts epithelial defects which have followed pressure or rubbing into grayish-white, round, or oval ulcers surrounded by a red areola; these ulcers heal in a short time by the formation of new epithelium. Treatment is unnecessary. (A miliary collection of epithelial cells in the raphé of the palate is a frequent physiologic condition of nursing infants.)

STOMATITIS

Catarrhal stomatitis is an inflammation of the oral mucous membrane which manifests itself only by redness, swelling, and tenderness of the mucous membrane, which tends to bleed easily, as well as increased salivation. The

condition heals in a few days, provided the mouth is kept thoroughly cleansed and a non-irritating diet administered. The abundant secretion of the oral mucous membrane does not possess a foul odor as in aphthous and ulcerative stomatitis. Etiologic factors are the irritation incident to eruption of the teeth and, far more frequently, ectogenic and endogenic infection of the oral cavity by various bacteria due to neglect of the mouth and teeth; in infants infection occurs not rarely due to careless swabbing out of the mouth (Stooss, Epstein).

Aphthous stomatitis is an inflammatory and at times epidemic condition of the mouth due to distinct forms of bacteria (also the etiologic factor of foot-and-mouth disease). The symptoms of catarrhal stomatitis are accompanied by the presence on the oral mucous membrane of small, round, grayish-yellow exudations surrounded by a red areola, or in place of them, after the epithelium has been removed, are seen, corresponding in size, ulcerated areas covered by a lardaceous material. The oral secretion has a somewhat foul odor. The general health is considerably disturbed and at times a fever of 39° to 40° C. [102.2°–104° F.] sets in. The painfulness of the inflamed mucous membrane interferes with the ingestion of food. Nephritis is an occasional complication (Seitz, Hagenbach). Recovery occurs in from eight to ten days.

Diagnosis.—When the aphthæ develop primarily upon the isthmus of the fauces it is possible to make an error in diagnosis and call the condition lacunar angina, at least until the affection has spread throughout the oral cavity.

Treatment.—Swab the aphthæ with 1 to 2 per cent. solution of silver nitrate, or the silver stick, or the tincture of rhatany may be applied. Wash the mouth with a disinfectant solution, [such as Dobell's solution, or a solution of chlorate of potash (3 per cent.), or hydrogen peroxid (1 teaspoonful to 1 ounce)]. When the ingestion of food is interfered with, anesthetize the mucous membrane with aneson.

PLATE 35

FIG. 1. **Aphthous Stomatitis and Beginning Ulcerative Stomatitis.**—The oral mucous membrane is diffusely reddened and swollen and has a moist, glossy appearance. Deposited on the mucous membrane is an exudate which is either rounded or irregularly formed; the deposits vary in size from a pin head to a lentil and are of a grayish-yellow color surrounded by a dark red zone (aphthæ). The gums show a livid discoloration; they are relaxed, tend to bleed readily, and project from the teethlike growths. In some areas the edges have a yellow, pulpy appearance, where beginning ulceration is also noted. Increased salivation; fetor of the breath.

FIG. 2. **Thrush of the Oral Cavity.**—The oral mucous membrane is dry and brick-red in color. Colonies of thrush are seen on the lips, the buccal mucosa, on the hard and soft palate, and especially numerous upon the tongue; these foci are either punctiform or spread out over a large surface. The larger deposits, which are less tightly adherent, have been partially loosened by the movements of mastication and, becoming dry, show a dirty yellow discoloration.

Ulcerative Stomatitis (see Noma).—Ulcerative stomatitis is a peculiar form of stomatitis, extending always from the neighborhood of the teeth, which is generally confined to the gums, and is characterized by a tendency of the gums to undergo ulceration and by a penetrating putrefactive odor of the oral secretions. It is caused by intoxication with mercurial preparations, constitutional anomalies (scorbutus, diabetes), or by various bacteria (specific micro-organisms found are spirochetæ, Bacillus fusiformis—Bernheim).

Symptoms.—The outer border of the gums is reddened and, later, discolored livid; it is covered by a yellowish pulp and tends to bleed easily. An inflammatory exudate swells and relaxes the gums to such an extent that they are elevated from the teeth like new growths. Through epithelial necrosis it is converted into a smeary and discolored ulcer, which possesses an extremely foul odor. The affection may spread by contact to neighboring soft parts. The tonsils may even be involved, on account of which, under certain circumstances, the disease may be mistaken for lacunar angina; when the disease process is confined to the tonsils and near-by structures we speak of ulcerative angina (*angina ulcerosa*). The general health suffers markedly from interference with nutrition and the absorption of putrefactive products. Healing occurs in



Fig. 1.



Fig. 2.

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from ten to fourteen days; in cachectic children, however, the necrotic area may spread and death occurs, due to general sepsis.

Diagnosis.—In the differential diagnosis lacunar angina and herpetic angina must be considered. In the latter an acute rise in temperature occurs, together with the appearance of herpetic vesicles upon the mucous membrane, which undergo rapid destruction.

Treatment.—Potassium chlorate acts as a specific in this condition (3 per cent. solution for gargling and 1 per cent. solution internally).

THRUSH

Thrush is a peculiar type of stomatitis which is due to the deposition of the fungus Saccharomyces albicans upon the oral mucous membrane.

Symptoms and Course.—The irritation caused by the presence of the fungus in the epithelium produces inflammation of the mucous membrane with redness, swelling, and pain. Small milk-white thrush colonies soon show themselves upon the mucosa, and if their growth is undisturbed they rapidly enlarge, and, coalescing, cover large areas of the mucous membrane with a thick, dirty white coat. Primarily only the tongue and inner surface of the cheeks are involved, but later the lips and palate are included. The process may occasionally descend to the stomach, rarely further. The entrance of the fungus into the blood current may lead to metastasis to the internal organs.

Thrush is not altogether a harmless disease. Aside from the local disturbances due to the presence of the fungus upon the mucous membrane, which interferes with the ingestion of food and makes that procedure painful, another property is to be considered—the tendency to cause fermentation. As a result, the swallowing of the thrush fragments, together with the products of fermentation, is not infrequently the cause of dyspepsia and even inflammation of the intestines (thrush enteritis).

Diagnosis.—When the diagnosis is in doubt (diphther-

itic deposit?), a microscopic examination should be made for the presence of the network of the mycelium, the egg-shaped cells, together with the small shiny spores.

Treatment.—The fungoid coat must be removed as soon as possible by swabbing with borax, boric acid, hydrogen peroxid (1 teaspoonful to 1 ounce), or normal salt solution. This is performed with difficulty if the condition is of long standing, and can only be accomplished at the expense of hemorrhage from the mucous membrane, since the mycelium has penetrated into the deeper layers of the

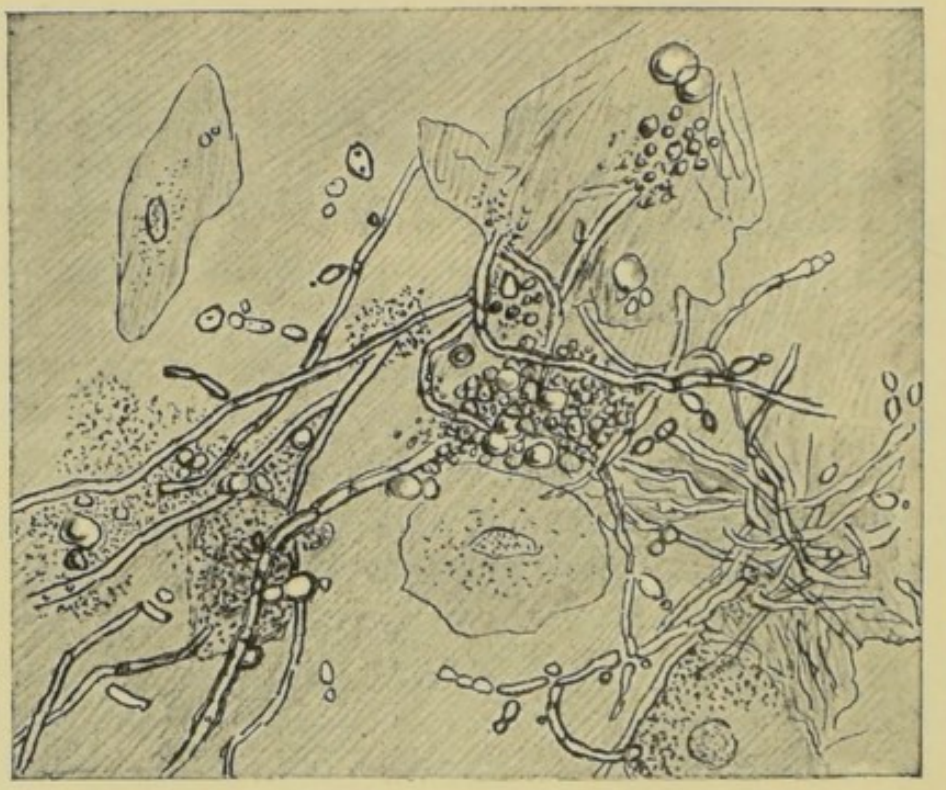


FIG. 112.—Microscopic picture of a thrush deposit removed from the mouth. Enlarged 350 times.

mucosa. After swabbing, paint with 1 to 2 per cent. silver solution (Henoch). This must be frequently repeated because of the tendency to relapse. As a preventive measure, the sucking bag of Escherich [containing borax] is recommended.

NOMA

Noma is a very rare gangrene of the cheek of bacteriologic origin. It begins with the development of a nodule on the inner surface of the cheek, which is converted into

a vesicle. The latter ruptures and leaves a grayish-brown scab behind. As the process progresses toward the skin a rose-colored and, later, brownish-blue spot appears on the latter, and finally an extensive black eschar forms. The inner surface of the cheek is converted into a foul-smelling, smeary, and gangrenous mass. Death occurs in from ten to twenty days. Spontaneous cure is rare and the mortality-rate is as high as 95 per cent. An early and complete excision gives the best results (von Ranke).

ANGINA

Non-specific angina is a very frequent disease of childhood. The direct causes of this condition may be streptococci, staphylococci, or pneumococci, which, being accidentally deposited during an acute cold, dyspepsia, etc., excite a catarrh and inflammation of the mucous membrane of the tonsils and the neighboring structures.

Symptoms.—The mild cases are accompanied only by redness, swelling, and active secretion of the tonsils—*catarrhal angina;* these manifestations develop with a moderate fever and difficulty in swallowing, but disappear in several days. In a severer grade of inflammatory irritation an exudate appears and the local and general symptoms are more marked. The tonsils are dark red, covered with a shiny mucus, and show disseminated yellow specks (lacunar exudate containing large numbers of bacteria and cells, but little fibrin)—*lacunar angina* (see Plate 25 and Fig. 96). The swelling and painfulness of the inflamed soft parts may develop to such a degree that difficulty in swallowing and even breathing results, and the speech becomes nasal. The disease is always accompanied by a high fever and dyspeptic phenomena. The neighboring lymph-nodes are swollen and an initial vomiting, chill, and headache are frequently met with. The fever disappears in several days and complete cure occurs gradually in one and a half weeks. Recurrences are quite common and occur sometimes at regular intervals (Fischl). Angina may in some cases be the point of exit of a septic

PLATE 36

Noma of the Cheek.—The whole left half of the face is phlegmonous, reddened, and swollen, and the weeping eye is closed by the swelling. The lower portion of the left cheek from the lower jaw as far as the angle of the mouth has been converted into a blackish eschar, which is surrounded by an inflammatory area from which the epidermis has been loosened. In the neighborhood of the ramus of the jaw perforation has occurred, from which, as well as from the angle of the mouth, an ichorous exudate is seen to flow. Death imminent. (Clinic of Escherich, Vienna.)

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infection, or in other cases it represents the primary focus of a subsequent organic disease, such as endopericarditis, appendicitis, and polyarthritis.[1]

Less dangerous, but extremely troublesome and painful, is the development of local suppuration and the formation of a tonsillar abscess. The latter may be suspected if after four or five days the fever fails to drop and the local and constitutional symptoms increase in severity. The involved tonsil is phlegmonous, red, and swollen; the secondary edema is often so marked as to threaten asphyxiation. An incision offers immediate relief.

Diagnosis.—The clinical differentiation between diphtheritic and non-diphtheritic angina may sometimes be impossible. In favor of the latter are a high fever, marked redness, swelling and painfulness of the soft parts, bilateral origin, slight extension, a rare tendency of the deposits to coalesce, and their gruel-like consistency, together with the fact that they adhere but slightly to the lacunar spaces; also a doughy swelling of the lymph-glands (hardening in diphtheria). However, all of these manifestations may fail, in which case a microscopic examination of the discharge will decide the diagnosis. In the non-diphtheritic type of angina this examination will reveal little or no fibrin, a large number of bacteria of various forms; whereas in diphtheritic angina a large amount of fibrin is present, few bacteria, but an over-

[1] In favor of the etiologic relationship between angina and rheumatism is the oft-proven fact that if the tonsils of an individual in whom frequent recurrences of rheumatism are preceded by angina are extirpated the rheumatism disappears.



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whelming number of Löffler bacilli, which are grouped in nests (see Figs. 93, 94).

Prophylaxis.—Strict cleanliness of the mouth and teeth. Treatment of carious teeth, the micro-organisms of which are frequently the cause of chronic recurring angina.

Treatment.—Rest in bed, light diet, Priessnitz's compresses, and spraying of the pharynx. Potassium chlorate internally and as a gargle; incision is indicated in case of abscess formation. [Where rheumatic infection is suspected, the salicylates are indicated.—Ed.]

HYPERPLASIA OF THE LYMPH-TISSUE OF THE PHARYNX

At the entrance of the nasal and oral cavities into the pharynx are a number of lymphoid organs, which are spoken of collectively as the *pharyngeal lymph ring*. This tissue in children is frequently subject to chronic hyperplasia. Hyperplasia of the palatine tonsil—which may be congenital or acquired by mechanical irritation—causes but slight disturbance, but when of a higher grade it may cause difficulty in swallowing and breathing. A portion of the tonsil is removed by tonsillotomy.

Of far greater clinical importance is hyperplasia of the pharyngeal tonsil (adenoid vegetations). The pharyngeal tonsil, a furrowed, sponge-like glandular structure at the roof of the pharynx, normally undergoes gradual complete involution until the age of puberty is reached. It is, however, frequently, on account of mechanical and thermic irritation, the seat of acute inflammation. Fever, dyspnea, and dysphagia arise. Sleep is frequently interrupted for want of air (falling back of the tongue, collection of tenacious nasal secretion) and nurslings even are not rarely subject to eclamptic attacks; at other times the sleep is extraordinarily deep—accompanied by enuresis (?). Should the affection remain strictly localized (angina pharyngea) it becomes the source of frequent error.

If such inflammations develop repeatedly or if the

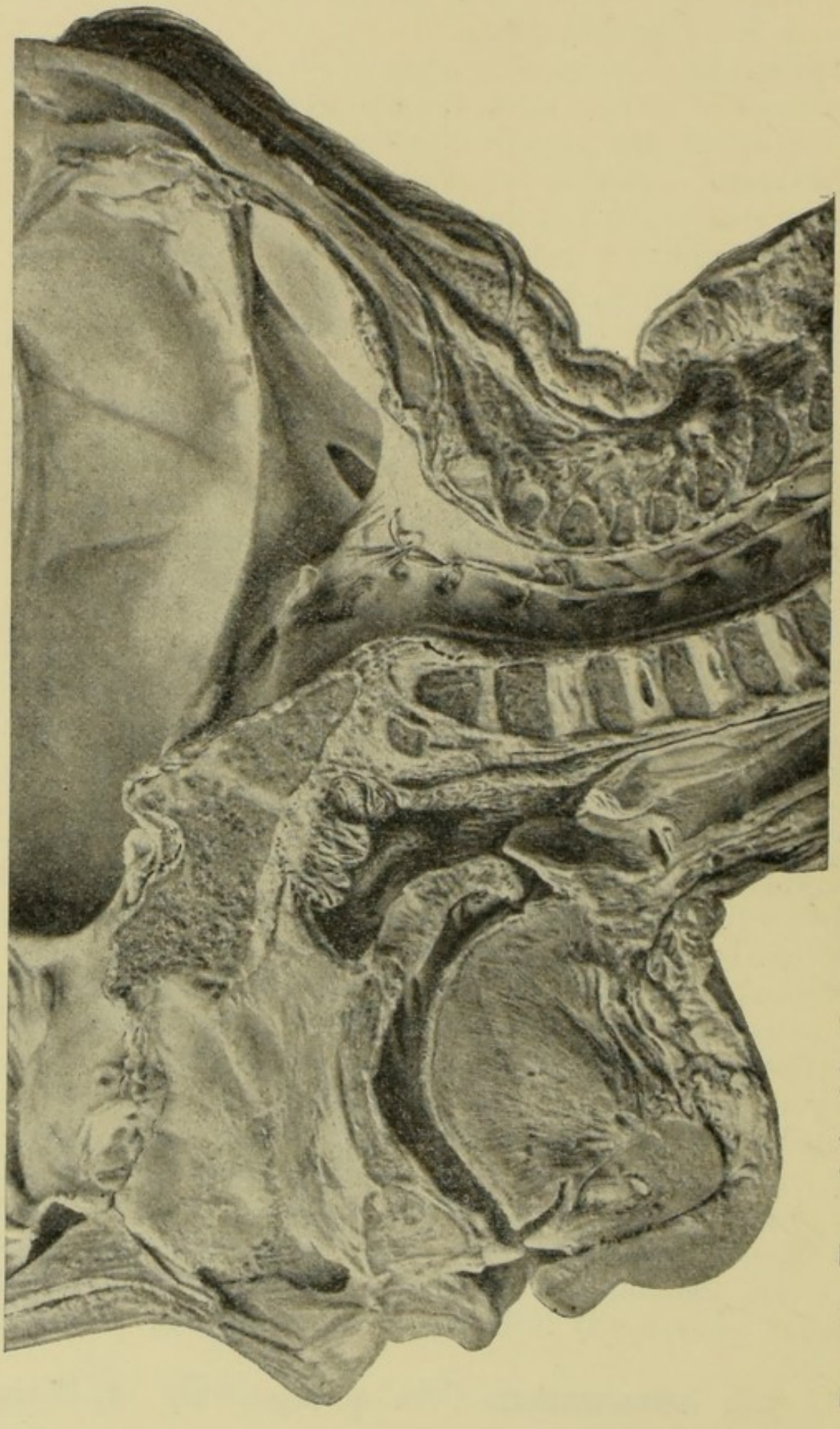


FIG. 113.—Hyperplasia of the pharyngeal tonsils. Section of the skull of a cnild one year old. The enlarged tonsil is seen on the roof of the nasopharyugeal cavity (below and to one side of it is the opening to the Eustachian tube).

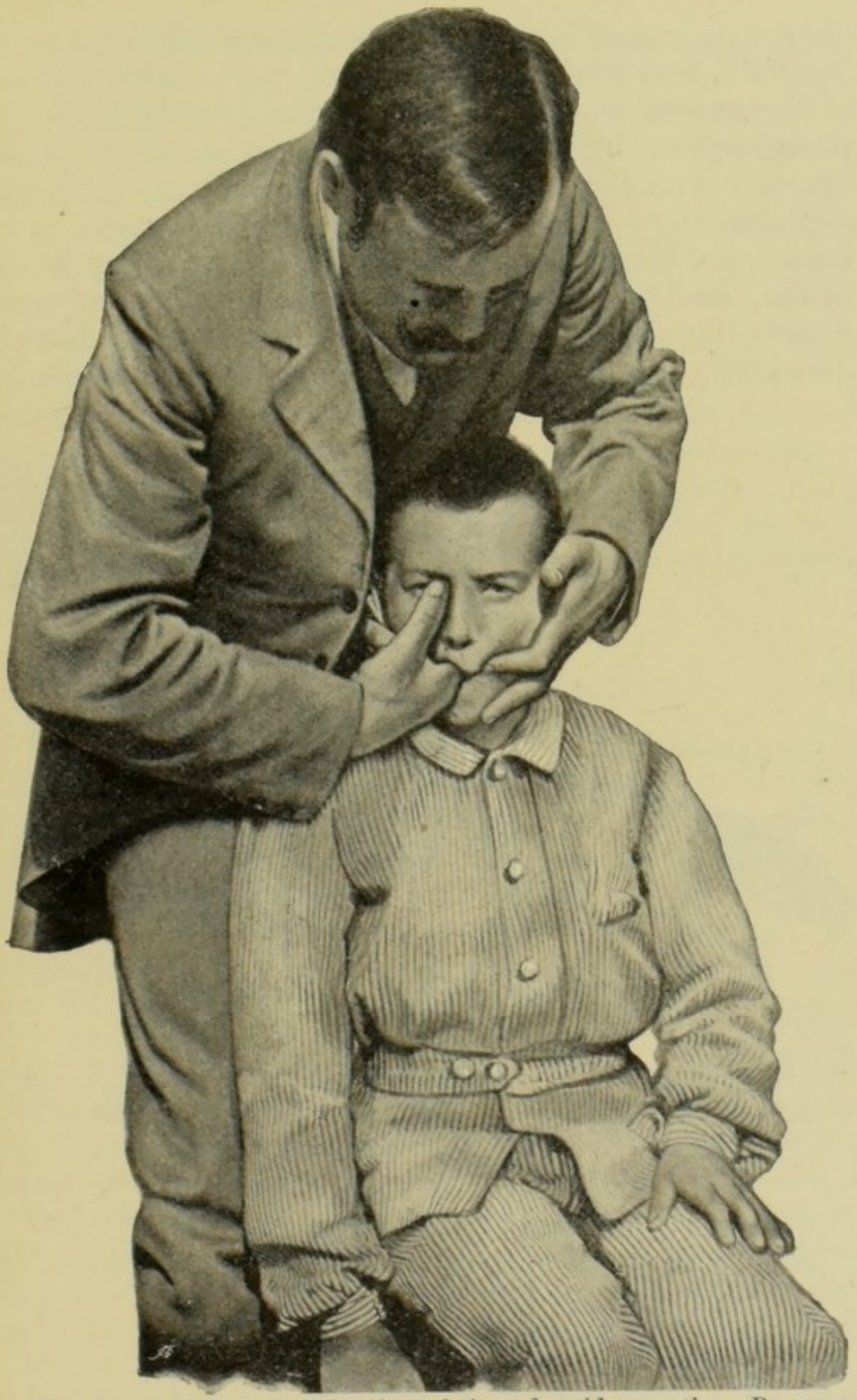


FIG. 114.—Digital examination of the adenoid growths. By pressing the cheek between the teeth it becomes impossible for the patient to bite the examining finger.

pharyngeal tonsil becomes hypertrophic for other reasons (scrofula, hereditary predisposition), marked disturbances of the general health will arise. The growths filling the nasopharynx, by displacement of the posterior nares, interfere with the functions of the nose as a respiratory and olfactory organ; by obstructing the pharyngeal opening of the Eustachian tube the sense of hearing is impaired, and by exerting pressure upon the pharyngeal vessels stasis of blood and lymph occurs at the base of the skull. The most significant of these disturbances is

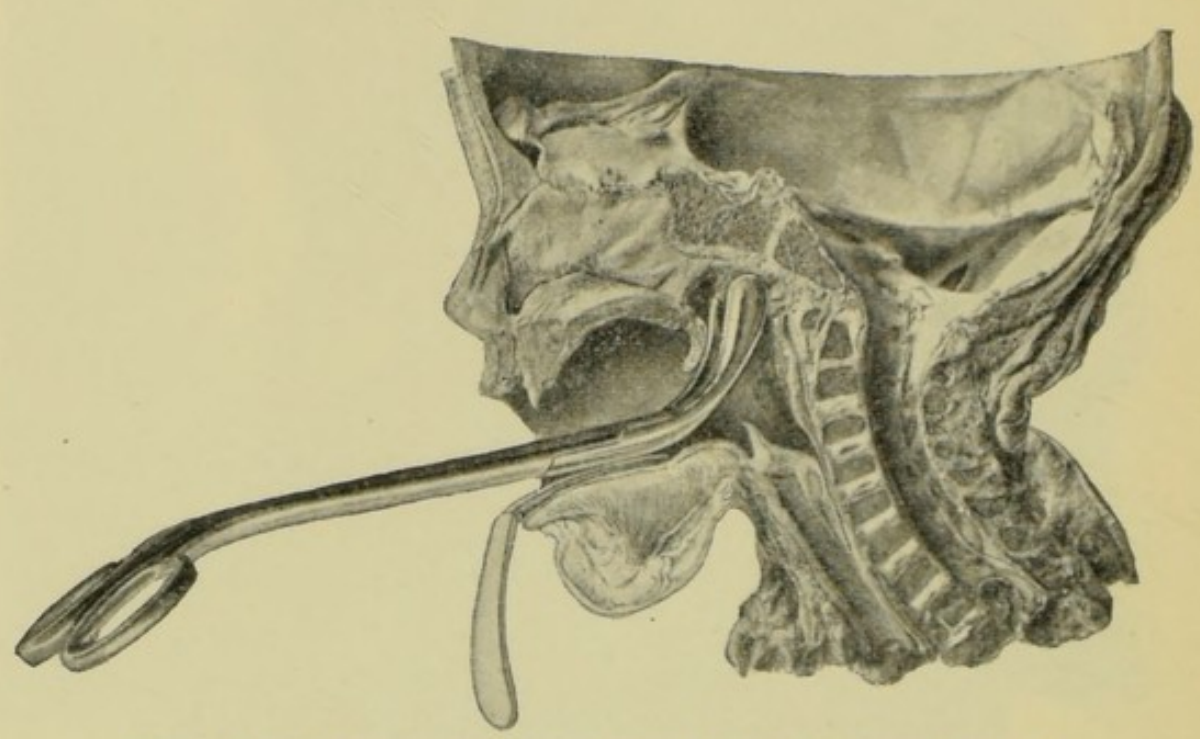


FIG. 115.—Adenotomy by means of Schech's spoon-forceps.

the interference with nasal respiration. The air enters only through the mouth, which is kept open, with resultant catarrh of the pharynx and air-passages; the breathing is noisy and during sleep, which is frequently interrupted and abnormally sound, it is snorting. The ingestion of liquids is (especially for nurslings) also interfered with. In the course of time organic changes follow: The nose, palate, upper jaw, and thorax undergo disturbances of development. The patient frequently complains of headache and loss of power to fix the attention upon any subject for any length of time (probably as

a result of the stasis). Furthermore, there are disturbances of speech.

The **diagnosis** is determined by digital examination or posterior rhinoscopy, although it may usually be made by the facial expression and the palatine speech. The eyes look sleepy and are half closed, the nose is noticeably small, and the mouth, which is always opens, has a somewhat stupid appearance (see Fig. 77). Inspection of the oral cavity shows an elevated palate which has the form of a pointed arch, and it will be noted also that the velum palati (which is generally thickened) does not lie back against the posterior wall of the pharynx in phonation.

The **treatment** consists in performing adenotomy by means of a ring-knife or spoon-forceps. The recovery is usually wonderfully rapid and permanent if radical operation is performed.

RETROPHARYNGEAL ABSCESS

Retropharyngeal abscesses are, as a rule, rather rare in childhood and are practically only observed in nursing infants. They may be due to suppuration of a retropharyngeal lymph-node or a burrowing abscess (v. Bókay). The abscess is generally situated in the neighborhood of the third or fourth cervical vertebræ and involves the esophagus and larynx. As soon as a certain size has been reached we note the development of dysphagia, regurgitation of liquids, snorting breathing, and, finally, dyspnea. Externally, swelling is noted on the affected side of the neck. Spontaneous rupture or artificial opening of the abscess is immediately followed by a disappearance of all the untoward symptoms.

Retropharyngeal abscess is distinguished from croup by the deep gurgling sound heard with the respiratory murmur (v. Bókay) and by means of digital examination, which in doubtful cases of laryngeal stenosis must never be neglected. The same symptoms are noted in the rare retrolaryngeal abscess which may occur with decubital ulceration after intubation, and also primarily in nurslings.

GASTRO-INTESTINAL DISEASES

GENERAL DISCUSSION

Although the gastro-intestinal diseases in older children run, on the whole, the same course as in adults, yet in the earlier periods of life they possess certain characteristics as regards development and course. Thus, severe symptoms are frequently called forth by injurious conditions which in the adult elicit none or barely any response; the disease process spreads rapidly over large sections or throughout the whole digestive tract; mild affections are often rapidly converted into serious disease forms, thus, dyspepsia may lead to catarrh and inflammation.

Etiology.—The causes are not to be sought for so much in external influences as in the especial predisposition in the nursing infant to diseases of the digestive tract, which are attributed to delayed development and to the peculiar anatomic relationships.

The miniature size of the nursling's stomach, the vertical position of that organ, the absence of a fundus, the weak closure of the cardia, and the irritability of the gastric nerves, all tend to favor vomiting, so that many children vomit after each meal (*habitual vomiting*) during the first three months until the fundus begins to form. This form of vomiting, contrary to that accompanying fermentation, is unassociated with nausea, retching, and, as a rule, without ill effects upon the general health.

The imperfect functional capacity of the gastric muscles for work and the relative length of the intestines, especially the large intestine, is frequently the cause of *habitual constipation* (not the only causes, however, as the same result is produced by improper feeding, overeating, and painful rhagades of the anus). If at the same time there exist congenital displacement and anomalous elongation of the colon, as well as a defect of its musculature, a high-grade obstruction and hypertrophy of the large intestine develops—*Hirschsprung's disease* (Jacobi, Hirschsprung, Concetti, Johannessen).

The weakness of the musculature, the tenderness of

the mucosa, the size and number in the latter of blood-vessels, the abundance of nerve-elements, and the peculiar fact that the myelin sheath of the mesenteric nerves is still imperfectly developed, account for the rapid exhaustion of the intestines and the sensitiveness of the mucous membrane toward irritation ; the enteralgias so common in infants may also be explained in that manner. The infantile digestive apparatus is sufficiently active as concerns absorption and secretion (Gundobin, Heubner) and, indeed, absorption proceeds more rapidly than in adults because of the relatively long intestines. On the other hand, the digestive function is so much greater in comparison with the body weight that this duty can only be performed properly provided it is not subject to the strain of too rich a diet or one assimilated with difficulty. We must also consider the fact that digestive ferments like rennin, pepsin, trypsin, etc., are not relatively delayed in development as regards their activity in young infants, yet, on the other hand, that function of fermentative substances, which in intermediary metabolism controls subsequent stages of digestion and especially assimilation of the absorbed mass, may be delayed in development in the earlier years of life. Such may be the cause of manifold disturbances of metabolism, which may also at times be the fundamental etiologic factor of diseased conditions, as, for example, the pedatrophy of children (Pfaundler).

Aside from the mentioned internal factors of disturbances of digestion in children, a whole series of *external influences* are to be considered, which may, with few exceptions, be traced to improper care or lack of it. Of first importance is unsuitable, insufficient, or overabundant food which may have undergone chemic or bacterial degeneration ; also infection from the child's surroundings, as well as thermic, mechanical, chemic, and also, probably, nervous irritation.

The *alimentary disturbances* play the most important rôle. Breast-fed children may also suffer from this condition when they receive too much food, or when the

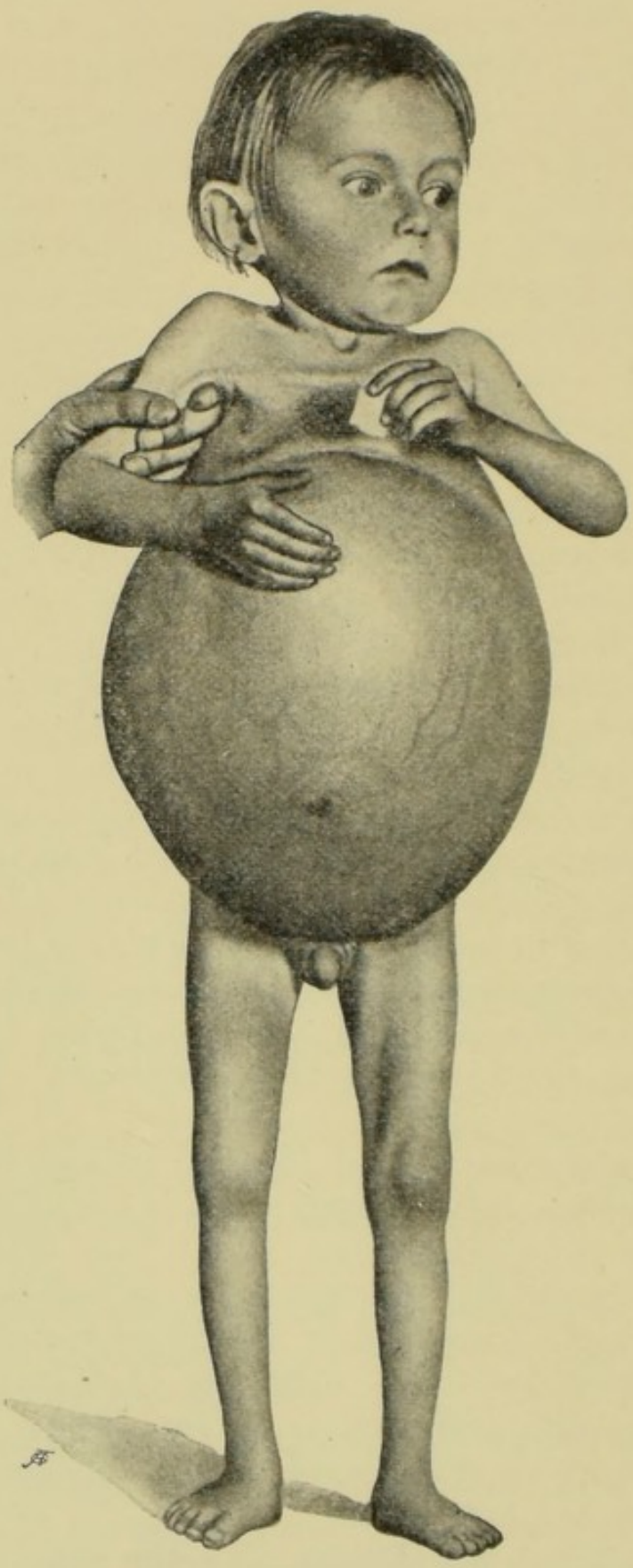


FIG. 116.—Hirschsprung's disease before treatment. (Clinic of Escherich.) For description, see p. 358.



FIG. 117.—Hirschsprung's disease six months after treatment. (Clinic of Escherich.) For description, see p. 358.

FIGS. 116, 117.—Hirschsprung's disease. Boy three years and nine months old. Since birth suffered from constant intestinal catarrh, obstinate constipation, alternating with diarrhea and increasing abdominal distention. On examination the abdomen measured in circumference 77 cm. [30.8 in.] and the body length 87 cm. [34.8 in.]. The diaphragm was situated abnormally high, the thorax short and noticeably enlarged at the base, abdominal walls abnormally distended and presenting dilated veins. Active peristaltic movements visible, extending toward the left side. Deficient abdominal pressure. Percussion : Distention of the abdomen by intestines filled with air. Auscultation : Splashing and gurgling sounds. Palpation : Liver, spleen, and kidneys plainly palpable, abnormally movable, the spleen enlarged and hard. Examination of the rectum: Abnormal dilatation of the lower section of the large intestine, which has been converted into a smooth-walled cavity measuring 20 cm. [8 in.] in diameter, whose anterior and upper walls are not palpable. Irrigation was followed by the discharge of foul-smelling flatus and partially liquid, partially clay-like, well-digested stools. Appetite good. Treatment: Regular irrigation with solution of thymol ; massage; faradization; binder to the abdomen. The child died in six months following surgical intervention.

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mother's milk has been injuriously influenced by gross errors of diet during the nursing period, or by the admixture of bacteria (staphylococci) from the ducts of the mammary glands (staphylococcus enteritis, Moro). Artificially fed children are more frequently threatened with gastro-intestinal diseases, especially when they are given a diet unsuitable to their age, as, for example, overabundance of starchy foods before the end of the third month (insufficient diastasic ferment). The infantile digestive functions may also be disturbed to a certain extent when fed with cows' milk, although the latter simulates in composition the nutritive elements of woman's milk, yet it is poorer in easily digestible constituents; furthermore it cannot be drunk immediately at its source, for it must first receive special preparation, on account of which its digestibility is lessened and often becomes contaminated by bacteria. Owing to the ability of the digestive tract of normally developed infants to functionally respond even after a short time to these dietetic changes, a diet which is digested with difficulty may be assimilated provided the amount of food ingested is not disproportionate to the digestive strength. Overfeeding—which is unfortunately so common—leads to exhaustion of the intestinal and glandular epithelium with gradually increasing fail-

ure of assimilation of the food, which, stagnating in the stomach and intestines, undergoes abnormal putrefactive changes (Biedert's "injurious food remnant"). The products of this process (fermentation of sugar-albuminous putrefaction) create local irritation and symptoms of general intoxication (Escherich, Heubner).

Still another cause of disturbances of digestion is the ingestion of food which has undergone chemic or bacterial decay. The gross impurities of cows' milk or infection with a specific pathogenic germ (tubercle bacilli, etc.) are less common, as these dangers are nowadays generally avoided, since it has become the custom to examine and boil the milk. On the other hand, the lapse of time before cows' milk can be administered tends to increase the action of lactic acid bacteria and other saprophytes. If the milk has been preserved in a low temperature it simply turns sour on account of fermentation and causes only local symptoms of intestinal irritation (Marfan, Escherich), whereas, if the milk has been preserved for a long time in a high temperature, as during the hot months, multiplication of those bacteria dependent upon heat will be noted, and the milk will receive certain toxic properties from their metabolic products which when ingested will cause pronounced manifestations of intoxication (cholera infantum).

A certain number of gastro-intestinal diseases are either directly or indirectly attributable to the *influence of improper nursing*. Indirectly they are due to impure and damp air, dark dwellings, lack of cleanliness and warmth, together with improper feeding, all of which rapidly exhaust and overcome the inherited resisting forces and with them the ability of the intestines to perform their functions. Directly, inasmuch as unhygienic conditions of life favor the development of true intestinal disturbances. These infections are similar in the beginning and course to the specific intestinal infections, such as typhoid, dysentery, and Asiatic cholera. They are caused by various forms of infectious germs (streptococci and bacilli similar to the colon bacillus), which enter the intestines by way

PLATE 37

FIG. 1. **The Stool of Melena Neonatorum.**—Blackish-red coagulated mass of blood with slight admixture of meconium. Dirty red areola.

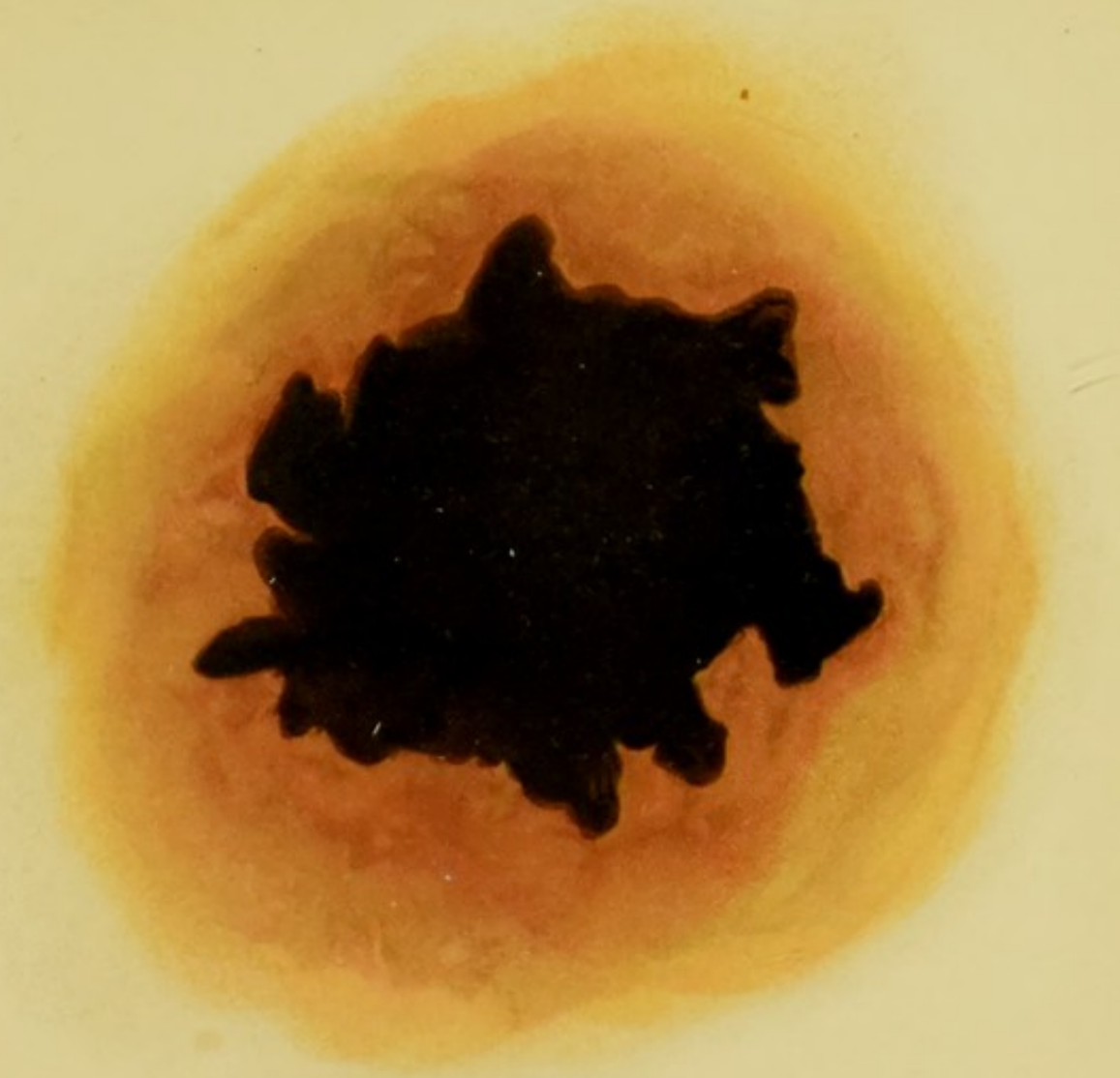
FIG. 2. **Dyspeptic Stool of a Breast-fed Child.**—Remnants of milk, whitish-gray and green colored fat and soap remnants enclosed in yellow liquid masses of mucus. Dirty yellow areola. Odor and reaction strongly acid. (Drawn by Dr. Moro, Escherich's Clinic.)

of the mouth or anus, through the air, by transmission from drinking-cups, etc. Here inflammatory changes are affected under certain circumstances, metastasis occurs through the injured intestinal walls, and general infection results. Streptococcic enteritis, described by Escherich, may be mentioned as a type of intestinal infection dependent upon the nutrition; a disease which tends to spread in hospitals and foundling asylums (Epstein, Rossi, Finkelstein, Escherich).

Symptomatology.—The manifold etiologic factors by no means indicate a similar variety of clinical symptoms, for, on the contrary, diseases of digestion in infants are usually accompanied by a comparatively uniform symptomatology. The preventive measures of the organism, consisting in vomiting, increase of peristalsis, watery and mucous stools, by means of which the action of the injurious intestinal contents is shortened or at least weakened, vary but slightly and according to the character, intensity, and duration of the injurious influences and the resisting forces of the individual. Even grave organic changes of the intestinal canal manifest themselves by only a few variable symptoms. It must not be forgotten also that the local changes observed at necropsy are frequently, in a certain sense, contrary to the severity of the symptoms during life and offer no satisfactory explanation for them (Heubner). Sharply defined disease pictures of intestinal conditions in childhood depend less upon the peculiarity of the symptoms than upon their characteristic grouping.

DYSPEPSIA

Dyspepsia is a result of injurious effects upon alimentation or disturbances of digestion and absorption due to organic weakness.





Dyspepsia following disturbances of fermentation (in bottle-fed children): Regurgitation and vomiting of non-coagulated milk, even if the vomiting does not occur until a certain time after feeding. Constipation follows the collection of undigested masses; formation of gas (enteralgia).

Stools.—Pale yellow, shaped in small or large firm nodules, which contain remnants of food and have a stale or foul odor.

Marked insufficiency of fat digestion; fat diarrhea (Biedert): Excretion of abnormal amounts of fat in the stools. A state of chronic diarrhea usually exists. Emaciation.

Stools.—White, fatty, mucoid; containing abundance of soaps, fatty crystals, fat-drops, and fat-plaques.

Acid-fermentation dyspepsia in breast- and bottle-fed infants; acid eructations, vomiting, and a sour odor from the mouth. The stools, which are increased in number and contain large masses of undigested or decomposed remnants of food, are passed with loud flatus and much restlessness on the part of the child. An abnormal amount of gas and acid formation takes place in the stomach and intestines with resulting distention of the abdomen, enteralgia, loss of appetite, and a standstill or loss of body weight. The course is afebrile.

Stools.—At the beginning these possess a strong acid reaction and odor (butyric acid); at first they are yellow in color, then vary between milk white and green (or brown when amylaceous food is eaten). Yellowish-white flakes and remnants, consisting of fat, alkaline soaps, and epithelium, imbedded in bacterial masses are found mixed with mucus in gruel-like stools. Microscopic examination discloses remnants of milk, fatty detritus, and single epithelial cells.

INTESTINAL CATARRH

Intestinal catarrh may be a sequel to dyspepsia or a prelude to inflammatory processes.

Symptoms.—The intestinal mucous membrane is hyperemic, swollen and relaxed, and a portion of the epithe-

PLATE 38

FIG. 1. **The Stool of Intestinal Catarrh.**—Aside from the lumpy, thread-like, yellowish-brown masses of mucus, we also see isolated grayish-green dyspeptic flakes and shreds. The whole is surrounded by an extensive pale, dirty green, sharply outlined zone. Acid reaction.

FIG. 2. **The Stool of Infectious Colitis.**—In the partly dark green, partly ochre colored, and partly colorless lumps of mucus are seen scattered hemorrhagic points and several large drops of blood as well as whitish flakes of pus. Narrow, greenish areola. Foul odor. Alkaline reaction.

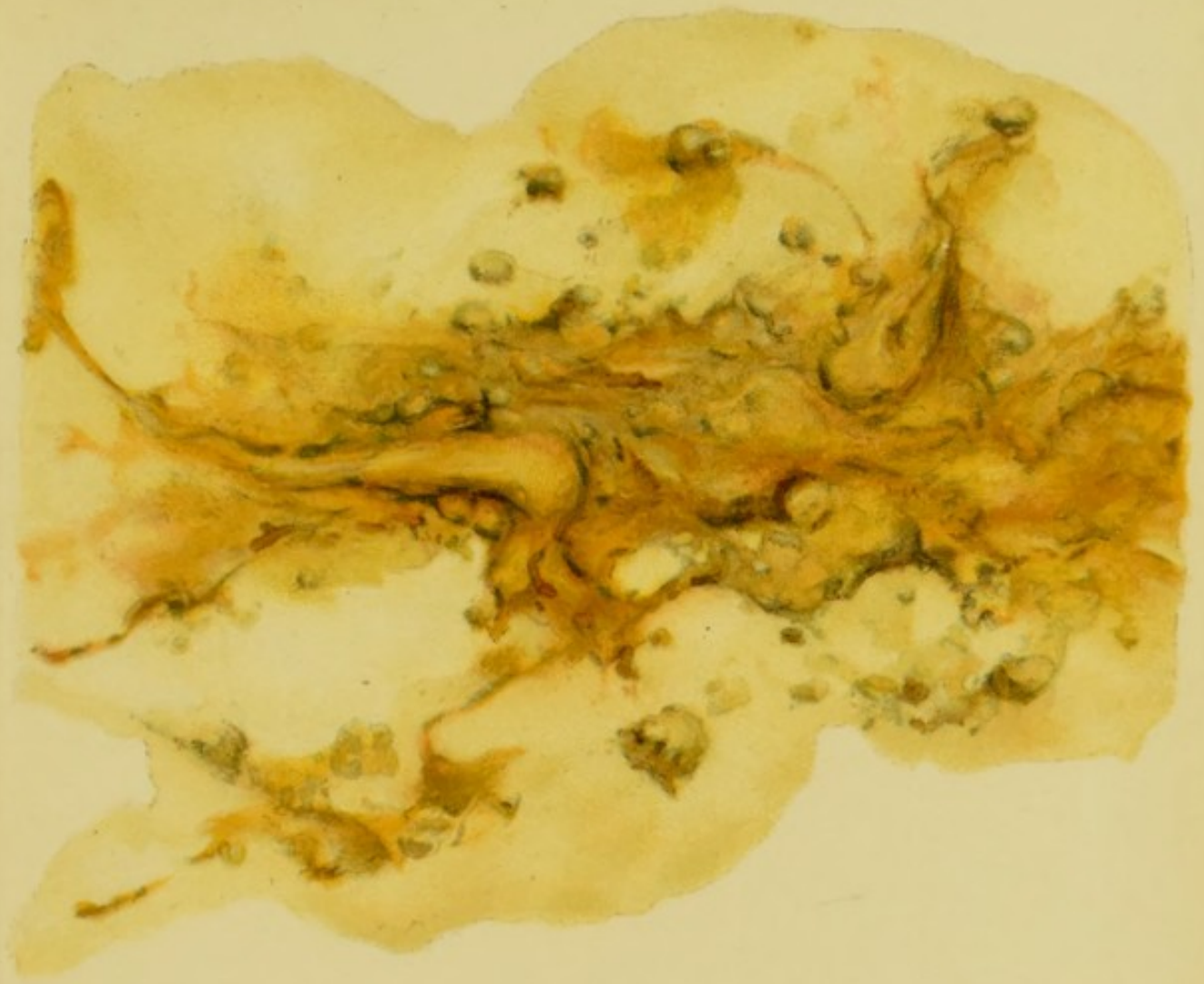
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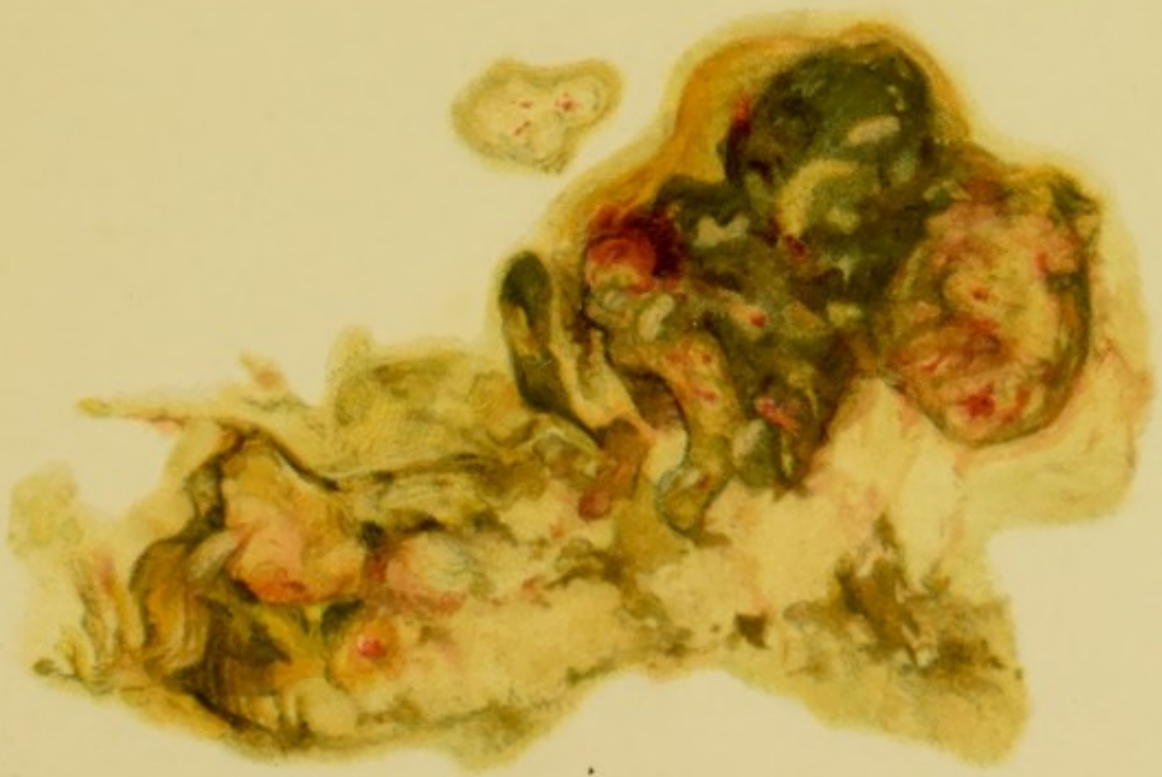
lium is destroyed; it may excrete a watery fluid and mucus. The follicles are swollen and the mesenteric nodes are injected. Severe local symptoms are accompanied by general toxic manifestations. These include vomiting, frequent and copious watery and mucous stools, decrease of urinary secretion, increased thirst, and active peristaltic movements in the abdomen, which is sensitive to pressure. The urine frequently contains albumin. A rapid decline, and at times clonic-tonic twitchings and toxic dyspnea are noted. Fever develops, especially when the stomach is markedly involved.

Stools.—These are passed in spurts, with much noise, and are at first similar to those of acid dyspepsia, yet contain a marked increase in water and mucus. Later they become less feculent and assume a more brownish appearance. The reaction is usually acid. Microscopically they show an abundance of mucus, a large amount of intestinal epithelium, and a large number of acidophilic bacilli, which stain by Gram's method in contradistinction to the normal intestinal bacteria, which react negatively to Gram's stain.

CHOLERA INFANTUM

Cholera infantum is a very acute condition accompanied by the symptoms of severe collapse, vomiting, and diarrhea, brought on by the ingestion of milk which has undergone ectogenic degeneration on account of preservation in too high a temperature (Escherich). It occurs most frequently in the summer months, infants between five and seven months of age are most prone to develop





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it, and that, too, of a severe type (Schlossmann). In the majority of cases death occurs in from one to six days in consequence of excessive loss of water and severe intoxication (the heart weakens, acute course, cyanosis; later, sclerema and hydrocephalus).

A most unfavorable prognostic sign is the vomiting of *coffee-ground masses* (blood).

Stools.—Greenish-yellow, watery stools, throughout which are interspersed colorless flakes of mucus. In some cases they consist only of a colorless and odorless liquid. Alkaline reaction.

INTESTINAL INFLAMMATIONS

Intestinal inflammations are diseases of the intestines which follow catarrhal and dyspeptic processes, or they may be primary.

Morbid Anatomy.—Swelling, inflammation, and, finally, suppuration and ulceration. Swelling of the mesenteric and inguinal glands.

The stools are considerably increased in number and contain mucus, blood, and pus. If only isolated portions of the intestines are involved, practically normal stools may be passed alternating with those of a pathologic nature. As a result of the excessive watery discharges per rectum, the secretion of urine is diminished and the thirst increased. If gastric catarrh or edema of the cerebral meninges is also present, vomiting develops. Symptoms of cerebral irritation are frequently present. The loss of body juices, loss of sleep, and spreading of the infection leads to a rapid decline. Complete cure does not occur even in favorable cases until after several weeks.

Sequelæ.—Thrush is favored by constitutional weakness; intertrigo is due to the irritation of the frequent stools and the concentrated urine; metastatico-septic processes are also met with. Secondary infections include ecthyma, furunculosis, phlegmasia, pneumonia, otitis, cystitis, nephritis, and pyelonephritis.

PLATE 39

Prolapsus recti of a moderate degree, due to straining and pressing during evacuation of the bowels. Secondary erythema of the nates. (Clinic of Escherich, Vienna.)

Other forms of intestinal inflammation to be considered are :

Gastro-enteritis, which usually represents only an exacerbation of dyspeptic or catarrhal disease, is especially likely to attack the small intestine. Not until a later stage is the large intestine involved.

The copious stools are watery and discharged in spurts ; and contain at the beginning only remnants of food, but consist later of an odorless or foul-smelling green or grayish-yellow mucoid fluid, with which are mixed blood and pus. The reaction is usually alkaline. The abdomen is frequently distended and tense, but the umbilicus not obliterated.

Colitis, a primarily localized inflammation of the large intestine, is frequently an expression of a true intestinal infection which develops at times in limited epidemics (Widerhofer's follicular enteritis). The discharges contain colon-like bacilli (Rossi, Finkelstein, Escherich, Celli), and more recently dysenteric bacilli of the Shiga-Kruse as well as of the Flexner type were found by American authors and Jehle (clinic of Escherich). Accordingly this disease seems to bear a close etiologic relationship to epidemic dysentery (" colitis dysenteriformis," Concetti).

The disease always has an acute onset and is usually accompanied by a high irregular and remittent fever. The general health is unfavorably influenced by colic, troublesome tenesmus, and sleeplessness, and the symptoms of collapse set in rapidly. The abdomen is retracted and the thickened descending colon may be palpated as a sausage-shaped tumor which is tender upon pressure. Prolapsus recti develops frequently, owing to straining and pressing during stools.

The *stools*, which are accompanied by tenesmus, consist



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exclusively of a little serous fluid and bile-stained gelatinous or colorless frog-spawn-like mucus, which is intermingled with more or less pus and fresh red blood in traces or in large amounts. The number of stools is enormous, sometimes as many as forty or fifty are passed per day. The amount of each discharge at the beginning is considerable, but rapidly lessens, so that finally only a small trace of mucus sprinkled with blood appears. The reaction is alkaline and the odor at the beginning is stale; later, however, when putrefaction of the albumin-containing intestinal secretion sets in, it becomes foul.

CHRONIC AFFECTIONS

Chronic affections frequently follow the foregoing conditions, but at times they are caused by the continued action of injurious factors (errors in diet and unhygienic dwellings). The mucous membrane of the intestines is frequently catarrhal, relaxed, and anemic throughout its whole extent, or it shows residue of a preceding inflammation. The follicles, Peyer's patches, and mesenteric glands are swollen. As an acute form of intestinal inflammation passes into the chronic stage all of the violent symptoms disappear without recovery. Accompanied by intervals of exacerbation and temporary improvement the course may stretch over several months, with resulting marked emaciation and loss of strength. The prospects for recovery lessen with the duration of the disease. The stools are discharged at irregular intervals and vary considerably in quality and quantity. The abdomen is often distended as a result of fermentation and putrefactive processes in the intestines, yet it is nevertheless soft, and the umbilicus is not obliterated. The appetite varies and vomiting occurs occasionally. Development of thrush is a frequent occurrence. Fever arises only when complications set in. Furthermore, we note all the results of disturbed absorption and nourishment and the loss of body juices; these include anemia, emaciation, and fatty degeneration of the organs—*atrophia infantum.*

ATROPHIA INFANTUM

(*Pedatrophia*)

Atrophia infantum represents a general wasting disease which arises whenever, for any reason, the digestive, the absorptive, and the assimilative power become inefficient. Pedatrophia may accordingly develop as a sequel to a previous acute gastric catarrh, or primarily in congenital weakness of the digestive function, or in improper feeding, in which cases the gastro-intestinal diseases are not the cause but the result of this condition (Escherich, Concetti). Since the digestive apparatus works at a great loss, the amount of absorbed and assimilated nutriment

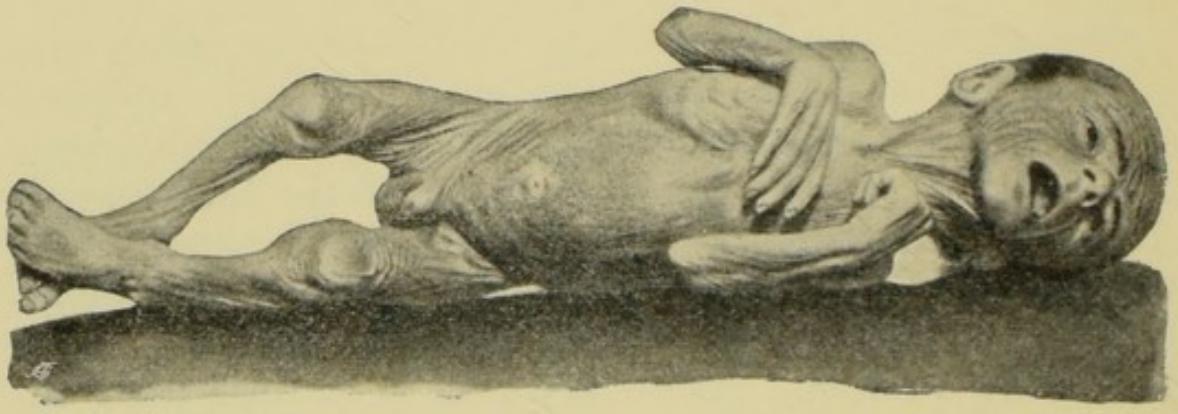


FIG. 118.—Pedatrophia. Five-month's-old child which emaciated to a skeleton from gastro-intestinal disease (weight, 3350 gm.). Movements of defense were barely noticeable (spasmodic seizures of the extremities); the skin is of a dirty color, dry, sallow, and can be lifted in folds. Panniculus adiposus almost completely disappeared. The face presents a wizened expression. The abdomen is retracted and soft. Thrush. Intertrigo of gluteal region.

necessary to meet the requirements of nutrition are no longer satisfied. The organism receives sparse and insufficient compensation for the body material consumed, at any rate not the supply necessary for body growth. The body is finally compelled to attack the supply of energy daily necessary to life which is preserved in its fat deposits—*i. e.*, it gradually wastes away (Heubner). If this disturbance of metabolism continues for a long period of time the following series of symptoms arise in the order they are given, which may, however, vary: Emaciation, loss of vital activity of the organs, and therefore height-

ened predisposition to secondary diseases of the skin, the mucous membranes, the lungs, the kidneys, and of the nervous system; gradual starvation and, finally, death.

Stools.—These depend upon the origin of the chronic affection, and in some cases hardly differ from the normal (so in primary atrophy), whereas in other cases are noted the characteristic dyspeptic, catarrhal, and enteritic stools which rapidly alternate with each other. During the final stage they are brownish, homogeneous, and soup-like.

Prophylaxis of Gastro-intestinal Diseases.—An attempt should be made to prevent the development of gastro-intestinal diseases whenever possible by providing the infant with natural nourishment, that is, the mother's breast milk, otherwise a proper substitute for it by means of fresh cows' milk, which is secured in clean stalls and prepared according to accepted scientific methods. Protect the child from overfeeding or insufficient feeding, and observe the rule that the amount of food administered equals in the first quarter year one-sixth, in the second quarter, one-seventh, in the third quarter, one-eighth of the body weight. (See Nutrition, p. 42.) Weekly determination of the body weight by means of scales is important. We should inform ourselves accurately as to every detail of nursing, ventilation, light, warmth, rest, and cleanliness (also as to the personality of the nurse). Intestinal infection and sepsis may be easily prevented in private practice by observation of the simplest hygienic principles. Wherever a large number of infants and younger children must live in a single room, as in children's hospitals or orphan asylums, the above-mentioned conditions are more likely to occur, and for their prevention far more energetic and expensive measures must be undertaken. A glance at Fig. 119 shows how in modern times it is possible, and with excellent results, to prevent infection by contact by means of the Heubner, Finkelstein, Schlossmann, and other maternity hospitals. The individual beds are isolated by glass walls ("boxes," partially open cells). The material necessary for the nursing of each individual child is usually kept prepared in

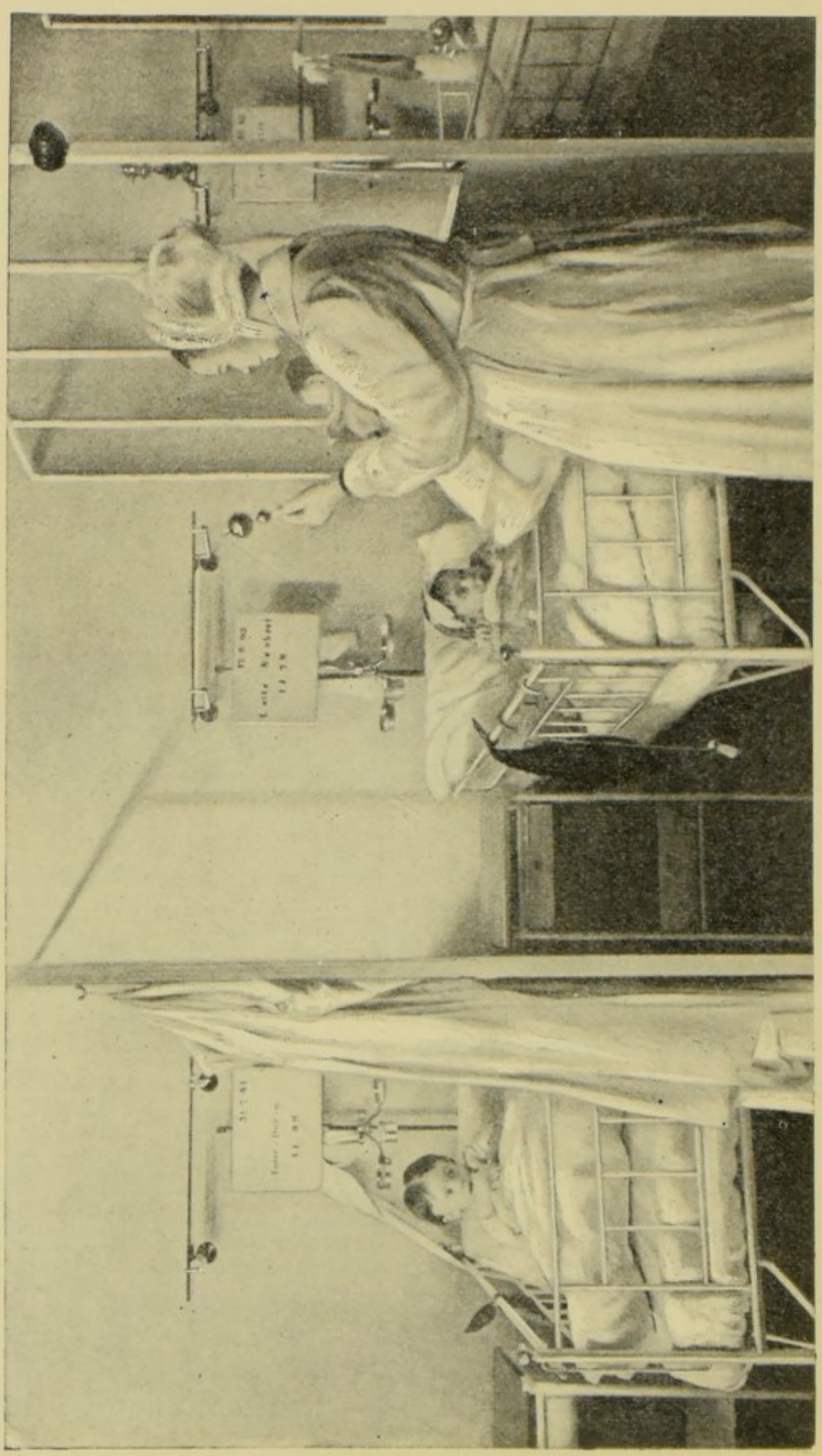


FIG. 119.—"Boxes" in the clinic of Heubner, Berlin.

the same enclosed room (individual drinking, washing, bathing, and night utensils, individual thermometers and other instrumentarium, and individual examination coat for the physician, etc.).

The **treatment** of gastro-intestinal diseases is causal, and it is, therefore, necessary to remove the etiologic factor. In the first place the protective functions of the body must be supported. The organism seeks in the various dyspeptic, catarrhal, and inflammatory affections to discharge injurious ingesta from the stomach and intestines by means of vomiting, increased intestinal excretion, and by peristalsis. We assist nature in recent and acute cases by cleansing the stomach and colon and by means of lavage,[1] and the small intestine by the administration of castor oil (½ coffeespoonful every two hours).

Secondly, as nature requires rest for the diseased organ, anorexia sets in. Accordingly, therefore, depending upon the severity of the case, food is given only at intervals varying between six and forty-eight hours (in cholera infantum intervals of several days); on the other hand, the increased demand for liquids should be satisfied after one-half- to two-hour pauses with smaller or larger amounts of weakly sweetened cold teas or alkaline mineral waters (Schlossmann).

[1] *Gastric Lavage.*—The instrumentarium consists of a Nélaton catheter (No. 18 to 20), a 50-cm. [20-in.] long rubber tube (both united by means of a glass tube), and a glass funnel having a capacity of 30 ccm. [1 fl. oz.]. The child lies with its hips raised upon its mother's lap, who rests her right foot upon a footstool and the left foot upon the floor. The physician introduces the Nélaton catheter by exerting light downward pressure upon the tongue, and passes it for 25 cm. [10 in.] into the esophagus (the alveolar border is 15 to 23 cm. [6–9.4 in.] distant from the cardia at the age of one year). After the gastric contents have been removed the stomach is washed clean with a .6 per cent. sodium chlorid solution at a body temperature.

Intestinal Lavage.—Instrumentarium the same as above, but in place of the Nélaton catheter an intestinal tube, 1 meter [32 in.] long, and in place of the funnel, a graduated irrigator, are employed. The child lies, with its pelvis raised, on its side or on its abdomen. The lubricated intestinal tube is introduced with a gentle pushing movement to such a height in high enemata that the fluid will again pass out beside the tube. The length of the large intestine during the first year of life is 50 to 100 cm [20–40 in.].

Thirdly, we must bear in mind, when normal feeding is again resorted to, that the functional activity of the digestive apparatus has become limited by the effects of the



FIG. 120.—Gastric lavage in infants.

foregoing diseases and, therefore, only a very small amount of nourishment should be given at the beginning. Accordingly, only minute quantities of food are given after

as long intervals of time as possible. During the intervals administer tea or mineral water to satisfy thirst.

In the regulation of *diet* we must next consider whether the affection concerned is associated with acid or alkaline fermentation (albuminous putrefaction). In the first case the diet is begun with egg-albumin-water, thin boiled rice, and rolled oats; later, veal broth may be given. In the case of albuminous putrefaction (*e. g.*, putrefaction of the intestinal secretions) give a carbohydrate diet (Soxhlet's nutritive sugar, infants' foods) or weak cream mixtures. After recovery sets in resort is again had to simple, fresh-milk nourishment. In cholera infantum the supply of liquid necessary for life is maintained by subcutaneous infusion of normal salt solution,[1] and when it is possible to again take liquids *per os* they must be administered at first in very minute quantities.

It is of the greatest importance in pedatrophy to obtain a diet which requires the least amount of digestive energy, and which will be more easily assimilable than the previous form of nourishment. Aside from women's milk, the looked-for results have been attained in mild cases by the use of buttermilk (Texeira de Mattos) or cream mixtures.

Medication is of little use in the treatment of gastro-intestinal disease in infants. Castor oil is generally indicated in the first stages; tannin and bismuth preparations are often very efficient in profuse intestinal secretion (tannigen or tannalbin, 0.25 gm., every two or three hours, bismuth subnitrate or bismuth salicylate in emulsion, 2.0 or 5.0 gm. : 100.0, in coffeespoonful doses every two or three hours). Drop doses of opium (careful dosage) in starch enemata relieve pain and give rest in severe enteritis. In other respects the use of drugs is often useless.

[1] Infusion of 100 to 150 ccm., twice daily, of a sterilized, physiologic salt solution at body temperature; or a solution of sodium bicarbonate 3.0 gm, and sodium chlorid 4.0 gm. : 1000. The solution is introduced underneath the abdominal skin by means of a curved infusion needle, a tube, and funnel, or it is injected by means of a large syringe. Massage of the tumor caused by the injected fluid.

Treatment of Constipation.—If the constipation is a result of muscular weakness the symptomatic use of suppositories or enemata is recommended, also light abdominal massage to strengthen the musculature. In case the

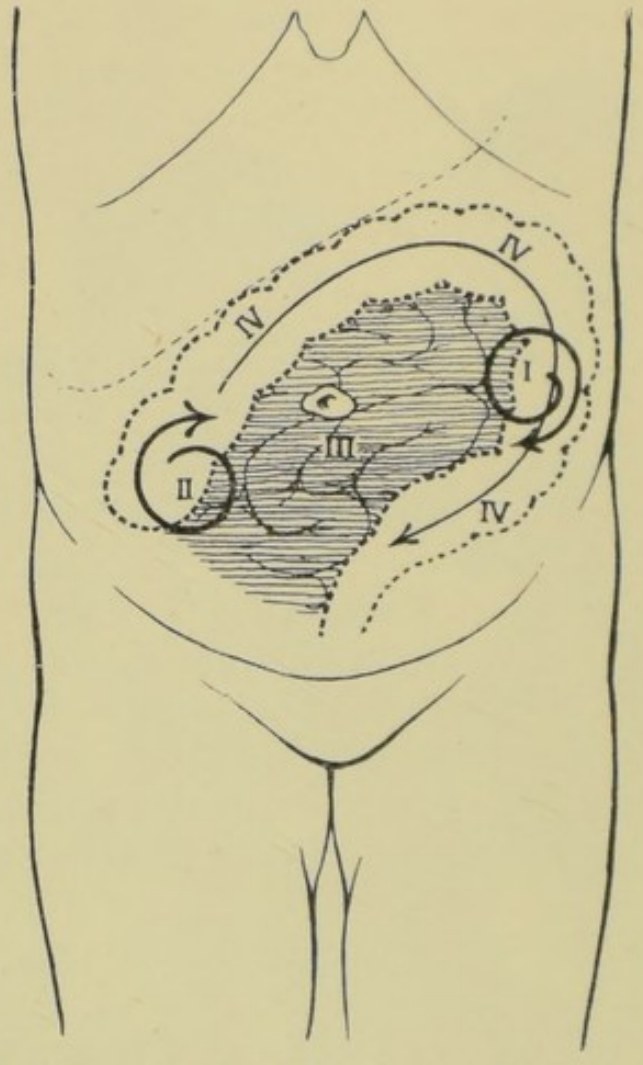


FIG. 121.—Abdominal massage in infants. I. Massage of the descending colon. Rotary movements of the hand, with simultaneous movement of the abdominal walls; pressure being increased in a longitudinal direction over the colon. II. Massage of the ileocecal region. (Same as I.) III. Massage of the small intestine. The hand placed flat upon the abdomen in the region of the umbilicus performs movements by pronation and supination, and pressure is exerted upon the abdomen at one time with the tips of the fingers and at another time with the ball of the hand. Finally, the center of the abdomen is tapped with piano-playing-like movements. IV. Stroking the colon throughout the whole course.

constipation is due to dyspepsia good results can only be obtained from as thorough a change of diet as is possible. If it be due to spastic conditions small doses of opium frequently act very promptly. Rhagades of the anus re-

quire applications with the silver stick. In enlarged colon (Hirschsprung's disease) complete cure may be obtained by means of methodic introduction of oil.

Treatment of Prolapsus Recti.—In Mild Cases.—Adhesive straps placed like tiles over the nates, which are

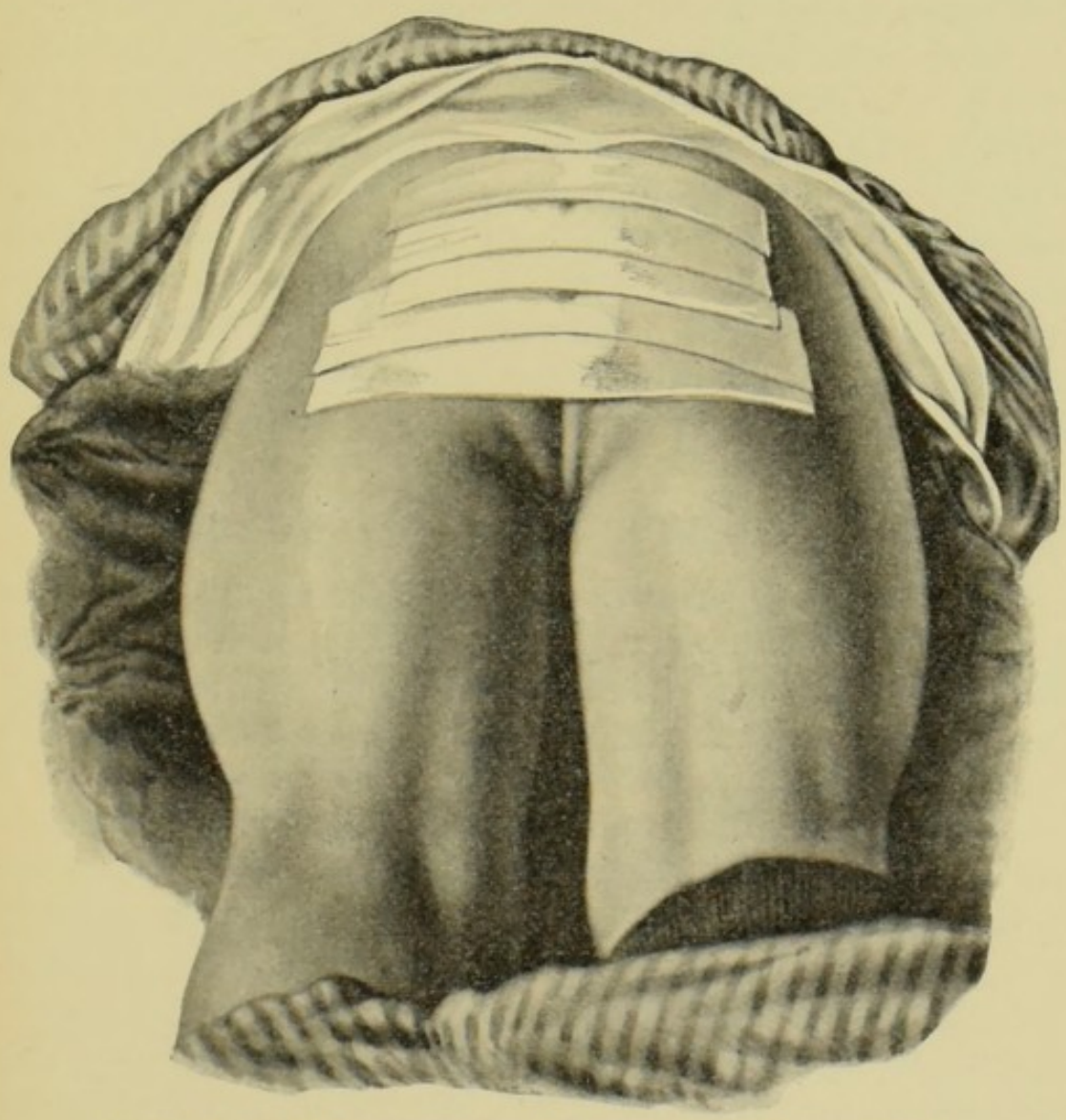


FIG. 122.—Adhesive-plaster dressing for prolapsus recti.

tightly pressed together. The plaster must extend over the perineum.

In Severe Cases.—Fixation of the rectum by means of longitudinally directed injections of paraffin. This is performed according to the method described by Spitzy (Children's Clinic at Graz): Melted hard paraffin (melt-

ing-point 50° to 55° C. [122°–131° F.]) is drawn into a sterilized and heated syringe, which is covered with a rubber tube to prevent the rapid dissipation of heat, and supplied with a straight, not too narrow, hollow needle which measures 8 to 12 cm. [3.4–4.8 in.] in length. The needle is inserted between the coccyx and rectum; the left index-finger is introduced into the rectum and guides the syringe as high up as possible. While the needle is slowly withdrawn about 5 ccm. of the paraffin are injected. The injected material hardens rapidly and when cooled forms an irregular longitudinal ridge which permanently prevents inversion of the rectum without causing any constipation. When carefully performed one injection is sufficient. Ill results are not noticed. The effect is more certain in action and less formidable than the methods formerly practised.

ATONY OF THE STOMACH AND INTESTINES

Atony of various sections of the digestive tract are of great practical interest. In the stomach such a condition is represented by gastric paresis (usually due to overfeeding or improper feeding, and is acquired during the first year of life—Pfaundler). In the intestines, where it is known as "intestinal atony," this condition plays an important rôle in the digestive disturbances of childhood. Anemic and weak girls who are approaching puberty are especially likely to suffer frequently from anorexia and obstinate constipation. The latter may be accompanied by a whole series of toxic and nervous symptoms (migraine, periodic vomiting, intermittent albuminuria, arhythmia of the pulse, and skin eruptions).

Treatment.—Scientific massage of the abdomen, certain exercises (performed on home gymnastic appliances), moist applications and douches upon the abdomen, faradization of the atonic portions of the intestines; later, baths and a vegetable diet. The abuse of purgatives is to be guarded against. Gastric paresis in infants responds rapidly and favorably to limitation of diet and intervals

between feeding. In obstinate cases the gastric contents present after two and a half hours should be removed systematically (without lavage).

APPENDICITIS

Disease of the appendix and its vicinity in children is practically analogous to that in adults. However, greater difficulty in diagnosis is frequently met with in childhood. Of assistance in the diagnosis are: The results of a bimanual examination by way of the rectum and the abdominal wall (painful tumors in Douglas' pouch); examination of the blood (leukocytosis in pus formation); the "facies abdominalis"; the constipation and the detection of an appendiceal tumor by symmetric palpation and percussion of the abdomen. In typic cases this tumor lies midway between the umbilicus and the anterior superior spine of the ilium; frequently, however, it lies deeper in the pelvis at the fundus of the bladder, to the right or even to the left of the median line. In such cases dysuria, which may lead to the mistaken diagnosis of cystitis, is a characteristic symptom.

Treatment.—Early operation during the intervals. Treat the attack as in adults.

CONGENITAL STENOSES AND ATRESIÆ OF THE GASTRO-INTESTINAL TRACT

Many fatal cases traceable to physical and digestive weaknesses are caused by stenosis of the pylorus.

Etiology.—Closure of the pylorus (incomplete) may be due to acquired hypertrophy of the muscularis, more frequently, however, it is due to functional spasmodic narrowing of the opening (Pfaundler). Spontaneous improvement and, finally, complete recovery occur in many cases in spite of most alarming symptoms, including vomiting after each meal and frightful loss of body weight. Recovery may be hastened by high enemata (for the existing constipation). Cataplasm to the abdomen and atropin internally or subcutaneously.

On the whole, the **prognosis** is unfavorable in stenosis and atresia of the intestines, excepting atresia of the anus (absence of communication between the blind end of the large intestine and the external cuticular covering), which is so favorably situated in many cases that it can be easily corrected by operative intervention (see Deformities). Congenital constriction or obliteration of the intestines is due to the development of peritonitis during embryonal

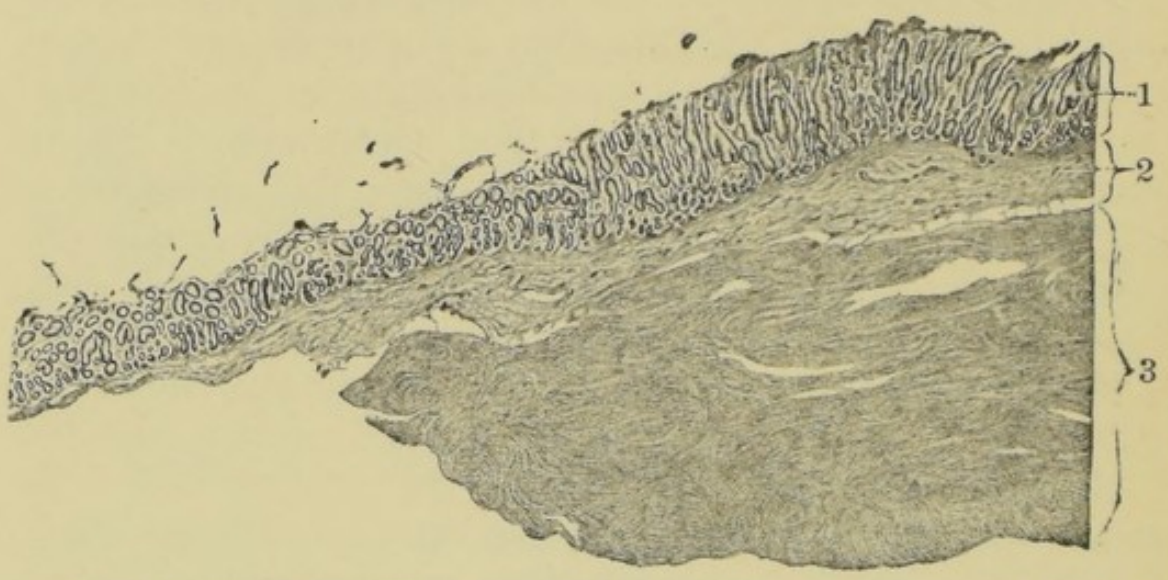


FIG. 123.—Congenital hypertrophy of the pylorus. Enlarged 30 times. The child presented since birth the following symptoms of pyloric stenosis: Vomiting always after the ingestion of food; diminished stools and excretion of urine; protruding and peristaltic contractures of the dilated stomach; finally, a small growth was palpable in the region of the pylorus. Temporary improvement followed. Pegnin milk and extract of belladonna. Gastric lavage was useless. Death occurred in four weeks due to inanition. Post-mortem examination showed circular thickening and hardening of the pylorus, whose lumen was about 3 mm. [⅛ in.] in diameter. 1. Mucosa. 2. Submucosa. 3. Hypertrophy of the muscularis.

life, also twisting and strangulation of the intestines (Epstein), as well as volvulus (Kohts) in a congenital abnormally long colon. This defect develops most commonly in the duodenum, at the end of the ileum, and where the descending colon passes into the S loop. In atresia, continuous vomiting (of food, bile, and blood) sets in as early as the first day of extra-uterine life, followed by death. In permeable stenoses the term of life depends upon the degree of constriction. Dilatation of the

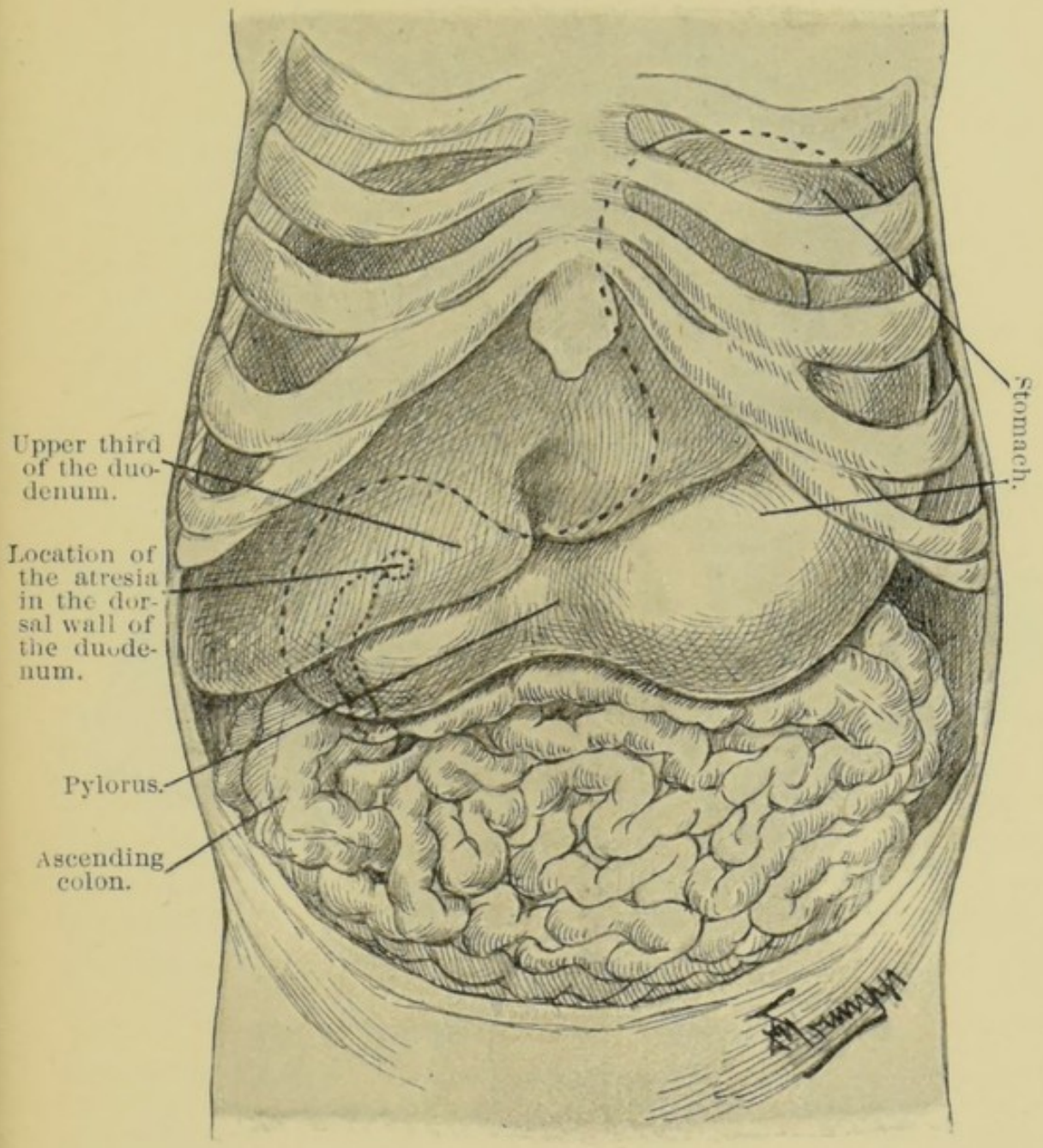
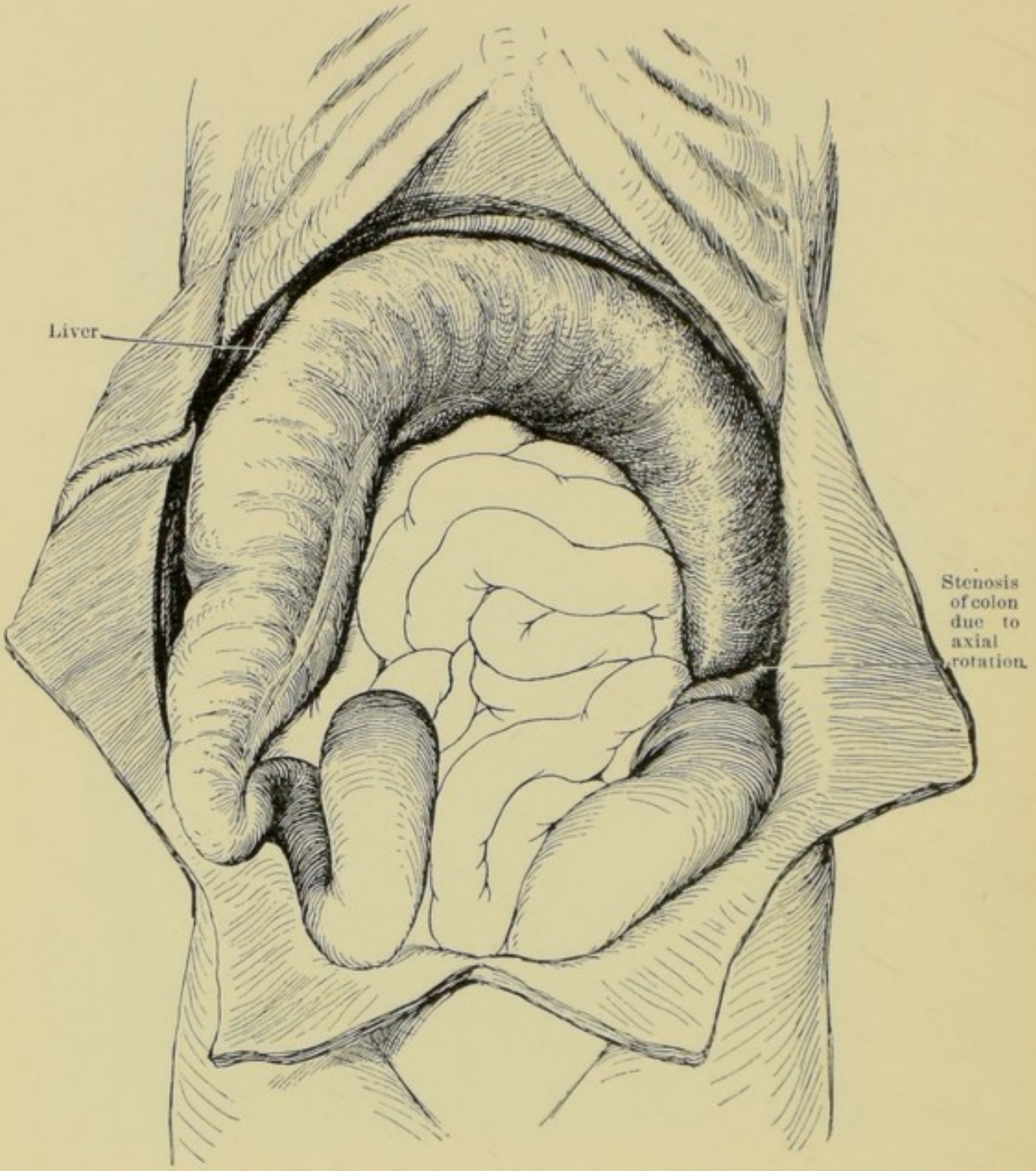


FIG. 124.—Congenital infrapapillary atresia of the duodenum. Twin child four days old. Premature birth (seven and a half months). After birth the child had two passages of traces of meconium, since then no stool. Nutrition decidedly disturbed; bloody discharge from the nose. Icterus, sclerema, lobular pneumonia. Albuminuria. Death fourteen hours after consultation. *Necropsy.*—Abdominal cavity: Enormous dilatation of the stomach and upper duodenum; a ring-shaped constriction between the two (pyloric valve). The middle section of the duodenum is converted into a white, fibrous, solid cord, in the upper end of which the papilla of Vater projects into the duodenum, which is here barely open. From this point a probe may be easily passed through the common duct and that of Wirsung. From the point of constriction downward the intestine is completely collapsed and empty.



FIGS. 125, 126.—Congenital gastric and intestinal stenosis and congenital displacement of the colon in an infant five months old. The abdomen was markedly distended since birth; vomiting; meconium was passed only after intestinal lavage. Breast fed. Curdled milk was not passed until the tenth day and then only after lavage. About every four weeks spontaneous, at first diarrheic and then gruel-like formed, stools were noted, and in the course of a few days the obstinate constipation was renewed, with vomiting at intervals. The spontaneous stools were preceded for several hours by violent pain. In the performance of intestinal lavage the tube met an unsurmountable obstruction about 22 cm. [8.8 in.] above the anus. Careful general and local treatment managed to keep the child in a fairly good state of nourishment for four weeks, but after that the health began to steadily fail. The child, who had become extremely weak through chronic inanition, died in five months

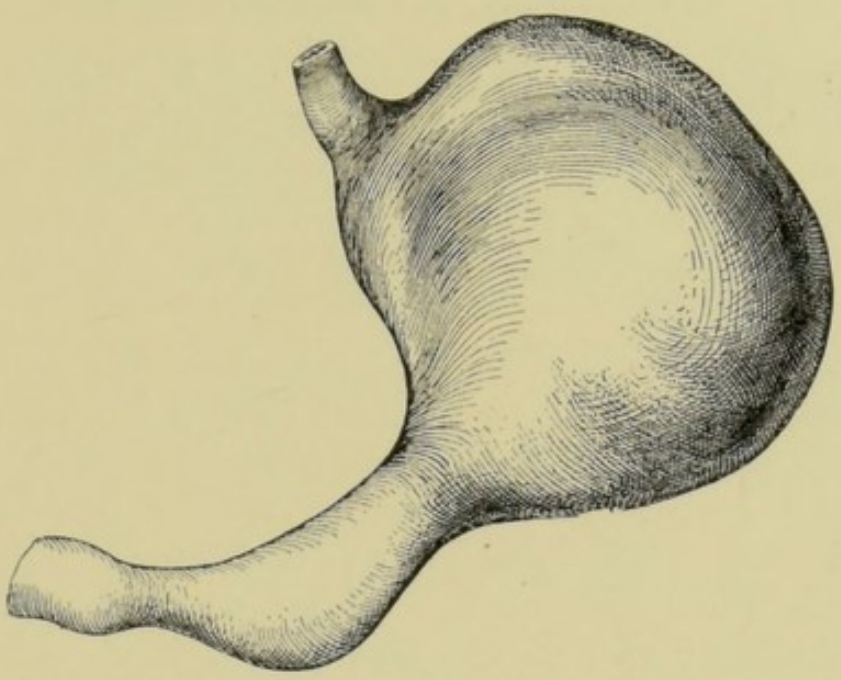


FIG. 126.—(See description under Fig. 125.)

intestine above the stenotic area always sets in primarily; later, hypertrophy of the musculature and, finally, paralysis of the latter and perforative peritonitis develop. The clinical symptoms are obstinate in spite of all treatment directed to the constipation, and death occurs often surprisingly quick under the symptoms of peritonitis or intercurrent diseases. Laparotomy may be attempted.

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from acute enteritis. *Necropsy.*—Abdomen: No signs of peritonitis. The colon was abnormally elongated and presented an abnormal course; at first it extended downward into the true pelvis and then, with a double loop, it passed laterally toward the right pelvic crest and upward. It stretched, markedly distended, in a uniform curvature from right to left along the lower border of the thorax and pushed the liver backward and downward, the stomach backward and upward, and made a semirotation on its long axis. Where the colon passes into the S flexure it was fixed by a peritoneal covering, and decidedly constricted by a complete turning on its axis. In the region of constriction the folds of half of the circumference of the intestines were arranged longitudinally. The pylorus was almost vertical in position; the stomach was elongated and constricted in two places: Directly before it enters the duodenum; at the border between the ventricle and the pylorus, especially upon the side of the greatest curvature. In consequence of these stenoses the fundus of the stomach was prematurely developed; the musculature was hypertrophied, and especially so at the stenotic areas. The three divisions of the stomach may be easily recognized—fundus, body, and pylorus.

INTESTINAL INVAGINATION

Invagination of the intestines is rather frequently observed in children, especially during the first year of life. It consists in the invagination of a contracted section of the intestines into a neighboring relaxed portion; the cause is unknown. The ileocecal region represents the site of predilection.

Symptoms.—The cardinal symptoms of this constantly serious disease are: Constipation; vomiting; passage of blood and collapse. The intussusceptum is frequently palpable as a movable hard tumor in the left and, less rarely, in the right side of the abdomen. The child usually dies from peritonitis. Spontaneous resolution occurs occasionally and, rarely, recovery follows sloughing of the necrosed intussusceptum. The so-called *agonal invagination*, which is frequently found accidentally at necropsy, is of no significance clinically.

INTESTINAL PARASITES

Parasites, such as oxyuris, ascarides, and tenia, are found in the intestinal tracts of about 40 per cent. of all children. The general health is often not disturbed by them for a long time, but at times we note abdominal pain, nausea, vomiting, itching of the nose and anus, and convulsions. If the condition persists for a long time, anemia and nervous irritability arise.

The **diagnosis** is determined by the discharge of parasites, or sections of them, or by the microscopic detection of their eggs.

The **Oxyuris vermicularis**, the thread- or spring-worm, is whitish yellow in color, spindle shaped, and measures from 3 to 10 mm. [$\frac{1}{8}$–$\frac{1}{2}$ in.] in length and 0.5 mm. [$\frac{1}{50}$ in.] in thickness. Its eggs have thin walls, oval in shape or flattened at the sides. The female deposits its eggs in the folds of the anus, which produce an itching that requires constant scratching; through the latter the fingers become infected with the eggs. This, leading to

autoreinfection, causes the oxyuriasis to become an extremely persistent disease.

The *treatment*, therefore, consists, in the first place, of absolute cleanliness of the anal region and the prevention of scratching. Give enemata of thymol or 1 coffeespoonful three times a day of santonin with castor oil, 0.2 : 60.0 ; or, better, 0.15 to 0.4 gm. of naphtalin in eight doses during two days, to be repeated after an interval of from one to two weeks (strangury) [Ungar].

The **Ascaris lumbricoides**, or round-worm, is grayish yellow or grayish red in color, measures 20 to 40 cm. [8–16 in.] in length, 0.5 cm. [0.2 in.] in width, and is pointed at the extremities. Its eggs show dark granules and have a thick concentrically striped membrane. The entrance of this parasite into the common duct, the pancreatic duct, or the appendix may excite an obstinate icterus and other inflammatory phenomena.

Treatment.—Trochisci of santonin or castor oil with santonin (0.2 to 0.25 gm. : 40.0) in coffee- or tablespoonful doses.

The **Trichocephalus dispar**, or whip-worm, is yellowish white in color, measures about 2 to 3 cm. [.8–1.4 in.] in length, and is pointed at the anterior end, but thickened at the posterior. It may cause a large number of enteritic symptoms.

Tenia mediocanellata is the commonest form of tapeworm in children and follows the ingestion of raw beef. It is 4 or 5 m. [12 or 15 ft.] long, shows a fine dichotomous division of the uterus, and its head is supplied with four suckers and no crown of hooklets.

Tenia solium develops in the body from the ingestion of raw pork. It is 2 to 3 m. [6–9 ft.] in length, narrower and thinner than Tenia mediocanellata, and its head is supplied with hooklets. The uterus shows dendritic division.

Treatment.—Freshly prepared extract of the male fern, 2.0 to 5.0 gm. ; kamala, 1.5 to 5.0 gm. ; flower of kusso, 8.0 to 15.0 gm. ; Kussein "Merck"; Helfenberg's tapeworm remedy (extract of fern with castor oil) ; extract of pumpkin seed given in three doses. In young children

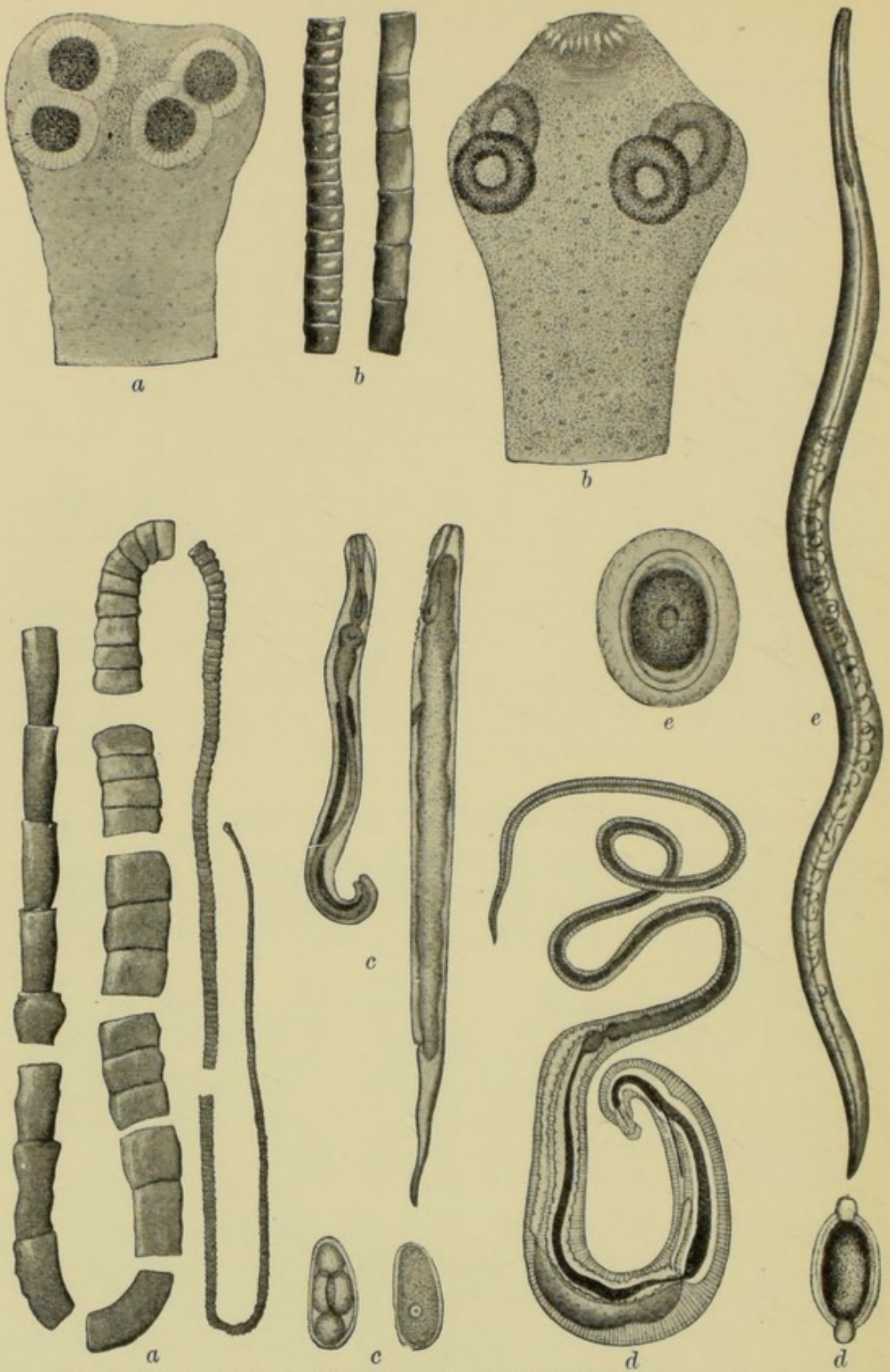


FIG. 127.—Intestinal parasites. *a*. Tenia mediocanellata (head, segments). *b*. Tenia solium (head, segments). *c*. Oxyuris vermicularis (male, female, eggs). *d*. Trichocephalus dispar (male, egg). *e*. Ascaris lumbricoides (female, eggs). (From Ziegler's *Text-book of General Pathology*.)

administer the latter through an esophageal tube. Care must be observed in its use in children before three years of age. The above treatment is to be followed by doses of castor oil. The cure may only be considered accomplished when the head of the tapeworm has been discovered in the stools.

DISEASES OF THE LIVER

ICTERUS

Aside from the physiologic *icterus neonatorum* and the symptomatic icterus due to septic infection in newborn infants, we also observe, but not rarely, jaundice following congenital defects, such as obliteration of the gall-bladder or bile-ducts. The latter forms run a rapidly fatal course accompanied by symptoms of severe disturbances of digestion and of the general health. Catarrhal icterus in children is comparatively rare considering the frequency of acute digestive diseases. The course is the same as in adults.

DISEASES OF THE PERITONEUM

ACUTE PERITONITIS

Acute peritonitis occurs primarily in septic infection of the newborn, also in traumatism, but it occurs far more frequently secondarily to intestinal ulceration, perityphlitis, intussusception, and certain infectious diseases, especially typhoid fever, dysentery, scarlet fever, etc.

The clinical **symptoms** and the morbid anatomy are essentially like those of acute peritonitis in adults.

The **prognosis** is comparatively favorable in traumatic peritonitis, but always bad in the septic forms. The older the child the better are the prospects.

CHRONIC PERITONITIS

Chronic peritonitis is nearly always of a tuberculous nature (see Tuberculosis), yet undoubted cases of chronic peritonitis are met which are not of such a character. It

is either a terminal stage of acute peritonitis or it arises primarily following traumatism, exposure to cold, or as a sequel to measles. A serous or serofibrinous exudate is found in the abdominal cavity, also manifold adhesions of intestinal loops due to inflammatory foci in the peritoneum, as well as thickening of the peritoneal coat of the intestines and the parietal peritoneum. The contracted omentum is also thickened.

Symptoms.—This disease leads to disturbances of nutrition; obstinate constipation alternates at intervals with an apparently groundless diarrhea. The child undergoes emaciation, turns sallow, and the skin and hair become dry, the latter brittle. The appetite and spirits show great variations. The large distended abdomen presents a marked contrast to the emaciated body. Excepting an uncomfortable feeling, subjective pains are rarely present.

Prognosis.—The prognosis is, on the whole, favorable. A complete cure follows careful nursing, yet intercurrent diseases, especially of the respiratory organs, represent serious complications.

Diagnosis.—In reaching a conclusion as regards the diagnosis it is of primary importance to exclude tuberculosis, then cirrhosis of the liver, echinococcus, and all diseases of the heart, lungs, and kidneys which are accompanied by marked disturbances of circulation, as well as diseases of the abdominal glands and other tumors.

Treatment.—The child must be provided with the most favorable hygienic circumstances of life. As much outdoor life, fresh air, and sunshine as possible (observe care in the employment of sun-baths as regards the amount of exposure). A non-irritating diet corresponding to the age and strength of the patient. General massage. Systematic rubbing with soft soap. Hydrotherapeutic measures according to the circumstances. Mild Carlsbad cure. External and internal administration of iodin preparations.

DISEASES OF THE GENITO-URINARY TRACT

DISEASES OF THE KIDNEYS

GENERAL DISCUSSION

THE metabolic processes in the growing organism require an increased functional activity of the kidneys, upon which the regulation of metabolism chiefly depends, hence the increasing predisposition of these organs to disease. Only slight differences are exhibited between the individual renal diseases of adults and children. However, in adults we observe chronic renal disease following more frequently chronic intoxications and general constitutional diseases; whereas in children acute inflammatory conditions predominate, which are chiefly due to infectious processes.

It is necessary in the *diagnosis* of renal disease to depend more upon the examination of the urine in children than in adults because of the absence or of the insufficiency of subjective symptoms and because of the difficulty in reaching the kidney in the physical examination of children. Therefore the chemic and microscopic examination of the urine must never be neglected in all affections which might cause nephritis.

To obtain the urine in infants we employ the apparatus recommended by Hecker (Fig. 128), which prevents the backward flow of the urine from the glass and is applicable for either male or female.

ALBUMINURIA

The presence of albumin in the urine is, as a rule, indicative of pathologic changes in the kidneys or in the deep urinary passages. In rare cases, however, a slight

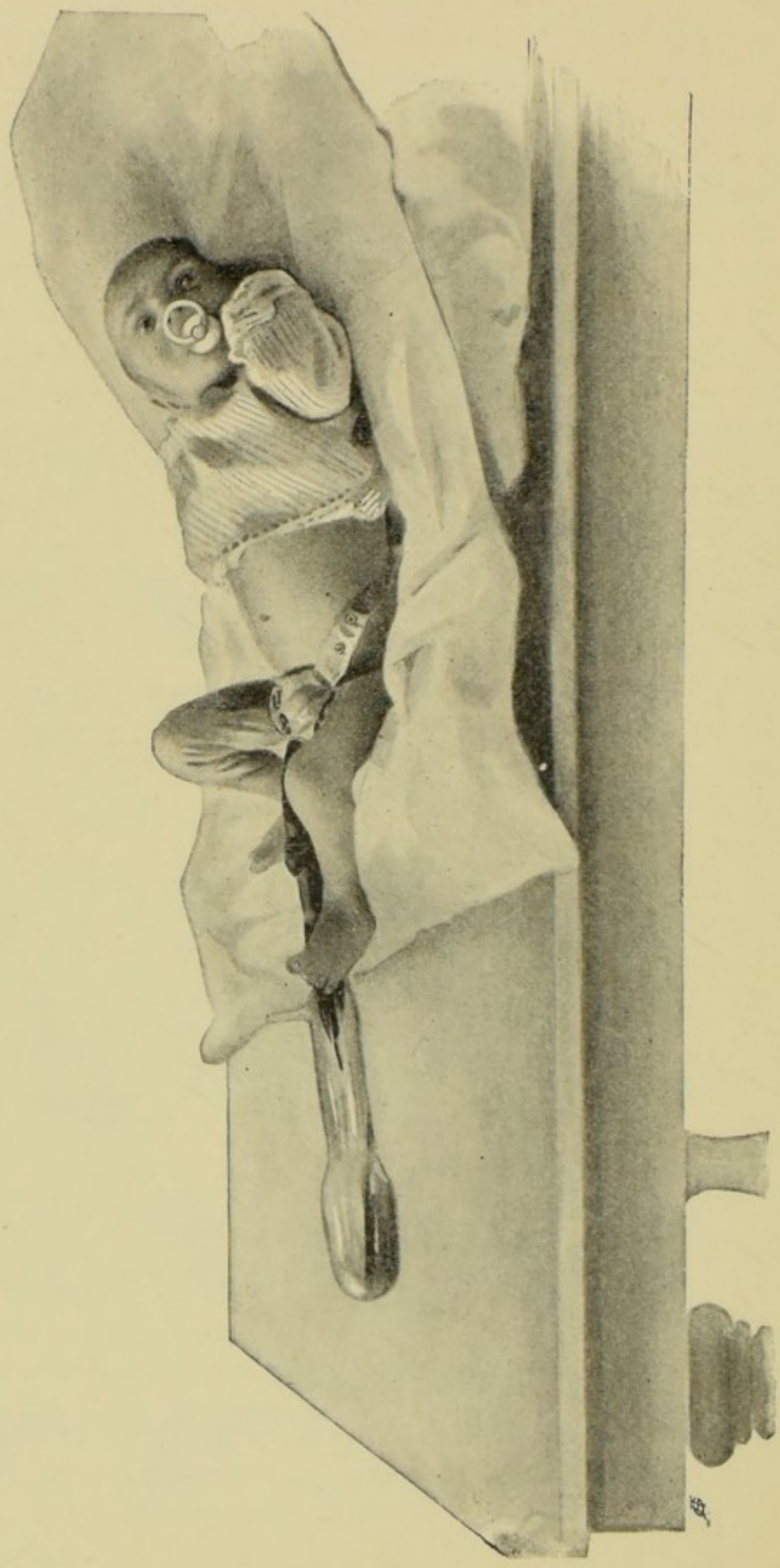


FIG. 128.—Hecker's urine vessel for infants.

transitory albuminuria is met with without demonstrable cause, especially after physical or mental overexertion, after cold baths, and after the ingestion of food containing an overabundance of albumin. Furthermore, an *intermittent* (cyclic) *albuminuria* is occasionally seen in girls or boys at the period of puberty, which depends probably upon congenital renal weakness and which may persist for years without producing any ill effects. It is noteworthy that this albuminuria occurs only when the recumbent is changed to the erect posture, on account of which the albuminuria of puberty is also designated as the *orthotic albuminuria* (Heubner). This type of albuminuria is distinguished from that of chronic nephritis by the absence in the urine of casts and blood-cells (Pribram). In making a diagnosis it must be borne in mind that a temporary albuminuria is also produced by pollution, masturbation, and menstruation.

HEMATURIA AND HEMOGLOBINURIA

Hematuria.—Blood is found in the urine because of the presence of red blood-cells in lithiasis, hemorrhagic inflammation of the kidneys or bladder, tuberculosis, hemorrhagic diathesis, and after trauma. The blood may therefore arise from various portions of the urinary apparatus. The urine, which is strongly albuminous, is turbid and possesses—according to the amount of blood present—a color varying between that of meat juice or blackish red. In hemorrhage from the urethra or bladder large blood-clots are present, whereas in hemorrhage from the kidneys the blood can only be demonstrable microscopically and blood-casts will be present.

Hemoglobinuria.—In hemoglobinuria the urine contains only hemoglobin and no red cells; this condition is noted in Winckel's disease, poisoning with calcium chlorate, extensive burns, and occasionally in congenital syphilis, scarlet fever, and diphtheria. The urine is clear, of a varnish color, similar to that of Malaga wine, and gives the blood reaction as strongly before as after filtration.

Hemoglobinuria is sometimes spasmodic, in which case syphilis or a preceding attack of scarlet fever are at fault; a similar condition at times follows exposure to cold or after physical fatigue (paroxysmal hemoglobinuria).

ACUTE PARENCHYMATOUS NEPHRITIS

The most frequent of the organic renal affections in childhood life is acute diffuse nephritis. It, like most forms of nephritis, is hematogenic and chiefly toxic-hematogenic in origin, and therefore always attacks both kidneys, usually to a fairly uniform extent. In the majority of cases a late action of the virus of scarlet fever is concerned, and less frequently the action of the diphtheritic toxin, pyogenic cocci, and other bacteria and bacterial poisons which are concerned in the various acute and chronic infectious diseases. Poisonous medicaments may also excite inflammatory irritation of the kidneys (internally, potassium chlorate, lead, and mercurial preparations; externally, carbolic acid and styrax). The relationship between nephritis with extensive inflammations and suppurative processes of the skin, intestinal disease, and colds is as yet unexplained.

Morbid Anatomy.—The kidneys in acute diffuse nephritis are enlarged, hemorrhagic, and contain a rich supply of blood, the medullary portion is hyperemic, the cortex enlarged; in advanced cases they are yellowish gray or speckled, their markings obliterated, and their glomeruli are prominent as dark red or gray granules. On microscopic examination the glomeruli show epithelial proliferation, on account of which the loops of the blood-vessels are compressed or obliterated, or the phenomena of hemorrhagic inflammation are seen (necrosis). The dilated uriniferous tubules show cloudy swelling, fatty degeneration, and desquamation of the epithelium, containing blood-cells and coagulated masses of albumin (casts). The interstitial tissue presents inflammatory changes in the blood-vessels and foci of round-cell infiltration.

It is noteworthy, depending upon the nature, the intensity, and duration of the injurious factor, that in one case a certain tissue element, while in another a different element, is attacked with especial severity. Thus, in scarlet fever, changes in the blood-vessels, particularly of the glomeruli, predominate, while in diphtheria the epithelium of the uriniferous tubules is most markedly involved, and processes of a septic nature are mainly characterized by inflammatory foci in the interstitial tissues (Heubner).

Symptoms.—Symptoms referable to nephritis are a result of a disturbance of circulation interfering with the functional activity of the kidneys, which in mild cases, however, are overshadowed by the symptoms of the primary disease. They are frequently limited to a moderate fever, gastric phenomena, moderate edema of the face and knuckles, and the characteristic findings in the urine. The amount of urine is diminished. It is discharged after much straining in small quantities, is cloudy, dark, and, on account of the admixture of blood, either of a meat-juice color, brownish red, or blackish. The reaction is acid, the specific gravity high, and it always contains albumin, blood-cells, and an abundance of casts (granular, hyaline, epithelial, and blood-casts), renal epithelium, and fatty detritus.

The less common serious forms are accompanied by chills and a fever as high as 40° C. [104° F.], headache, vomiting, severe pain in the renal region, oliguria varying in amount from 150 to 100 ccm., and even anuria, edema of a more or less marked degree, and the collection of exudates in body cavities, together with dyspnea. The pulse is of high tension, diminished in frequency, and may be arhythmic. The skin has a wax-like pallor.

Course.—Improvement and recovery with increased excretion of urine and disappearance of albuminuria may gradually set in, even in the severest types, after a duration of weeks and after repeated relapses. Life may, however, be threatened by extensive circulatory interference, and by the retention of the urine and its poisonous meta-

bolic products in the collecting tubules. Such disturbances include dilatation and hypertrophy of the left ventricle, edema of the glottis, lungs, and brain, and also uremia (coma and convulsions).

Prognosis.—Inasmuch as cases which are mild at the beginning may rapidly change to severe forms of nephritis, the prognosis must always be dubious, yet it is, in general, more favorable in children (excepting infants) than in adults. The conversion of acute into chronic forms is fairly rare. Possible complications are pneumonia, pleuritis, endo- and pericarditis, and meningitis.

The **diagnosis** can only be made with certainty when the urine constantly contains a marked amount of albumin and when the sediment presents an abundance of casts; also when the amount of urine is diminished simultaneously with the occurrence of albumin and blood in the urine, with the subsequent development of dropsy. The single symptoms, like dropsy, albuminuria, and hematuria, occur also independently of nephritis, as in heart and pulmonary disease and in anomalies of the blood.

Treatment.—Rest in bed; diet should consist largely of milk; increase diuresis with lemonade and mineral water, and diaphoresis by means of hot baths and other sweating procedures. For the uremia cause depletion by way of the intestines; ice-cap to the head, enemata of chloral, lumbar puncture (Seiffert), and, if necessary, venesection (Baginsky) followed by infusion of normal salt solution (Leube). In hemorrhagic nephritis, ice-cap to the renal region. A child's spoonful of the infusion of ergot (2 : 100) every two hours.

CHRONIC NEPHRITIS

The various types of chronic parenchymatous nephritis have occasionally been observed even in children. Its origin may sometimes be traced with a certain amount of surety to a preceding infectious disease, but in other cases the etiology is not clear and the course is so atypic that it is difficult to classify the condition under the known divi-

sions of renal diseases. Of the more characteristic forms, the interstitial type, *contracted kidney*, is more common than the parenchymatous form, *large white* or *swollen kidney*. In the former case the urine is increased in amount, its specific gravity is lower than normal, and the amount of albumin is slight, whereas in the latter type of renal disease the urine is sparse in amount, saturated with albumin, casts, and blood.

Amyloid degeneration of the kidneys is comparatively rare in children (*lardaceous kidney*). It occurs in association with amyloid degeneration of the spleen and liver, prolonged suppuration of the bones, chronic pulmonary and glandular tuberculosis, and syphilis. The urine obtained by catheterization is pale and contains a large amount of albumin. Profuse diarrhea and persistent dropsy are usually present.

The **prognosis** of the contracted and of the white kidney is absolutely fatal, but that of the amyloid kidney depends upon the duration of the causal condition.

Treatment.—A mild and not too strict dietetic régime. Avoid exposure to cold; hot douches and warm baths. As diuretics, employ caffein, digitalis, potassium acetate, or camphor.

URINARY CONCRETIONS

Uric-acid infarcts in the newborn are frequent in the first days of life and usually disappear when sufficient fluid is ingested. This may become so excessive during the first days of life because of the small amount of urine secreted that the collection of uric-acid salts and free uric acid in the straight uriniferous tubules leads to the formation of uric-acid infarcts. The irritation due to the collection of nitrogen-containing excrementitious material causes the kidney to become hyperemic, and as a result of the circulatory disturbances albumin is excreted (euglobulin). Examination of the urine, which is decreased in amount, shows small quantities of albumin off and on during the first two weeks of life, few hyaline casts and epithelium, as well as yellowish-red granular

portions of the infarcts, that is, uric-acid crystals. The organ presents the changes of hyperemia and yellowish-red striations in the pyramids.

Nephrolithiasis.—Renal sand, gravel, or calculus forms with especial frequency in children during the first year of life, and in the pelvis as well as in the parenchyma of the

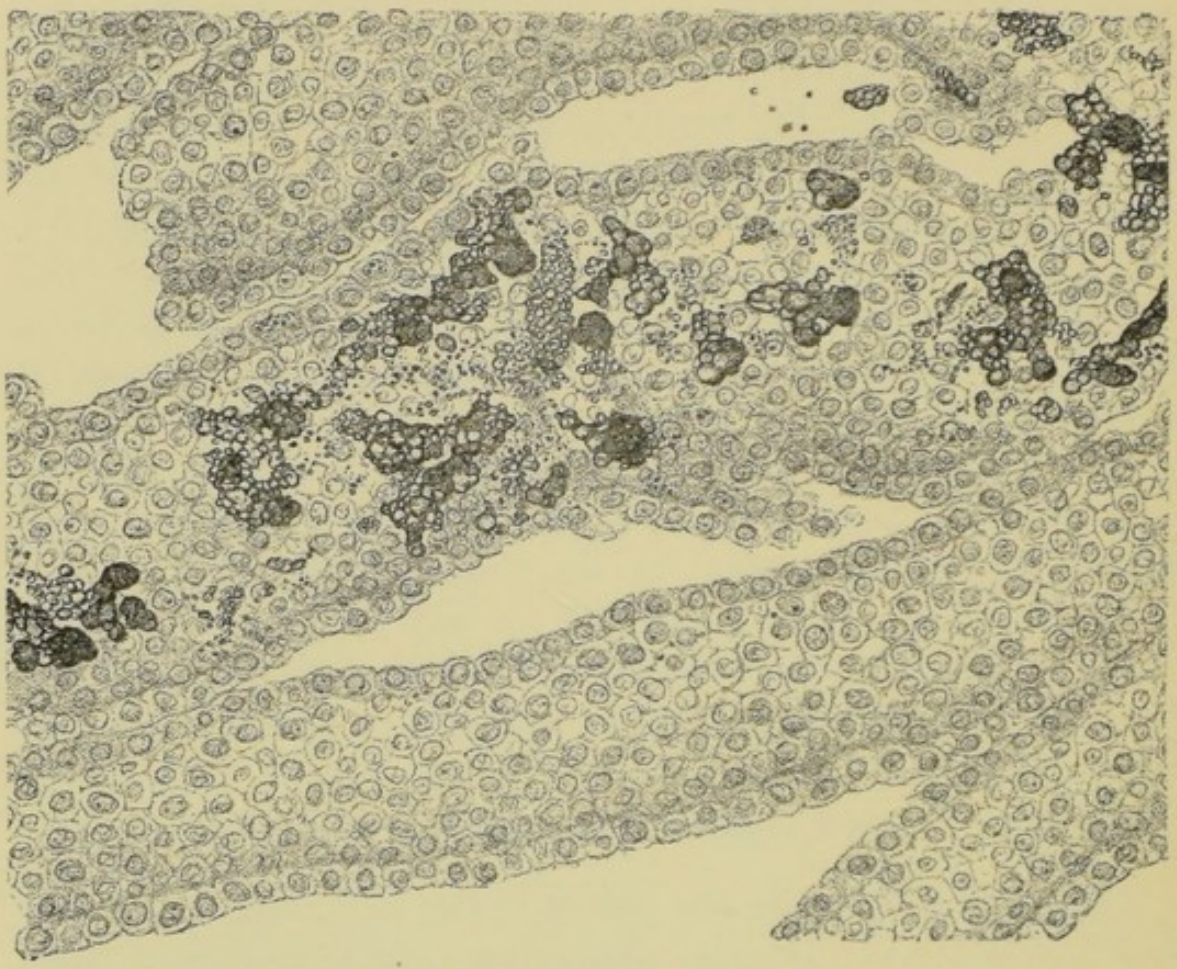


FIG. 129.—Uric-acid infarcts in the kidney of a newborn infant. A freshly isolated uriniferous tubule from the medulla, which is partially filled with spheric and gland-like concretions. Enlarged 280 times. (From Dürck, *Atlas of General Pathologic Histology.*)

kidney. As etiologic factors we have over- and insufficient feeding (especially with food rich in nitrogen), profuse discharge of body juices, and marked loss of tissue —vomiting and diarrhea, atrophy (Comby). The concretions consist mainly of free uric acid and uric-acid salts and, more rarely, of calcium oxalate, cystin, and phosphates. Renal sand and gravel usually effect no manifestations of disease, and the renal calculi only after they

have reached a certain size (lentil to bean size). Then they may call forth hemorrhages and inflammatory processes in the renal pelvis or parenchyma (pyelitis, pyelonephritis); or, in case of difficult passage through the ureter, renal colic and even hydronephrosis may be caused.

Chief Symptoms.—Restlessness or pain upon micturition. Pains in the region of the kidney radiating toward the bladder. Frequent discharge of small amounts of bloody urine containing an abundance of sediment. Passage of concretions.

Treatment.—A diet poor in nitrogen; massage and gymnastics, carbonate and alkaline waters. For calcium-oxalate stone give sodium phosphate (2 to 10 per cent.), for phosphatic concretions administer citric acid. Hot applications to relieve colic.

Vesical Calculi.—Vesical calculi are equally as frequent as renal calculi, especially in boys up to ten years of age (40 per cent. of all cases of lithiasis). The nucleus of the stone is formed, as a rule, by uric-acid concretions washed out of the kidneys; less rarely they are formed primarily in the bladder from the sediment of alkaline urine. The process is favored by mechanical obstructions to the outflow of the urine, as in phimosis and similar conditions. The color, form, and size of the stones vary considerably; in rare cases they may fill almost the whole bladder (the stones enlarge in the bladder by the deposit in concentrically arranged layers of cystic urinary sediment).

Symptoms.—The disturbances caused by cystolithiasis are partly mechanical and partly inflammatory in nature: Displacement of the neck of the bladder by a stone may lead to more or less severe disturbances of micturition. The irritation of the mucous membrane by the stone produces vesical catarrh and inflammation.

Characteristic Phenomena.—Temporary pains, radiating toward the perineum and glans penis, are caused mainly when the body is moved or shaken; frequent discharge of feces and urine; frequently sudden stoppage of the

urinary stream, after which the urine is passed only in drops or, indeed, it cannot be passed at all for hours or even days (emptying of the bladder is sometimes possible when the posture is altered or, if spasm of the bladder exists, in a hot bath); in some cases in place of retention we observe incontinence of urine. The condition of the urine: Sometimes clear, at other times turbid and containing cystic sediment, or also blood and fragments of concretions. Noteworthy in cystolithiasis of children is the frequent tendency to *prolapsus recti* and the inclination of boys to manipulate the penis (often elongated) during retention of urine (Henoch). Small concretions may obstruct the urethra, prevent the passage of urine, and produce painful infiltration of the perineum, scrotum, and penis.

The *diagnosis* is made certain by examination with the sound (metallic sound, clicking).

The *prognosis* is doubtful. If of long standing, marasmus results. The development of ascending nephritis is by no means a rare event and the danger of uremia may threaten life. Stones possessing a rough surface may occasion deep-seated ulceration of the vesical mucous membrane and, later, pericystitis and fatal perineal abscesses.

Treatment.—As in case of nephrolithiasis. Removal by operation as soon as possible.

PYELITIS

(*Pyelonephritis*)

Inflammation of the mucous membrane of the pelvis of the kidney and of the renal pyramids followed by consecutive inflammation of the renal tissue is also observed in children, particularly as complications of cystitis (*ascending nephritis*), nephrolithiasis, and after scarlet fever. The disease runs a course similar to that of adults. A serious sequel is the development of renal abscess.

HYDRONEPHROSIS

Renal concretions, anomalies in position of the kidney, anomalies in the formation of the ureters (abnormal length), or disease of neighboring organs may displace a ureter and prevent the flow of the urine into the bladder on the affected side. In consequence the urine is dammed back above the obstruction, the renal pelvis and the upper portion of the ureter become dilated, and marked compression of the kidney substance results. If the condition is not relieved the renal pelvis may finally form a cyst the size of an adult's head (in congenital hydronephrosis birth is interfered with), in which only remnants of the kidney, which has been atrophied from pressure, can be found. When the hydronephrosis is extensive the lumbar region on the affected side presents an immovable fluctuating tumor over which the percussion-note is dull, and tympanitic if the colon lies above it. Dyspnea, constipation, and shooting pains in the legs develop as the condition progresses. As the healthy kidney assumes the duties of the diseased one no symptoms of general disturbance set in. Radical operations are followed by permanent recovery. If, however, the other kidney becomes diseased, death soon follows the development of edema and uremic symptoms.

DISEASES OF THE BLADDER AND SEXUAL ORGANS

POLLAKIURIA AND ENURESIS

Two varieties of disturbances of the bladder are frequently observed early in life and during the years of puberty. In one, called *pollakiuria*, the urine is passed voluntarily, but frequently abnormally. In the second form, called *enuresis*, the urine is passed involuntarily at night and, less rarely, during the day.

Pollakiuria is generally combined with nocturnal enuresis. This disturbance may depend upon hysteria or neurasthenia (weakness of the central inhibitory mechanism) or it may be of reflex origin, as in phimosis,

balanoposthitis, lithiasis, bacteriuria, oxyuriasis, adenoid vegetations, etc. This anomaly rarely persists beyond puberty, and the general health remains uninfluenced, aside from the psychic depression seen in older children.

Treatment.—Remove the cause. Institute general hygienic, dietetic, and hydrotherapeutic measures (limit liquids, one-half minute treading of water in the evening, elevation of the end of the bed, awaken the child at regular intervals during the night). Tonics, such as tincture of nux vomica. In hysteria, temporary effect is frequently obtained through painful procedures, such as the use of the faradization brush; subcutaneous injections; the passing of bougies. More recently it has been recommended to use automatically ringing electric bells to control and cure enuresis (Pfaundler, Hutzler).

CYSTITIS

Catarrh and inflammation of the vesical mucous membrane is influenced in children as in adults by mechanical, chemic, bacterial, and other forms of irritation, as by vesical calculi, certain medicaments, and bacteria which have invaded the bladder. The entrance of micro-organisms into the bladder may be an accompaniment to various inflammatory affections of the genito-urinary apparatus or of its neighboring organs. Bacteriuria may, however, exist without producing inflammatory irritation of the bladder or without constitutional symptoms. The most frequent form of cystitis—colicystitis (Escherich)—in childhood, especially during the nursing period, is usually a secondary manifestation of vulvovaginitis or inflammation of the colon. In these conditions the Bacillus coli passes from the infected external genitalia through the urethra into the bladder, and perhaps also by way of the blood- and lymph-vessels through the damaged intestinal epithelium and pelvic connective tissue. The infected urine primarily causes only mild symptoms of irritation (bacteriuria), later, catarrhal and inflammatory changes of the vesical mucous membrane (cystitis), and, finally, the passage of the bacteria through the upper

urinary tract causes a marked purulent inflammation of the pelvis of the kidney and, indeed, of that organ itself (pyelitis, pyelonephritis, ascending suppurative nephritis).

The urine in colicystitis is cloudy and turbid, similar to a bouillon culture of bacteria; its reaction is acid, odor foul, and a slight amount of albumin is present. It contains a large number of colon bacilli and pus-cells, less commonly, red cells and desquamated epithelium; casts are absent. In consequence of the irritation the urine is passed frequently and in small amounts. Locally, the region of the bladder is sensitive on pressure. The remaining clinical symptoms, fever and disturbance of the

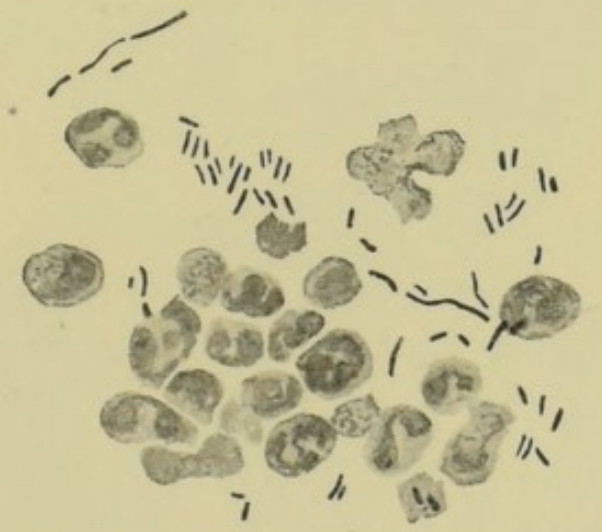


FIG. 130.—The urine in colicystitis.

general health, vary considerably according to the intensity, duration, and extent of the process. Pronounced cases may run a chronic, intermittent course, which stretches over many weeks and months; or the disease may end fatally, due to an ascending nephritis.

Chief Characteristics.—Dysuria; absence of casts in the urine, which is rich in pus and bacteria. In the presence of diphtheria and articular rheumatism we should consider the possibility of a diphtheritic or tuberculous cystitis.

Treatment of Colicystitis.—Rest in bed; a non-irritating diet; urotropin or salol (0.25 to 0.5 gm) three to five times a day.

PREPUTIAL EPITHELIAL ADHESION

In newborn boys a partial or total epithelial adhesion exists normally between the inner layer of the prepuce and the glans penis. Dysuria develops if the urethral orifice is partially covered, and the retention and degeneration of the smegma (the result of stagnation of urine) causes irritation and inflammation accompanied by a purulent secretion—*balanoposthitis.* The adhesions are easily loosened with a blunt sound; they may also disappear spontaneously within the first year.

PHIMOSIS

Epithelial adhesion of the prepuce is frequently combined with constriction of its inner layer (more rarely also the outer)—*phimosis.* The results of this anomaly, which

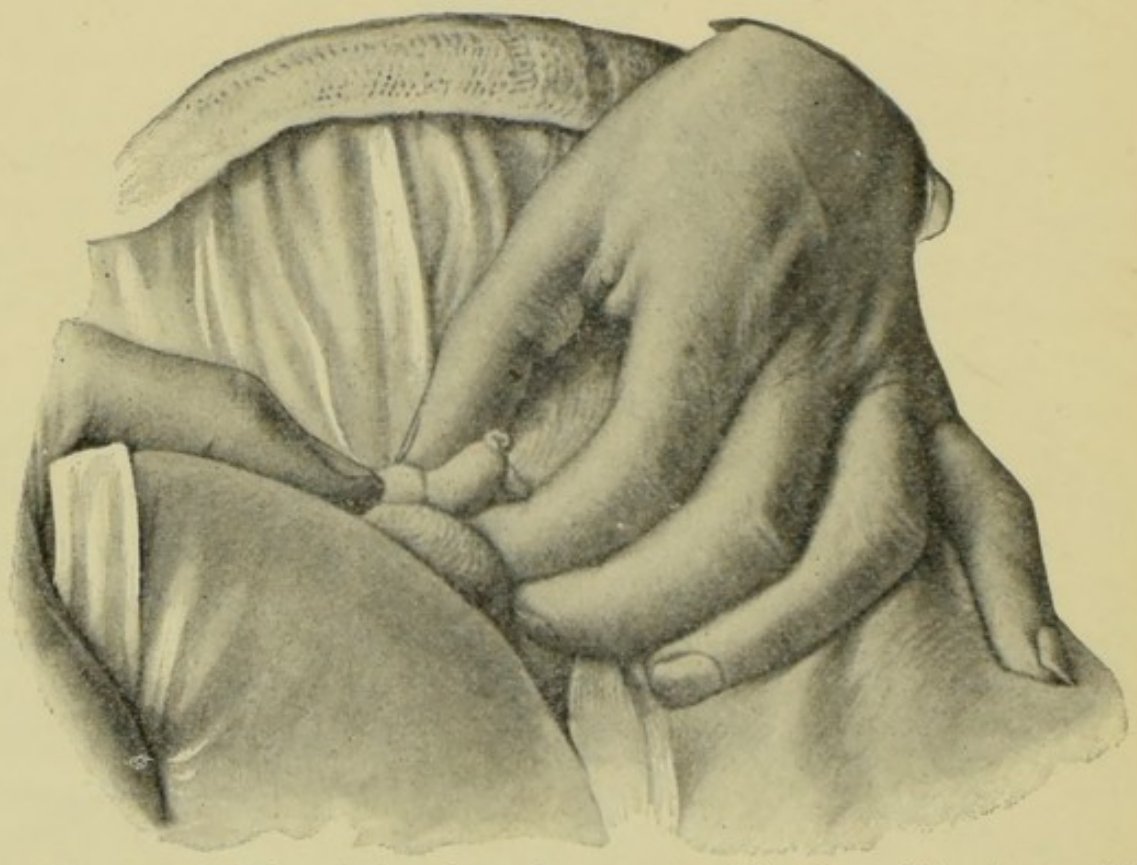


FIG. 131.—Phimosis in a boy three years old. Reposition impossible on account of marked constriction of the inner preputial layer (hour-glass shape).

is secondary to inflammatory conditions—balanitis, balanoposthitis—or congenital in origin (in which case the pre-

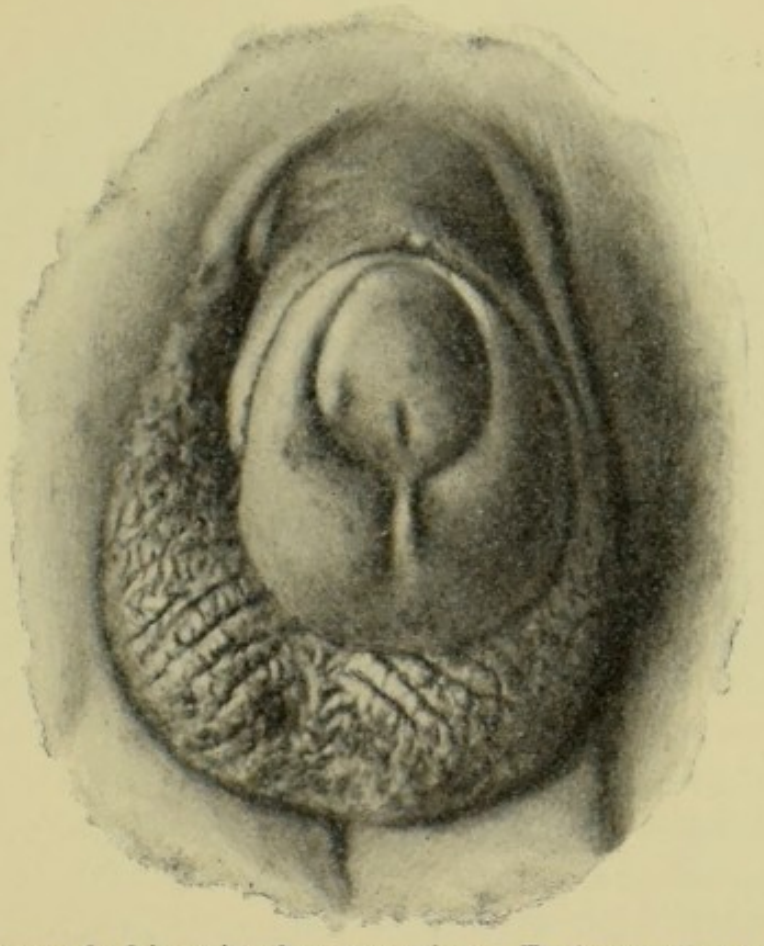


FIG. 132.—Case of phimosis after operation. Postoperative edema (third day). Prepuce divided to the retroglandular sulcus. No suture.

puce is usually elongated and thickened), are to a certain extent the same as in preputial adhesion and consist of

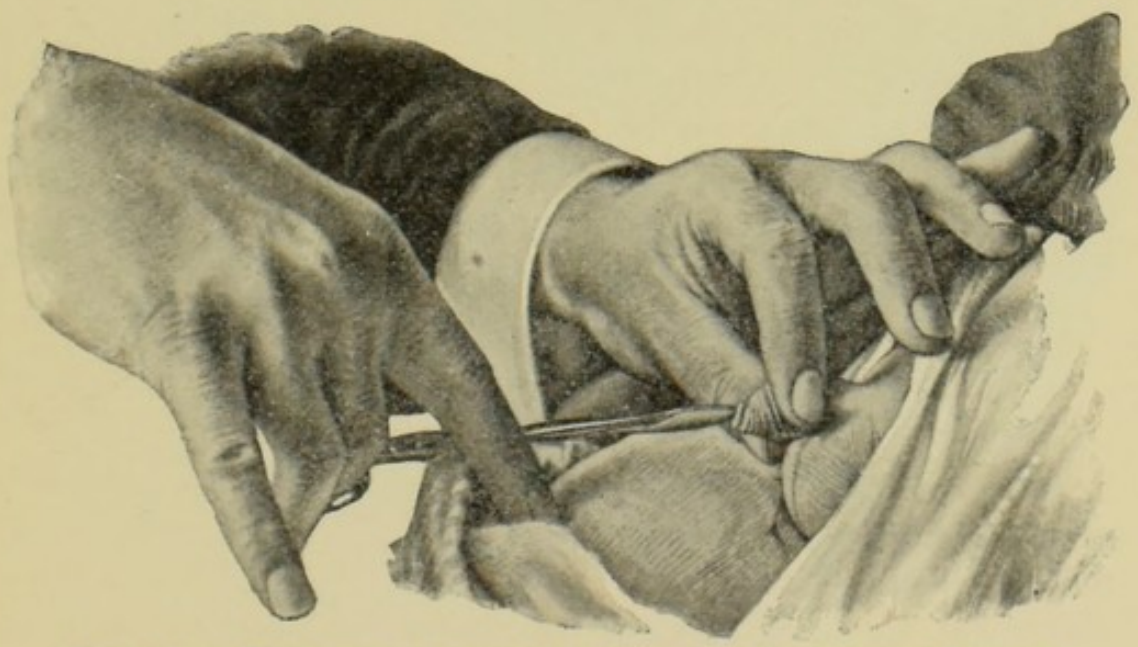


FIG. 134.—Phimosis. Attempt to stretch with dressing-forceps.

dysuria and balanoposthitis. Excessive straining may lead to the development of hernia and prolapsus ani. Triad: Phimosis, umbilical hernia, hydrocele. Increased tendency to lithiasis and masturbation also exists. Congenital phimosis of a moderate grade usually disappears spontaneously with the growth of the penis. On account of the sequelæ a marked constriction requires an early operation.

In cases in which a ring-shaped constriction occurs when the foreskin is drawn back, the outer and inner preputial membranes should be divided on a grooved director as far back as the retroglandular sulcus. Suture is unnecessary. Dress with aluminum acetate solution and, after a few days, with airol paste. Elevate the penis to lessen the postoperative edema which always develops.

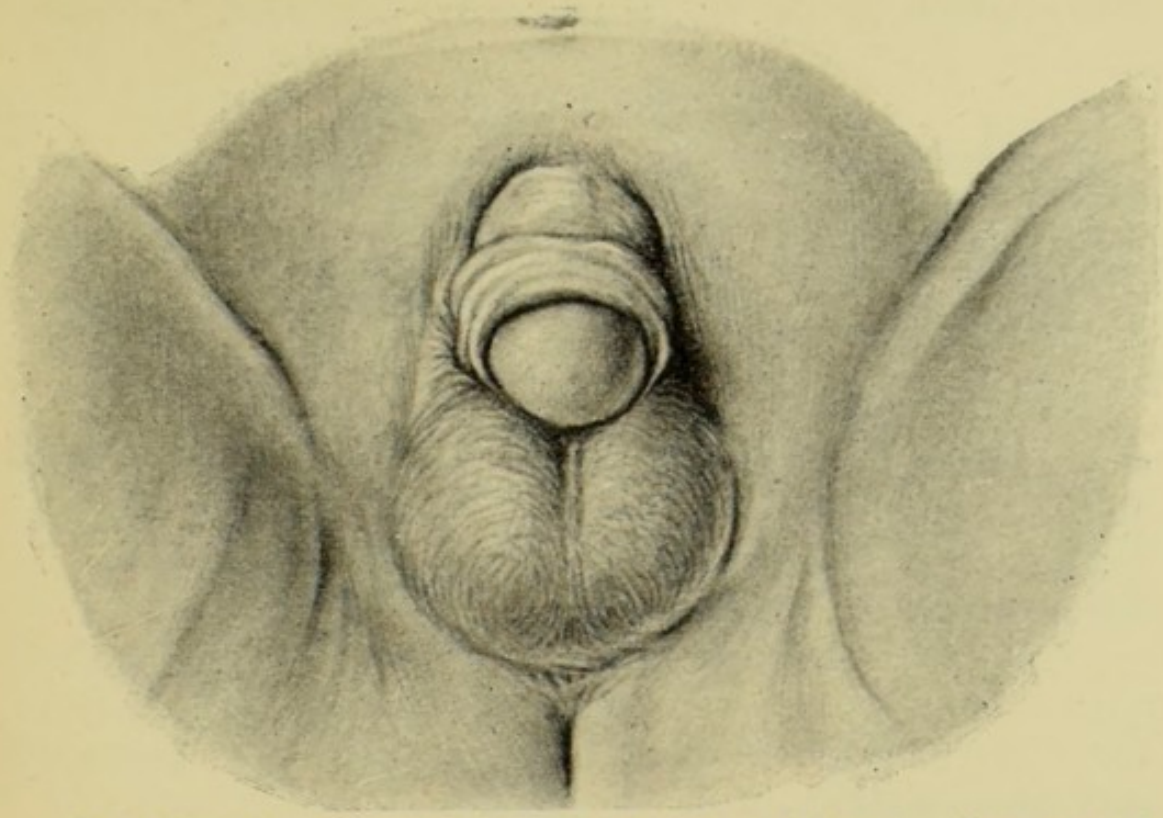
In all other cases the prepuce may be drawn back without a bloody operation after a preliminary loosening, which is made especially easy in a hot bath or in narcosis (later stretch with dressing-forceps). Eczema of the preputial border is a contra-indication.

HYPOSPADIAS. EPISPADIAS

Hypospadias and epispadias represent a congenital incomplete fissure formation either on the ventral or on the dorsal surface of the penis, in which the urethra is partially closed or present in a rudimentary form. The penis is usually very short and the glans fairly well developed. The resulting disturbances of function may become troublesome in later years. The treatment is operative.

UNDESCENDED TESTES

Ectopia Testis.—If for any reason during the sixth or seventh fetal month the beginning of the descent of the testes to the scrotum is prevented, they remain either above the inguinal ring in the abdominal cavity or in the ring itself (if one-sided, we have *monorchism*; if bilateral,



FIGS. 135, 136.—Hypospadias. Dorsal surface of penis. Boy one and a half years old. Penis 2 cm. [.8 in.] long. Glans penis is disproportionately large. The urethra is an open groove 13 mm. [½ in.] long, which extends from the scrotum to the glans. Where this groove approaches the glans penis it flattens out. By means of a plastic operation the penis was made longer and the orifice of the urethra was formed at the tip of the glans.

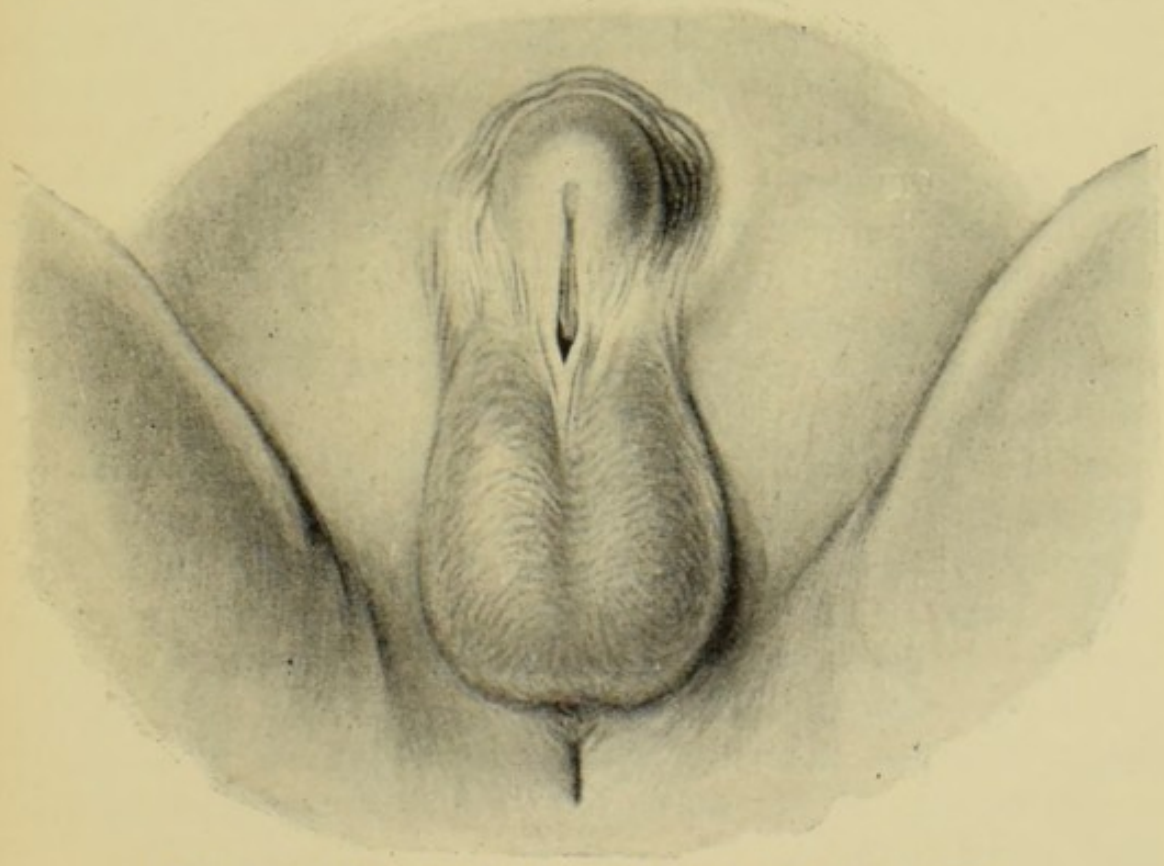


FIG. 136.—Hypospadias, ventral surface of the penis.

FIGURE 133

Descent of the Testes (according to Stieda-Pansch).—**a.** Position of the testis at about the fourth fetal month. b. Position of the testis at about the sixth fetal month. c. Position of the testis in the ninth fetal month. Development of the peritoneal vaginal process. d. Position of the testis at birth. Development of the true tunica vaginalis. (From Sultan, *Atlas of Abdominal Hernias.*)

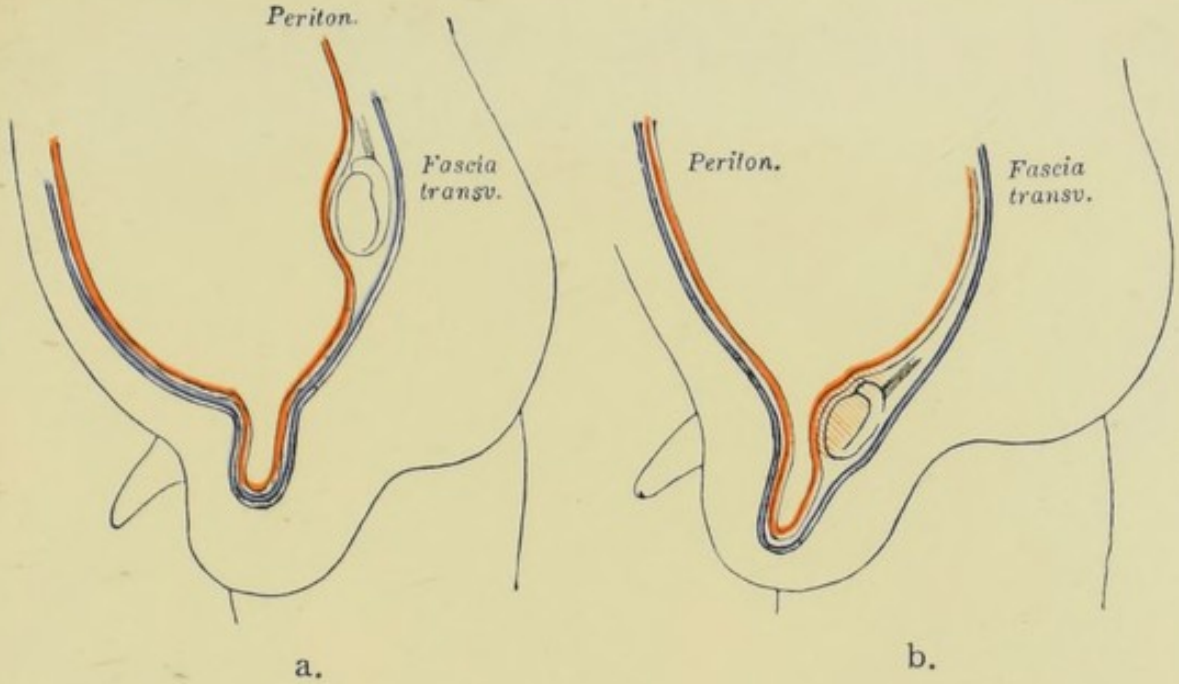
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cryptorchism), or they miss the route to the inguinal ring and pass under the skin, Poupart's ligament, or peritoneum (*ectopia testis*). Permanent retention of the testes causes degeneration of these glands.

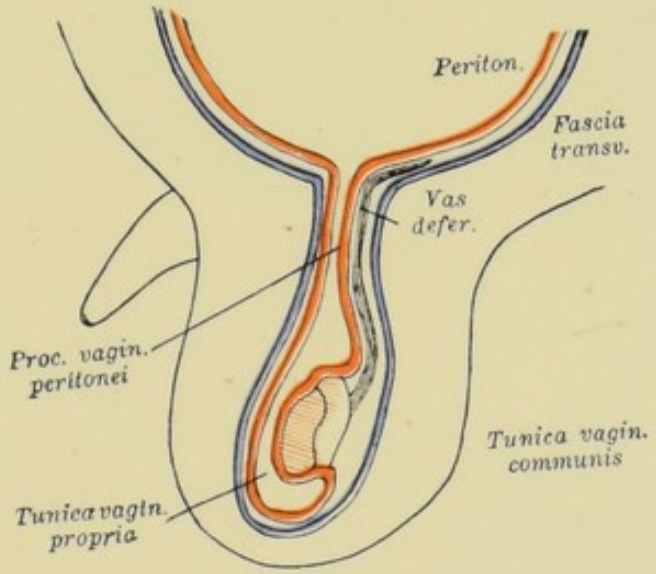
Treatment.—As soon as the testes become palpable they may gradually be forced downward by means of a hernia-truss-like bandage which is supplied with a fork-shaped pad opening downward.

HYDROCELE

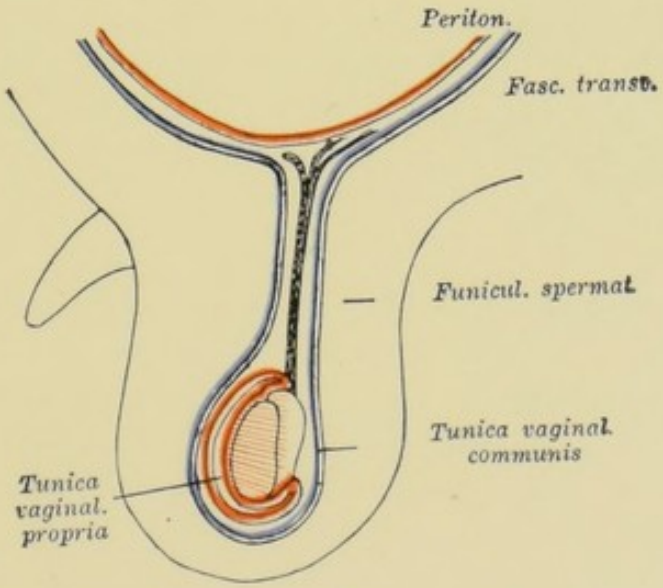
The term hydrocele designates a collection of serous transudate in the true tunica vaginalis of the testes or in the vaginal process which is not yet closed (hydrocele communicating with the abdominal cavity). Hydrocele is either congenital or acquired and develops acutely or in a chronic manner. In hydrocele of the testes the swelling, which is either unilateral or bilateral, lies within the scrotum and has the shape of an egg or a pear, is smooth, tense, fluctuating, transparent, and not displaceable, excepting in *hydrocele communicans*. The testis lies back of the mass. In funicular hydrocele of the spermatic cord one or more oval, round, or spindle-shaped tumors will be observed above the testis. This condition is distinguished from inguinal hernia by the position of the testis back of the mass, by the tenseness, transparency, and percussion-note, by the inability to displace it, and by the absence of enlargement on coughing and straining. Spontaneous recovery occurs usually in from three to six weeks; otherwise employ iodin preparations, puncture and extirpate.


Periton.
Fascia transv.
Periton.
Fascia transv.

a. b.


Periton.
Fascia transv.
Vas defer.
Proc. vagin. peritonei
Tunica vagin. communis
Tunica vagin. propria

c.


Periton.
Fasc. transv.
Funicul. spermat.
Tunica vaginal. communis
Tunica vaginal. propria

d.

Fig. 133.

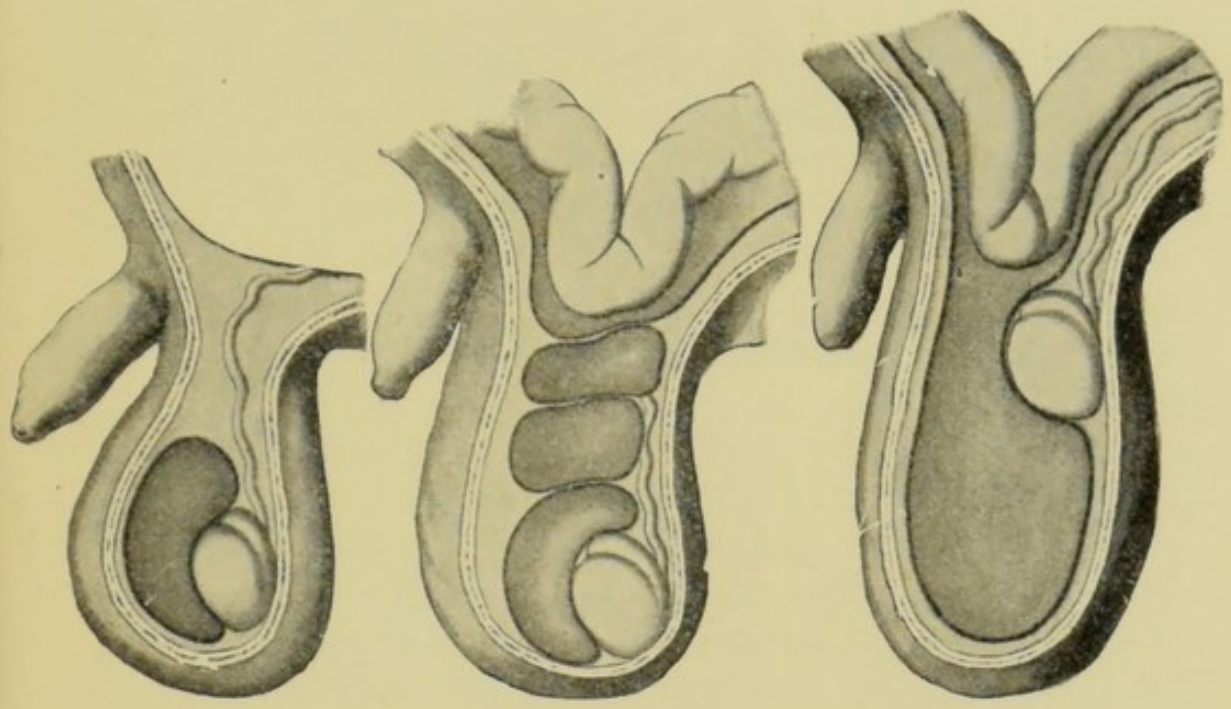


FIG. 137. FIG. 138. FIG. 139.

FIG. 137.—The usual picture of hydrocele of the testis. "The true tunica vaginalis is stretched by a collection of fluid, over which the peritoneum is smoothly drawn."

FIG. 138.—Hydrocele of the testis, funicular hydrocele of the spermatic cord, and inguinal hernia. "The vaginal process of the peritoneum has become adherent in various places, on account of which several pouches, one above the other, were formed. At the base of the scrotum is a hydrocele of the testis, above two hydroceles of the spermatic cord, which are joined to the hernial sac."

FIG. 139.—Hydrocele communicans and secondary inguinal hernia. "The vaginal process of the peritoneum failed to close on account of the incomplete descent of the testis. A hydrocele communicans developed, which also became the hernial sac because of the entrance of a coil of intestine. This condition is also termed *hernial hydrocele*." (From Sultan, *Atlas of Abdominal Hernias.*)

CELLULAR ATRESIA OF THE VULVA

Analogous to the preputial adhesion in boys, we occasionally meet with adhesion of the labia minora in girls, which may lead to retention of the urine. Loosen with a sound or knife.

VULVOVAGINITIS. GANGRENE AND PHLEGMASIA OF THE VULVA

Inflammation of the external genitalia in girls occurs frequently as a result of infection with gonococci, pyogenic staphylococci or streptococci, or the diphtheria bacillus. It may also be due to non-specific affections, uncleanliness, the entrance into the vulva of oxyurides or intestinal bacteria, the introduction of foreign bodies for masturbation, or weakening conditions like anemia and chlorosis.

The non-specific catarrhal vulvovaginitis is generally accompanied by a slight whitish, mucoid discharge from the vagina. The local disturbances are mild. The affection disappears after the removal of the cause. Gonorrheal infection is usually due to carelessness of a mother or nurse infected with gonorrhea, but may sometimes follow rape, and in rare cases it develops intrapartum. The infectiousness of the condition is great and often leads to sudden spreading of the disease to all the members of a family or to all the inmates of a hospital ward.

Symptoms.—The vaginal lips are red and swollen and, like the entrance to the vagina, covered with pus. Pressure on the perineum causes a discharge from the vagina and, frequently, also from the urethra of a thick, yellowish-green pus in which gonococci are more or less abundant.

Involvement of the urethra causes dysuria. Subjective symptoms may be absent. Recurrences are common. Gonorrheal cystitis is not an uncommon complication of gonorrheal urethritis.

The **diagnosis** is insured by finding the characteristic micro-organisms.

Treatment of **gonorrheal vulvovaginitis** may be limited to rest in bed, a non-irritating diet (milk and vegetable food), thorough cleanliness of the vulva, and sitz-baths in a decoction of oak bark. Intravaginal irrigation by means of Nélaton's catheter with a lukewarm disinfectant solution under slight pressure is indicated in many cases. At

the beginning use 4 per cent. boric acid, .5 to 1 per cent. protargol, or .2 per cent. itrol; later, 2 to 4 per cent. aluminum acetate or .5 per cent. zinol. Beware of auto-infection (ophthalmoblennorrhea) and contact infection! The non-specific form of vulvovaginitis after the cause has been removed heals generally in a few days by means of cotton tampons soaked in a solution of aluminum acetate.

Phlegmon and **noma** of the vulva and vagina which follow infection with pyogenic cocci develop at times in very badly neglected children, in trauma, and in infectious diseases which run a malignant course (scarlet fever, diphtheria, measles, and typhoid fever). The constitutional or local symptoms, as in phlegmonous or gangrenous involvement of any mucous membrane, are of a severe type. Energetic and antiseptic management and the administration of a strengthening diet are imperative. (For Diphtheria of the Vulva, see that disease.)

DISEASES OF THE SKIN

GENERAL DISCUSSION

DISEASES of the skin are very common in children and, indeed, far more frequent in infants than in older children or adults. On the one hand, they are the expression of the extraordinary sensitiveness of the infantile skin toward various—and to a certain extent—trivial external injurious influences, and on the other hand, the manifestations of an existing dyscrasia, a result of disease of the digestive apparatus or of the nervous system.

The *treatment* must, therefore, not be purely symptomatic, and great stress should be placed upon the discovery of the causal disease. Furthermore, the tenderness of the infantile skin must be borne in mind and when possible only bland remedies are to be employed, which, when properly used, will prevent further injury to the skin and lead to a cure in most cases.

NEVI

Nevi, or *mother's marks*, are congenital anomalies of development. We distinguish between pigmentary and vascular nevi. Pigmentary nevi are referable to excessive deposition of pigment in the rete Malpighii. Their color varies between light brown and black. The skin itself may remain unchanged and its surface smooth—*nevus spilus;* or the skin may be wart-like or rough and supplied with coarse hairs—*nevus verrucosus;* or a pronounced tumor-like thickening of the skin exists covered thickly with hair—*nevus pilosus.* In rare cases the pigmentary nevi involve a whole region of the body.

Vascular nevi are due to abnormal blood-vessel growth and are congenital in origin or acquired at an early

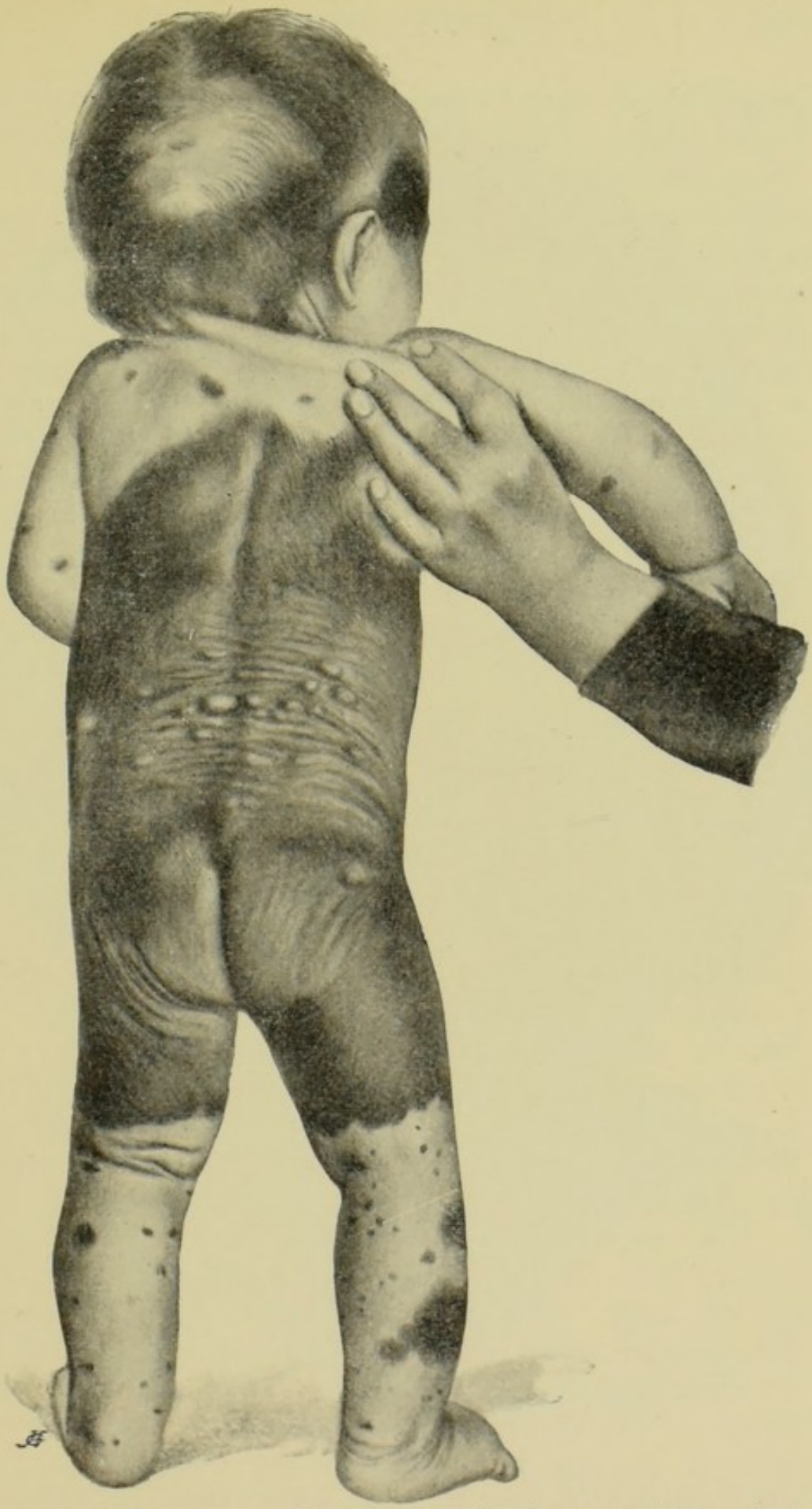


FIG. 140.—Nevus pilosus. Large grayish-black nevus pilosus which covers the body like a pair of swimming trunks. It is thickly covered in certain areas by black hairs and a large number of benign growths (fibroma molluscum). There are also disseminated over the body smaller and larger nevi, which are nearly all thickly covered with hair. (Clinic of von Ranke, Munich.)

period of life (proliferation and new formation of blood-vessel walls). This condition occurs mainly in the papillary and upper layer of the skin or the corium. The smooth or swollen skin presents specks, which may vary in color from a flaming red to bluish red—*nevus flammeus* or *angioma simplex;* or elevated, swollen, and even pulsating growths are seen, the skin of which, sometimes smooth and other times rough, allows the dilated vessels to shine through—*angioma cavernosum.* Nevi increase in size during the early period of life, after which they generally remain unchanged. A nevus flammeus may also disappear spontaneously. When an angiocavernoma grows to an excessively large size it may become dangerous by pressure upon the surrounding organs.

Treatment.—Excision, cauterization, electrolysis, galvanocautery, and radium treatment.

SEBORRHEA

An increased activity of the sebaceous glands exists in the newborn child as in the fetus, consisting of the excretion of epidermoid cells which have undergone fatty changes and an active regeneration of epidermis. If this function, which is physiologic in the newborn, should continue throughout the first few days of life, and if the activity becomes abnormally increased, we have a diseased condition which is designated, according to whether it is spread generally over the body or whether it is limited to individual body areas, as universal or local seborrhea.

The commonest situation for local seborrhea is the *scalp.* The products of this process, which are termed by the laity "scabs," consist of fat, dust, loosened epidermis, and hair; these substances form yellowish-brown or dirty colored, greasy masses, which are brittle like cheese or dry and possess in some cases a foul odor. This mass of material is spread over the whole scalp either in thin or thick layers or it is found only in certain foci. The scalp beneath this scab is pale and moist, as if



FIG. 141.—Seborrhea of the head and face. Child seventeen months old. The scalp and upper half of the face are coated with an uninterrupted layer of thick, dirty sebum. (Clinic of Escherich, Vienna.)

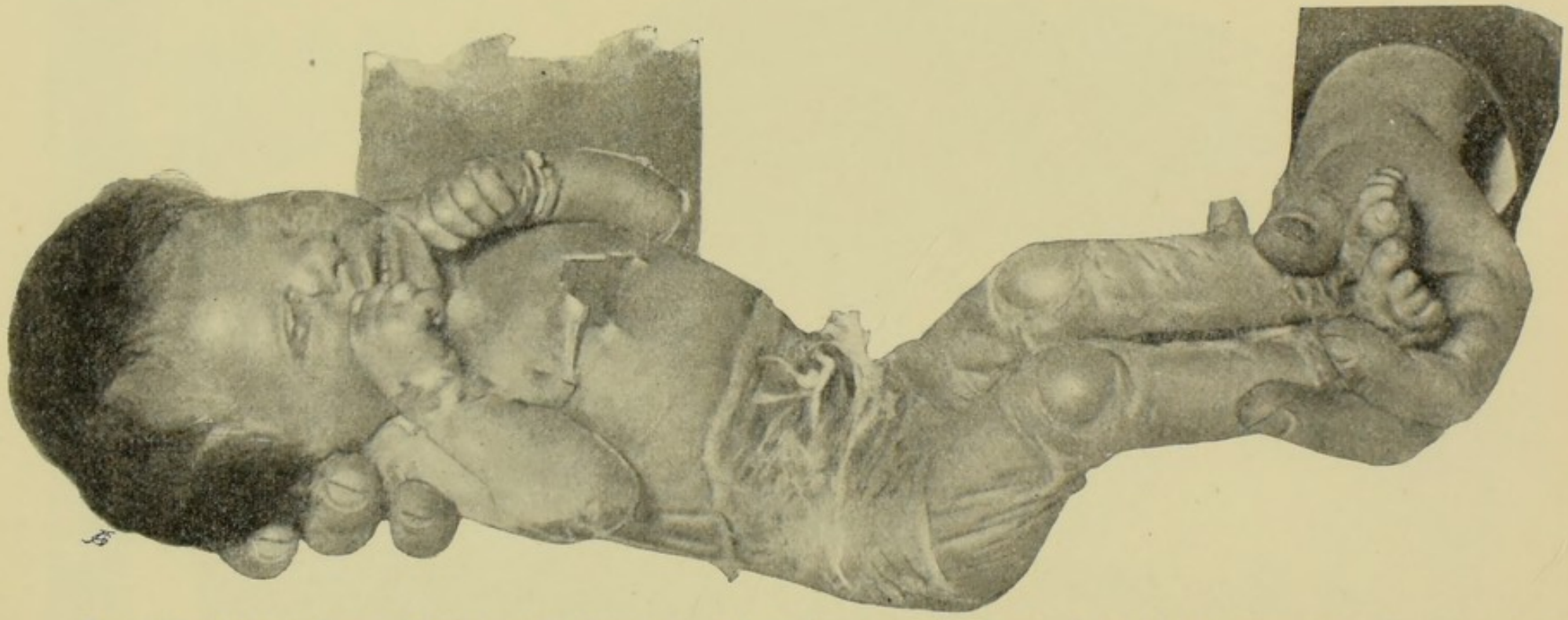


FIG. 142.—Severe form of universal seborrhea. Child two days old. The whole body, excepting the scalp, is covered with a rigid, parchment-like coat. In certain areas, especially at the joints, this coating has become loosened and the red shiny skin is exposed to view. The skin coating feels hard, crepitates on pressure, and interferes with the movement of the child to such an extent that it lies almost motionless with its arms flexed and legs slightly drawn up. Crying and nursing are impossible. The eyes are contracted into narrow slits with a slight amount of eversion of the eyelids. The small ears are fastened to the head with sebum The more brittle portion of the epidermis was rapidly loosened by means of soap and water. Death occurred on the twenty-eighth day, probably due to chronic sepsis which originated at the umbilicus.

covered with oily drops of sweat, and not rarely it is inflamed and eczematous from the macerating action of altered skin secretion. The portion of the scalp covered by hair is less damaged; the hair loosens and falls out and disk-shaped areas of baldness result.

Universal seborrhea of the newborn infant, also designated as *cutis sebacea* and *congenital ichthyosis*, is due to a constant renewal of the *vernix caseosa* (after-birth), which dries and covers the whole body with a horn-like substance. The stiff coating of the skin varies from a yellowish- to a brownish-red color and possesses a varnish-like gloss (according to Hebra it resembles the skin of a half-roasted sucking pig). The body presents a statue-like immobility on account of the tightness and stiffness of this coating.

If the mouth is involved the act of nursing is impossible; yet the mouth, eyes, and anus usually remain unaffected. Deep tears in the covering on the face and at the joints show the lamellar structure of the deposit of sebum. It may be drawn off in these regions in large fragments. The skin underneath this coating is slightly red, shiny, and soon becomes covered again with masses of sebum. The child soon dies from inanition and loss of body warmth (Kaposi, Escherich).

Treatment.—The sovereign remedy in the treatment of seborrhea is sulphur, in the form of 10 per cent. sulphur and Lassar's paste or sulphur baths. The masses of sebum may be loosened by means of warm oil, cod-liver oil, butter, or boric-acid ointment, and removed with lukewarm soap-water (glycerin soap).

ICHTHYOSIS

(*Fish-scale Disease*)

Ichthyosis is a skin disease transmitted by heredity, which consists of thickening of the corium, together with a uniform hypertrophy of the papillary bodies and and an increase of cuticular pigment. The sebaceous glands are atrophic (contrary to congenital ichthyosis).

PLATE 40

Universal Seborrhea (Cutis Sebacea, Congenital Ichthyosis) of a Mild Type.—The body of a child eight days old, covered with a thin brownish-red, shiny layer of sebum, which has been torn in many places by the voluntary movements of the child, and appears, therefore, to be composed of a large number of small and large scales which have white borders. Recovery followed the usual treatment of seborrhea. (Clinic of von Winckel, Munich.)

Symptoms.—As a result of the interference with the function of the sebaceous glands the skin is dry, markedly scaly, wrinkled, and rough on account of the thickening and prominence of the normal furrows of the skin. In a more severe form of this disease we note, in place of the furrows, more or less deep painful fissures (especially in the region of the elbow-joint); as a result of this fissuring, scaly, horn-like plates are formed, which are pigmented a dirty grayish-brown to grayish-green color and have pale borders. Severe cases are accompanied by the formation of true horns. Active shedding of the horny mass occurs.

The disease generally attacks almost the whole area of the body symmetrically, especially the extensor surfaces of the extremities; the face, genitalia, palms, and soles, however, remain uninvolved. On the other hand, in exceptional cases the arms and soles are alone affected—*ichthyosis palmaris et plantaris.* Almost as rare is *ichthyosis follicularis*, in which the horny formation involves only the skin-follicles (Lesser). Ichthyosis generally begins in the course of the first or second year of life and continues practically unchanged throughout life. The general health is only slightly disturbed.

Diagnosis.—Trophoneuroses may occasionally simulate ichthyosis, yet they are limited to narrow confines and do not arise symmetrically. Lichen pilaris may be mistaken for ichthyosis follicularis, yet is rarely met before puberty.

Prognosis.—Absolute cure is rare.

Treatment.—Warm soap-baths, followed by rubbing in of fat, soft-soap, or 5 to 10 per cent. sulphur ointment.



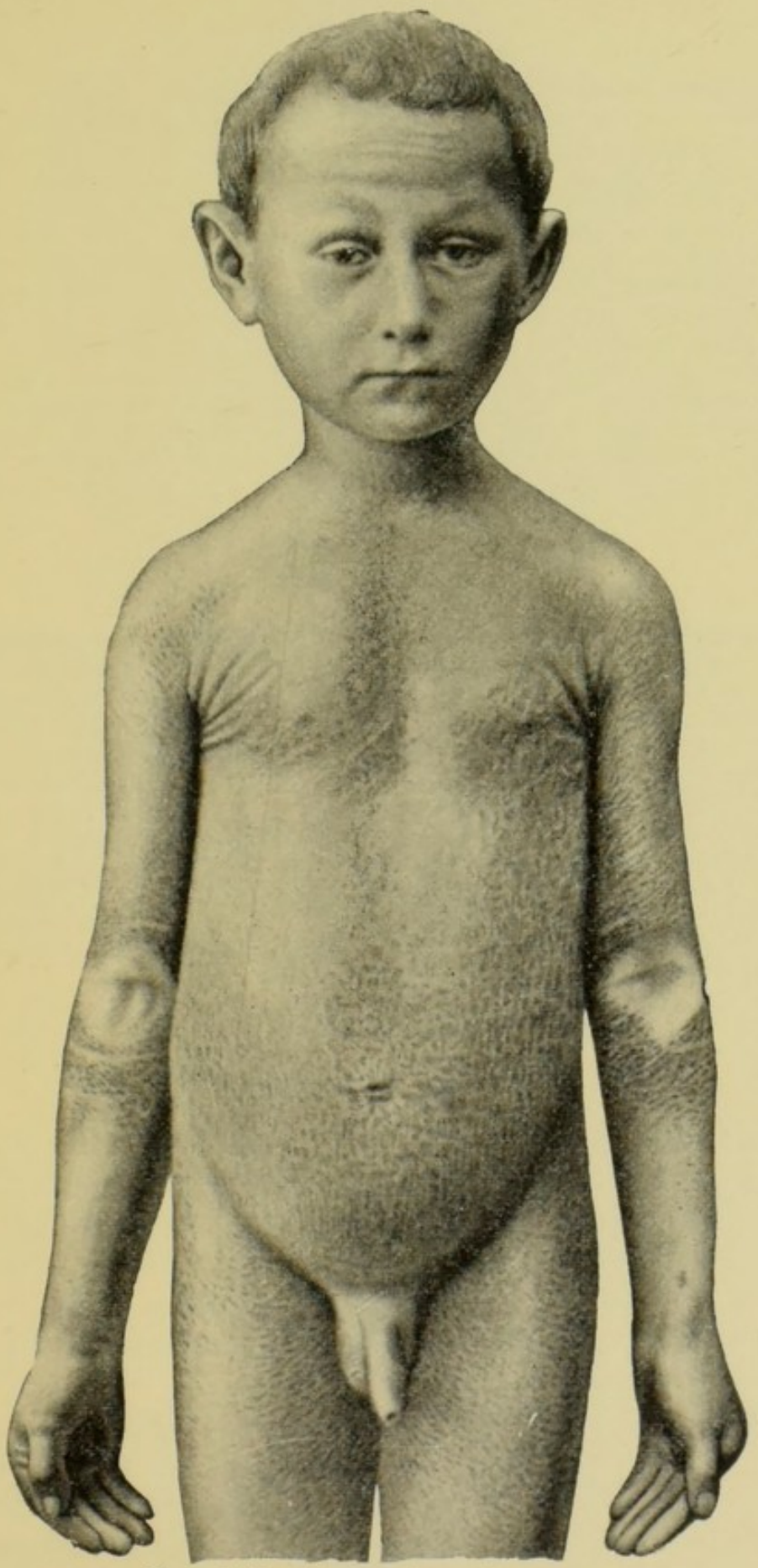


FIG. 143.—Ichthyosis. (See text.)

PLATE 41

Pemphigus Neonatorum.—Child four weeks old. Vesicles appeared in the skin one day after birth. Parents and midwife healthy. Child emaciated, anemic, and weak. The vesicles are covered with a sallow skin all over the body. They are most abundant on the flexor sides of the lower extremities. Their color is whitish or grayish yellow and they vary in size from that of a lentil to a silver dollar. (The palms of the hands, as well as the soles of the feet, are free, excepting one large vesicle on the left great toe.) The smaller vesicles are tense, whereas the larger ones are relaxed and their membranes wrinkled or torn and collapsed. The contents of the vesicles consist of a serum which is clear or turbid with pus (the bacterial findings showed the presence of the Staphylococcus pyogenes aureus). In many areas nothing but scabs remained or, where these have been removed, specks are noted which are red and weeping or covered with a delicate membrane surrounded by a white epidermic ring. High septic fever. Death in four days after admission. (Clinic of von Ranke, Munich.)

PLATE 42

Pemphigus Syphiliticus (Exanthema papulo-vesico-pustulosum).—"Infant six days old. Papular and vesicular eruption to the size of a pea on the legs and soles of the feet which contained pus and surrounded by an inflammatory areola. On the following day the extensor surface of the lower extremities, nates, and back also became covered with a profuse papular eruption. A vesicular and papular eruption occurred between the papules. The nose remained uninvolved. Death on the seventeenth day from bronchopneumonia and gastro-intestinal catarrh. At necropsy, among other changes, infiltration of the liver and spleen was found to be present." (From Mraček, *Atlas of Syphilis.*) Primary development of copper-colored papules and the secondary conversion of these into pustules. In pemphigus neonatorum, on the contrary, we note primarily the formation of vesicles.

PEMPHIGUS NEONATORUM

Pemphigus neonatorum is a contagious skin disease which generally runs a favorable course and is accompanied by the formation of vesicles; it develops sporadically and sometimes also endemically and epidemically. The vesicles are due to the exudation of a serous fluid into the rete which causes the production of vesicles. The etiology of the disease is unknown.

Symptoms.—A number of lentil- to pea-sized hemispheric vesicles develop in various parts of the body during the middle or at the end of the first week in children enjoying good health; occasionally their appearance may be accompanied by a slight rise of temperature. The vesicles





are transparent, grayish red, or yellowish, surrounded by a narrow red areola, fairly tightly distended by serum, and easily ruptured. With the appearance of repeated crops we may finally note thirty to fifty vesicles of varying size and stage of development. Large vesicles (as large as a twenty-five-cent piece) become flatter and more relaxed and are the last to rupture, after which the contents dry up. After the shell of the vesicle peels off the skin is seen to be slightly red, still moist, but already covered with a delicate tissue and surrounded by a white ring of epidermis. The disease runs a favorable and afebrile course which terminates in two weeks, provided no septic complications set in.

The *malignant form* occasionally observed is accompanied by the formation of huge vesicles on previously reddened skin, runs a high febrile course, and usually ends fatally (Baginsky, Bloch). (For Differential Diagnosis from Syphilitic Pemphigus, see that disease.)

Treatment.—Avoid mechanical injuries; prevent bacterial infection; favor desiccation of the vesicular contents with dusting-powders; when extensive exposure of the corium occurs resort to baths of oak-bark decoction (1 pound boiled in 3 quarts of water for a bath).

DERMATITIS EXFOLIATIVA

An increase of the physiologic exfoliation of the skin and a diffuse serous infiltration of the rete Malpighii develops, occasionally without a known cause, in prematurely born and unhealthy children during the first few days of life. This leads to relaxation and loosening of the skin to an extreme degree, and in certain areas to a smooth vesicular elevation of the same (similar to large relaxed pemphigus vesicles).

Symptoms.—The skin at first shows a rosy tint all over, after which it is speckled with an erythema-like red color. The horny layer of the skin gradually swells and appears finally as if macerated. In this stage the pressure of a finger is sufficient to displace the superficial layer of the

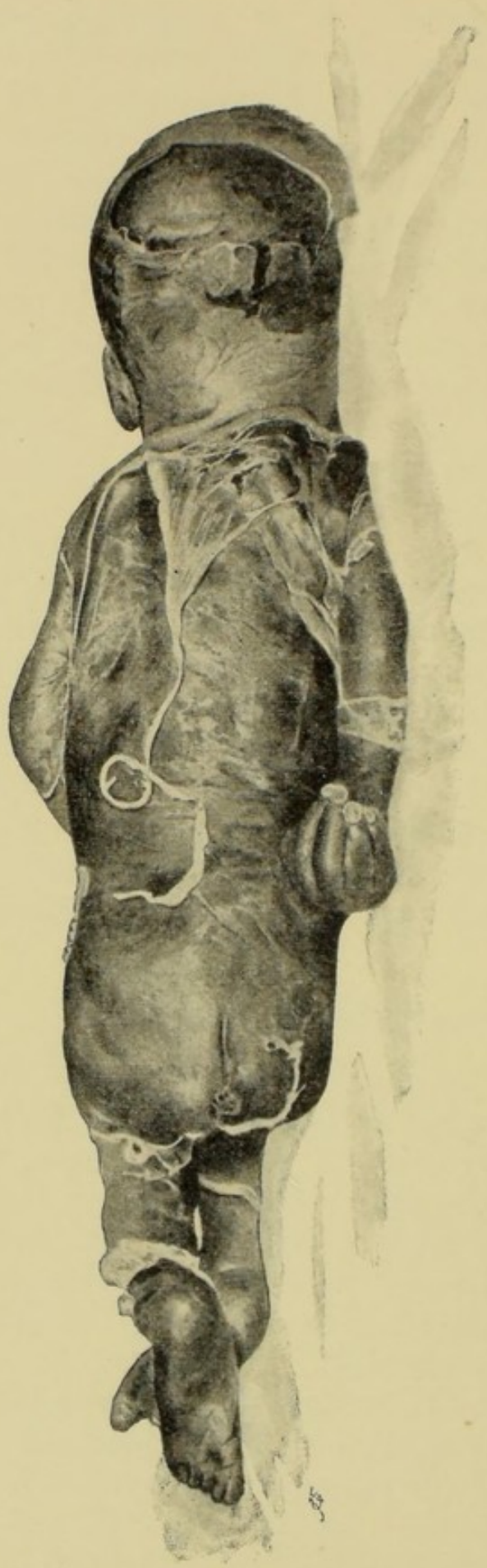


FIG. 144.—Dermatitis exfoliativa. Child twelve days old. The superficial layer of the skin of the whole body is swollen and transparent (like glass); it appears as if scalded, is relaxed, and to a large extent completely loosened from the corium (epidermolysis). The loss of warmth and fluids, as well as septic infection, led to death on the tenth day of the disease. (Ritter.)

skin upon its base, and on account of its cohesive properties it may be drawn off in large fragments. Beneath it is seen the red weeping corium. Such a displacement and tearing of the epidermis occurs with every movement of the patient, and hence we often see large sections of the superficial skin loosened and hanging down in large shreds or rolled up like a knot. Vesicular separation of the skin is usually seen only in dependent regions and on the extremities, and not rarely to such an extent that the superficial layer of the skin represents a glove-like coating of the affected part, which may often be drawn off *in toto* (Escherich). The disease itself is not accompanied by fever or other marked disturbances of general health. The majority of the patients, however, die from general sepsis. It may be possible to save a life now and then when the epidermolysis is not too extensive and when the outward conditions of life are favorable. The specific treatment is that of acute pemphigus.

SCLEREMA NEONATORUM

Sclerema neonatorum is a disease characterized by hardening of the skin and loss of body temperature, which may develop in the form of sclerema edematosum, sclerema adiposum, or a combination of both forms.

Sclerema edematosum is a result of weakened cardiac action and disturbance of heat production in sickly, prematurely born infants who are suffering from myocarditis, nephritis, or syphilis, and who are victims of unfavorable and unhygienic circumstances of life. Slow capillary circulation and abnormal permeability of the blood-vessel walls lead to edema of the subcutaneous tissue and, later, to dense infiltration of the skin and hardening of the panniculus adiposus.

Symptoms.—The disease begins generally in the lower extremities, with coldness, edema, and hardening of the calves and legs. It then spreads to the trunk and in the course of a few hours or days it becomes universal. The skin of the affected parts is tense, shiny, white, mottled, or reddish. At the commencement of the disease it is

still movable upon its base, can be lifted up in thick stiff folds, and pits on pressure. At a later stage the edema disappears from the primary foci, the skin becomes dry, stiff, immovable, and turns a dirty yellow or brownish color. The stiffening of the skin interferes with the body movements and with nursing and gives the face a peculiar senile appearance. The whole body feels cold and lies stiff, as if frozen.

Sclerema adiposum, or fat sclerema, is a result of excessive loss of water and serum after exhausting diseases, especially cholera infantum. The consequent disturbances of circulation and lowering of the body temperature may, in conjunction with improper feeding and insufficient application of external heat during the first week of life, lead to a finely granular coagulation and stiffening of the subcutaneous adipose tissue (Knöpfelmacher, Siegert).

Symptoms.—The disease spreads rapidly, but spares, as a rule, the anterior surface of the neck and trunk. The skin is dry, without gloss, and dirty yellow in color; it does not pit on pressure nor is it movable on its base. The whole body is stiffened and immobile like a corpse. Both affections, the sclerema edematosum and the sclerema adiposum, possess in common the constant and progressive falling of the body temperature about 2° to 3° C. [1.8°–3.6° F.] daily, until 29° or 25° C. [84.2° or 77° F.] is reached; also a rapid lessening of all other vital functions, which leads, as a rule, to death in a few days or, at the latest, in two or three weeks.

Prophylaxis and Treatment.—Children in danger of sclerema should be provided with the best possible circumstances of life and, above all, the loss of energy through dissipation of heat must be prevented (wrap in flannels, cotton, hot-water bottles, or incubator). In exceptional cases it is possible to save children suffering from sclerema by the application of hot moist packs, hot baths (30° to 32° R. [86°–89.6° F.]), as well as by increasing the capillary circulation by massage, the cardiac activity by stimulants.

ECZEMA

Eczema is the commonest skin disease of childhood. It is characterized by an itching polymorphous eruption, and anatomically by an exudative dermatitis which is chiefly confined to the uppermost layers of the skin. This dermatitis consists of a pronounced serous exudation and cellular infiltration. The etiologic factor may be any skin irritant of a chemic, mechanical, thermic, or parasitic nature. In many cases this irritation of the skin is only the exciting cause, the real factor being a dyscrasic constitutional anomaly or a disturbance of metabolism, as in scrofula, rachitis, status lymphaticus, obesity, prolonged disturbance of digestion, and especially as a consequence of overfeeding.

Symptoms.—Depending upon the nature and duration of the injurious influence and upon the individual predisposition, eczema manifests itself in the form of a diffuse reddening and painful edematous swelling of the skin, or in the form of pale or red itching nodules which become rapidly converted into vesicles or pustules (*Eczema erythematosum, papulosum, vesiculosum, pustulosum*). These manifestations may undergo resolution in a few days or rupture, and scratching of the vesicles and pustules causes the development of weeping eczema (*Eczema madidans*), which heals with the formation of crusts and scabs, but not until several weeks have elapsed (*Eczema crustosum, squamosum*). Healing may, however, be considerably delayed by the continuation of exudation, which causes stasis of the pus-altered serum underneath the crusts (*Eczema impetiginosum*) or even purulent destruction of the tissue (*ecthyma*).

Eczema may also occur diffusely and spread over the entire body or remain confined to certain areas of predilection (scalp, mouth, lobule of the ear, cheeks, around the eyes, nates, genitalia, and the inner surfaces of the arms).

A *cyclic eczema* is occasionally met with in which the described alterations occur in regular order over a course

PLATE 43

Chronic General Eczema.—Child a year and a half old, who suffered since the first year of life from a skin eruption which began in the face and then spread over the whole body. Otherwise healthy and well nourished. The skin, especially of the back, is reddened and covered with numerous irregularly grouped yellowish-red to dark red small nodules of about the size of a pin's head, also honey-colored or brownish crusts and whitish scales. In certain areas the skin is markedly infiltrated.

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of from three to four weeks. More frequently, however, the disease spreads at the periphery and heals centrally, or through irregular recrudescences the disease appears in different parts of the body, presenting at one time this, and at another time that, pathologic change, and running a chronic course spread over several months.

In acute localized eczema the general health is but slightly influenced, excepting perhaps the effects of prolonged itching. In general eczema and in case of delayed healing the bodily nutrition suffers from fever, sleeplessness, lack of appetite, and loss of serum. Complications to be considered are lymphadenitis, furunculosis, phlegmon, and gangrene.

Course and Prognosis.—If the dermatitis remains superficial, as is the rule, recovery is complete and without destruction of tissue; otherwise superficial scars remain (always in ecthyma). If the disease lasts for years, the disturbance in the nutrition of the skin leads to the formation of permanent tissue changes, including pigmentation, thickening of the skin with degeneration of the hair-follicles, sweat, and sebaceous glands.

The **prognosis** depends upon the cause. If the dermatitis proceeds to gangrene and phlegmon, collapse and death may follow the development of eclampsia. Sudden death has been observed, even in the absence of these changes, in children who are encumbered with lymphatic constitutional anomalies—*eczema death* (Feer).

Chief Characteristics of Eczema.—The redness of the skin in eczema disappears on pressure; eczematous papules and vesicles are usually crowded closely together in an irregular arrangement and are never of long duration.



Removal of the eczematous crust exposes the red moist skin, yet no loss of substance from ulceration (excepting ecthyma). In eczema of long duration we note, occurring simultaneously side by side, the various eczematous types, with apparent infiltration of the affected portions of the skin. The forms of eczema noted as especially frequent in children are:

Eczema Sudamen or Miliaria.—This is a papular form of eczema which is caused by the sweat, and consists of closely crowded red papules of about the size of millet seeds. On the tip of the papules are found minute vesicles which are clear as water or whitish on account of the turbidity of their contents. This type passes not infrequently into the weeping form of eczema.

Eczema Intertrigo.—This is an erythematous form of eczema of skin folds which have become macerated by rubbing against each other, as on the genitals, the nates, and on the folds of the thighs, axilla, and neck. It is frequently complicated by a papular eczema. When of long duration a loss of epidermis results and the weeping type of eczema develops. When neglected, gangrene arises.

Crusta Lactea or Porrigo Larvalis.—This is a chronic impetiginous facial eczema which is especially peculiar to the nursing period. It develops generally in overfed infants and frequently even as early as a few weeks after birth, and predominates as an impetiginous crusted and squamous eczema in the forehead, cheeks, and ears. It persists for many weeks and even months.

Impetigo Contagiosa.—This is an acute pustular eczema caused by micro-organisms and transmitted by contact. It is distinguished from the non-contagious impetiginous eczema by the size of the pustules (as large or larger than lentil seeds). It is generally confined to the face, yet isolated impetiginous vesicles are occasionally met with scattered over the whole body (auto-infection with the fingers). The disease begins with the development of disseminated red papules which are rapidly converted into vesicles and superficial pustules. The latter remain

PLATE 44

Crusta Lactea.—Overfed infant eight months old. The eruption, which causes much itching, has existed for three months. The scalp is covered with grayish-green masses of sebum. Where the latter has been scratched off we may observe the darkly reddened skin covered with hair, which is bloody in some areas and covered in other places with brownish-red crusts and fat-drops. The forehead, the neighboring portions of the cheeks, and the areas around the mouth are covered with partly fresh and incrusted multiple confluent pustules. The skin of the whole face is markedly red and rough like plush.

PLATE 45

Impetigo Contagiosa.—The eruption, from which also a brother and two playmates suffered, is claimed to have existed for two weeks. Previous to this time only the skin of the face was involved. That region presented several dozen single or irregularly grouped pustules, some of which are small (about lentil-seed size), tense, and resting on a red and somewhat infiltrated base; whereas others are larger, flat, relaxed, and, to a large extent, confluent. A continuous deposit appears on the upper lip and chin; this is covered by a honey-yellow to grayish-green, tough, elastic scab, underneath which is pus, removal of which exposes the red moist corium.

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circular in outline and isolated at the beginning, but later they coalesce and form, upon the eruption of new crops, irregular figures. The gumma-like crusts which form are of a dirty yellowish-green or brownish-red color if blood be present, and fluctuate on account of the pus which has collected beneath them. Very similar to impetigo contagiosa is the eczema facialis impetiginosum, which is usually due to pediculosis.

Ecthyma.—This is a pustular eczema in which the inflammation also attacks the uppermost layers of the corium and proceeds to ulceration of the tissue. It is apparently only a secondary manifestation of scratching and follows the same cause in scabies and prurigo. As a rule ecthyma consists of isolated pustules, about the size of a pea and surrounded by a red areola, on the extensor surfaces of the lower extremities, on the buttocks, on the back of the hands and feet, and, rarely, on the face and other portions of the skin. Healing is accompanied by scar-formation.

Ecthyma cachecticorum, which occurs in anemic and atrophied children and in those weakened from disease under the manifestations of sepsis, may run a fatal course.



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Treatment of Eczema.—Remove the cause (in every case alter the diet; provide a vegetable dietary; avoid overfeeding) and regulate the intestinal functions. Keep the

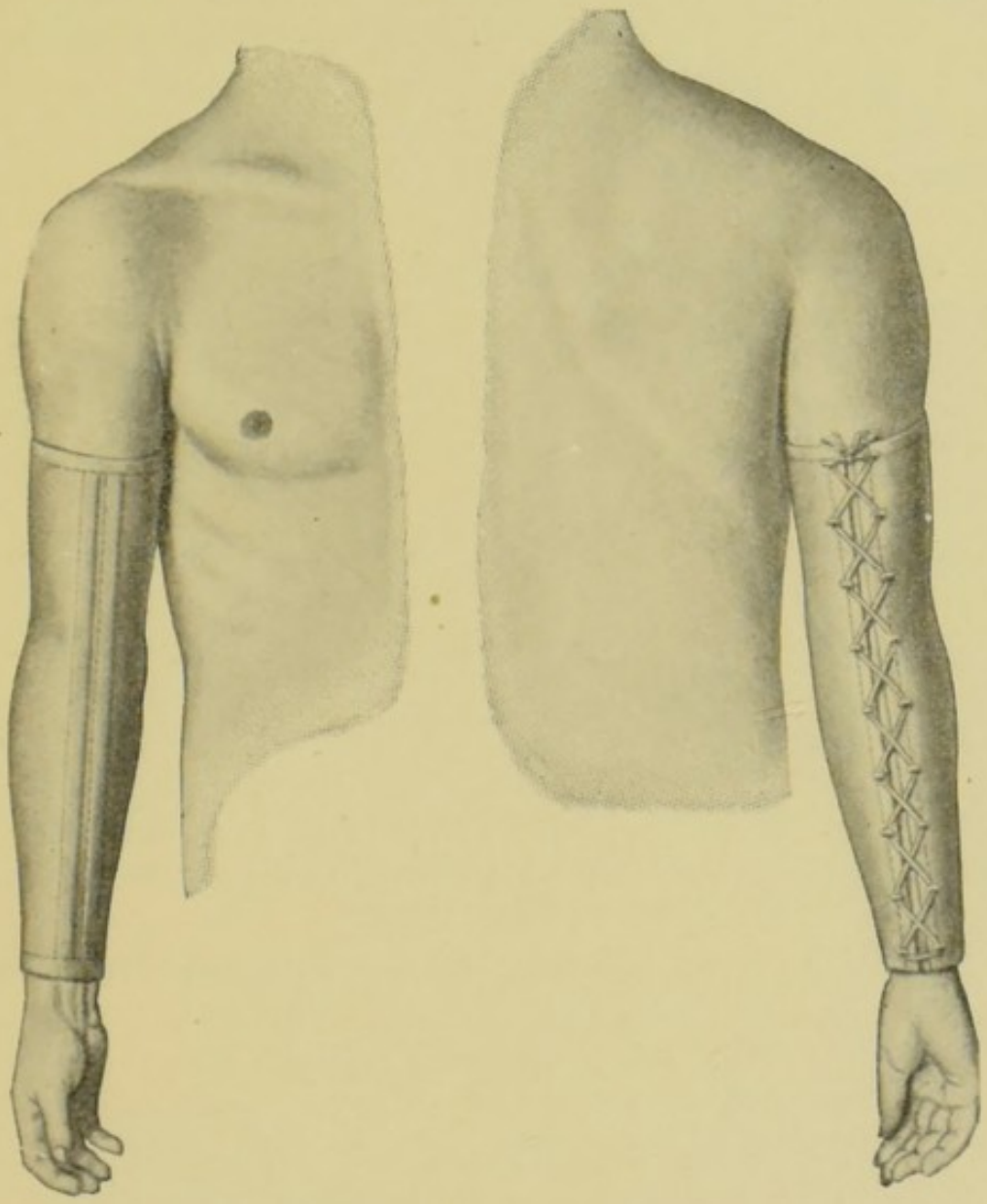


FIG. 145.—Arm bandages of Eversbusch. Sleeves reaching from the middle of the arm to the wrist, which are composed of two layers of drilling, between which two or three small wooden splints are sewed. The sleeves are so applied that the splints lie upon the flexor surface. They are fastened by lacing on the extensor surface. These arm bandages permit free movement of the hands, but prevent flexing of the arms and hence scratching of the face. They are also recommended in children who have undergone intubation, in order to prevent the removal of the tube by the thread to which it is fastened.

skin thoroughly cleaned and avoid fresh injury. Attempt to assist and hasten the natural healing process with as

bland remedies as possible. Avoid rubbing, pressure, or wetting of the eczematous areas. Hence prohibit the wearing of too tight or too warm clothing or such as will irritate the skin (wool). Remove as rapidly and as carefully as possible from the diseased portions of the skin urine, feces, sputum, and vomitus by means of gentle swabbing with cotton (dry or soaked in sweet oil). Limit bathing and washing to the uninvolved portions of the body. If, however, occasional cleaning is necessary, employ cotton (do not use a sponge, which is cleaned with difficulty) and only boiled water to which has been added a little aluminum acetate or a 1 per cent. solution of boroglycerin; dry by gentle swabbing and not by rubbing. Add to the baths wheat-bran or potassium permanganate solution until a rose-red color is obtained.

The *arm bandage* of Eversbusch is employed to prevent injurious scratching and the diseased areas of the skin are protected by an ointment dressing or by zinc oxid adhesive plaster. The itching is lessened by washing with alcohol or by the addition of menthol, carbolic acid, etc., and the use of dusting-powders, ointments, or pastes. To secure healing in children it is sufficient, as a rule, to remove the causal condition and avoid injurious factors.

The following *dermatotherapeutic measures* are recommended: In case of eczema caused by the sweat, miliaria, and intertrigo use bland dusting-powders (zinc oxid, 5.0 gm.; talcum venet., 30.0 gm., with or without $\frac{1}{2}$ to 1 per cent. menthol); macerated areas are first painted with a 2 to 3 per cent. solution of silver nitrate. In other acute forms of inflammatory eczema (papular, vesicular, pustular) make cold moist applications with aluminum acetate (15 : 500). In the crusty impetiginous type of eczema resort to mechanical removal of the scabs after they have been previously softened with aluminum acetate applications, ointments, or oil dressings; afterward apply aluminum acetate until the inflammation has undergone resolution and, finally, use a drying paste (Lassar's paste). In isolated impetigo contagiosa remove the scabs, paint

with silver nitrate solution, and apply a paste. Ecthyma should be first treated with corrosive sublimate dressings and later with applications of aluminum acetate. Of value in squamous and chronic eczema is an ointment of sulphur or 1 part of liquor carbonis detergens with 9 parts of zinc paste. Occasionally very efficient results are obtained by altering the diet, and especially in the administration of buttermilk.

PRURIGO

Prurigo is a chronic inflammatory affection of the skin which begins with an obstinate urticaria and a characteristic papular eruption at about the eighth to the twelfth month of life. The disease is accompanied by marked itching and persists, as a rule, throughout life. The anatomic findings are similar to the papular and chronic forms of eczema.

Symptoms.—The minute papules, which do not develop generally until the second year of life, are pale or red, of a dense consistency, and occasion excessive itching. They appear chronically in the form of repeated eruptions and predominate on the extensor surface of the extremities, on the nates, in the sacral region, and at times in other localities, but they never appear on the flexor surfaces. The papules are generally scratched and covered with small bloody scabs, which remain in place even after the disappearance of the papules. Continued eczema causes streaky excoriations and the secondary development of all the different varieties of eczema. Finally, the skin, especially on the legs, which are always most severely attacked, presents brownish pigmentation, infiltration, thickening, and dryness. The lymph-nodes, especially the crural, undergo indolent swelling and feel like flat pebbles. The sleeplessness and the loss of body fluids cause a rapid decline in health and the child looks pale and haggard.

Chief Characteristics.—The skin of the extensor surfaces of the extremities is covered with minute papules and scabs, and later with secondary eczema. The integument

PLATE 46

Erythema Exudativum Multiforme.—Girl fourteen years old. Little round decidedly red papules developed without a demonstrable cause upon the backs of both hands. They enlarged rapidly and upon spreading to the fingers occasioned itching and pain. The general health was otherwise undisturbed. The eruption, which covered nearly the whole back of the hands, coalesced into areas varying between a five-cent piece and a silver dollar; their wall-like elevated edges, which were joined by curved lines, were dark red in color, whereas their centers were somewhat pale, bluish red, and presented in several areas brick-red spots (fresh papules beginning at the center—erythema iris). Repeated occurrence of fresh crops. Recovery in five weeks. (Clinic of Escherich, Vienna.)

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is scratched, pigmented, and infiltrated. The flexor surfaces of the joints are always uninvolved. Glandular swelling. Continual itching. A chronic course marked by recurrences.

Prognosis.—A temporary improvement is all that may be hoped for. Recovery from the severe types is impossible.

Treatment.—Anoint with 1 to 3 per cent. naphtol ointment three times a week in the evening. Wash in bath with naphtol and sulphur soap. Cod-liver oil, regulation of the diet, and sojourn in the country.

ERYTHEMA EXUDATIVUM MULTIFORME AND ERYTHEMA NODOSUM

Erythema exudativum multiforme and erythema nodosum are inflammatory dermatoses which follow irritation of the vasmotor centers. The etiology is still uncertain; they are influenced to a certain extent by diseases of the internal organs (auto-intoxication). Characteristic of the angioneurotic erythema is the development of red specks, whose periphery is dark red and whose center is colored a bluish red. The dark red color is due to active congestion with blood, and the central blue coloration is caused by consecutive relaxation hyperemia (stimulation and paralysis of the vasoconstrictors). The relaxation of the blood-vessel walls may also lead to the escape of hemoglobin, serum, and even red blood-cells, which cause dis-



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coloration and the formation of papules, nodules, vesicles, and hemorrhages (Kaposi).

Erythema Exudativum Multiforme.—This disease consists of simultaneous and symmetric development of disseminated, flat, or somewhat elevated red specks about the size of a pin's head on the backs of both hands and feet, as well as on neighboring portions of the forearms and legs. These spots rapidly enlarge to the size of a silver dollar and tend in many instances to coalesce. The centers of the larger specks appear bluish red, and when hemoglobin escapes from the vessels they present various colors, from blue to yellow, green, and brown. If fresh primary specks appear in the same areas they assume, on account of the hematin already present, a brick-red color (erythema iris). According to the grade of exudation we note the formation of papules, nodules, vesicles (erythema papulatum, urticarium, vesiculosum, bullosum; with a peripheral vesical border: herpes circinatus, herpes iris). The affection may, in the course of time, attack the skin of the whole body, and also the tracheal and laryngeal mucous membrane. It runs, as a rule, an afebrile course accompanied by moderate itching and terminating in from two to six weeks.

Erythema Nodosum (Dermatitis Contusiformis).—Capillary stasis in and beneath the skin of the backs of both feet and legs, more rarely of the thighs, nates, and upper extremities, leads to the formation of dense, painful swellings and nodules about the size of a hazel-nut. Their appearance is accompanied by gastric disturbances and swelling and painfulness of the joints. The skin covering the papules is rose red in color, but in from two to three days shows the gradual development of the same play of colors seen in the eruption of erythema exudativum multiforme. The papules undergo resolution in from eight to fourteen days, yet the appearance of fresh crops of eruption may prolong the disease over six weeks or more.

Treatment.—There is no especial treatment for erythema exudativum multiforme. To allay the itching employ

menthol or carbolic acid, 1.0 gm., to spiritus vini gallici, 150.0 cc. Erythema nodosum requires rest in bed, cool applications, and the administration of the salicylates for articular pains.

LICHEN URTICATUS

(*Strophulus, Tooth-rash*)

Lichen urticatus is an angioneurotic eruption accompanied by much itching and developing frequently at the time of the first dentition ("tooth-rash"); in the latter case it is probably excited reflexly from the dental nerves. It occurs, however, also in children who are not teething, practically so only in the second period of childhood, from as yet unknown causes (in many cases it can be referred to an insect bite).

Symptoms.—Without any constitutional disturbance various parts of the body, especially the neck, lower extremities, soles of the feet, and the palms of the hands, become covered with red specks which rapidly grow into wheals the size of a lentil seed. Their centers fade and assume the appearance of wax-like vesicles (similar to varicella); they are, however, distinguished from vesicles by their extremely dense, horn-like consistency. The excessive itching causes the eruption to be scratched sore. The repeated occurrence of fresh eruptions may prolong the disease over weeks, months, and even years.

Treatment.—Anoint with remedies which will relieve the itching, such as 1 to 2 per cent. carbolic acid solution or menthol, 1.0 gm.; glycerin, 3.0 gm.; spiritus vini gallici, 150.0 cc., and spiritus ætheris, 15.0 cc. Apply bland dusting-powders to the areas which are still moist. In obstinate cases administer a laxative.

URTICARIA

Urticaria is a disease belonging to the angioneurotic forms of dermatoses and is characterized by the development of wheals. It develops suddenly, following certain internal and external forms of irritation, vaccination,

insect bites, burns, dyspepsia, intestinal parasites, certain foods (strawberries), and psychic influence, and disappears rapidly in a few hours or days, accompanied by moderate scaling of the skin, and occasionally leaving yellow specks behind.

Symptoms.—The wheals, which are circumscribed elevations of the skin due to the collection of a serous fluid in the rete, are white or rose red in color and surrounded by a red areola. They vary in size from a lentil seed to that of a twenty-five-cent piece, and tend frequently to coalesce into extensive irregular figures. The face and joints are attacked by preference (when localized in the orbital region no wheals develop, but the lids are red and edematous). The appearance of wheals is associated with itching or burning, which increases when resting in bed. Fever is occasionally present. Recurrences are especially frequent in nervous subjects.

Treatment.—When possible remove the cause. Locally apply cooling lotions and the salicylates in the powder form. [Laxatives are indicated in the persistent forms. Belladonna or atropin in minute doses brings relief.—Ed.]

LICHEN SCROFULOSORUM

Scrofulous children, especially at the age of puberty, occasionally develop gradually and unnoticeably an eruption which itches but slightly and consists of flat, pale red or yellowish-brown papules. The latter vary in size from a millet seed to a pin's head, are but slightly resistent, and possess a scaly summit. They are arranged in groups or in circles, chiefly upon the trunk, less rarely upon the extremities, and remain unaltered for months, after which they gradually fade and undergo resolution accompanied by moderate exfoliation. Anatomically the local process consists of a cellular infiltration and exudation in the neighborhood of the orifices of the hair-follicles (Kaposi). The cause of this condition is scrofula, and other symptoms of that disease are always present, especially marked lymphatic swelling. (Since the genetic

PLATE 47

Lichen Scrofulosorum.—A girl nine years old presented the typic symptoms of scrofula, including chronic conjunctival and nasal catarrh and swelling and hardening of the cervical and axillary nodes. An eruption is seen on the skin, chiefly on the trunk, which during the two months of its existence has undergone no noticeable change. Innumerable pale, brownish, millet-seed-sized flat papules are seen irregularly grouped and partly arranged in continuous lines and crescents. Single papules are also noted on the skin of the upper arms and thighs. Slow recovery followed the external and internal use of cod-liver oil.

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relationship between scrofula and tuberculosis has been more thoroughly studied there has been a tendency to also call lichen scrofulosorum miliary tuberculosis of the skin.)

Chief Characteristics.—Appearance of homogeneous, pale-red or yellowish-brown soft papules covered centrally by scales, which occur in groups or are arranged in circles. They attack, as a rule, only the trunk and persist for weeks and months without undergoing conversion into vesicles or pustules. Accompanied by symptoms of scrofula.

Treatment.—Oil the dry, papular skin with cod-liver oil two or three times a day. Treat as in case of scrofula.

HERPES

Herpes is an ephemeral eruption of a group of vesicles on the face or on the genitalia, and spreads independently of the course of the nerves. It is a frequent concomitant to febrile diseases, but arises also in healthy children without a demonstrable cause.

Symptoms.—Pin-head-sized vesicles which rapidly coalesce appear with a moderate burning sensation and itching on a section of skin previously reddened. They are clear as water and are arranged either in round or irregularly formed groups. In one or two days they become turbid and purulent, followed by desiccation and the formation of scabs. Healing occurs within a week. The diagnosis is easily made, even when the vesicles coalesce



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or when the scabs are lost (through maceration or scratching), by the constant circular form of the eruption.

Treatment.—Bland dusting-powders; for marked swelling make applications with aluminum acetate (1 tablespoonful to 1 pint of water).

SCABIES

(*Itch*)

Scabies is an itching eruption caused by the presence of the itch-mite (Acarus scabiei), which, burrowing itself in the skin as deep as the rete, causes eczematous changes. The mite, which is transmitted by contact, attacks by preference the interdigital folds, the flexor surface of the wrist, elbow, and knee-joints, the gluteal and axillary folds, the prepuce, and in children also the palms of the hands and soles of the feet. The disease may spread further from these central sites, but the head always remains uninvolved. The burrows appear in the hands and feet as irregularly curved whitish lines dotted with dark points. In other regions they are represented by long papular reddened elevations, the surface of which appears as if scratched with a needle. The point at which the mite enters the epidermis is marked by a small pustule or, after it has died, by a pear-shaped epidermal exfoliation. The mite lies at the end of the burrow and may be recognized macroscopically as a whitish-yellow point glistening through the corneum. The dark and almost black points in the burrows are the feces of the mite. The eczema (papular, vesicular, and pustular formation) is partly primary, due to the activity of the mite, and partly secondary, due to scratching on account of the intense itching.

Chief Characteristics.—Itching of the skin, which is increased by the warmth of the bed. A peculiar type of eczema which predominates in the areas of predilection, *the flexor surfaces of the joints*, from which the face as well as the head is exempt, and which consists, almost without exception, of isolated eruptions which do not spread to a larger eczematous area until the lapse of a long time.

PLATE 48

Scabies.—A minute papular and, in some areas, pustular eczema, causing itching, developed on the whole body with the exception of the head, in a girl thirteen years old, whose sisters suffered from the same affection. The eruption, which occurs mainly in isolated areas, shows much scratching and is covered with bloody scabs. The eczema is most marked in the flexures of the joints. The picture of the hands shows the eruption following chiefly the interdigital folds and deeper skin furrows. The first hand shows several burrows (not well reproduced). The disease has existed for two weeks. Recovery in five days by means of energetic rubbing with a sulphur ointment.

Mite-burrows, which are especially prominent and numerous in the locations of predilection.

Treatment.—Destroy the mite and its eggs and afterward cure the eczema. To fulfil the first demand rub energetically with [Wilkinson's ointment: Precipitated calcium carbonate, 10 parts ; sublimed sulphur, 15 parts ; oil of cade, 15 parts ; soft soap, 30 parts ; lard, 30 parts. —Ed.], to be repeated on four successive days (painful but radical cure). Give a cleansing bath on the fifth day and put on fresh body- and bedclothes. Future baths are to be limited on account of the eczema.

PEDICULOSIS CAPILLITII

Pediculosis capillitii is pre-eminently an impetiginous eczema of the scalp which is caused by the head-louse (Pediculus capitis), and which is frequently accompanied by swelling of the lymph-nodes with the formation of abscesses. Insomnia on account of the itching. Anemia. Diagnosis is established by the discovery of the louse or by its eggs, which are found adhering to the hair. Disseminated eczematous pustules at the edge of the scalp are always suspicious.

Treatment.—To kill the lice rub the head with petroleum, but in case of pronounced inflammation it is wiser to employ a 10 per cent. white precipitate ointment. The nits may be removed by combing the hair with a fine comb which has been immersed in acetic acid. Treat the eczema.



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HERPES TONSURANS

Herpes tonsurans is a contagious itching eruption caused by the Trichophyton tonsurans. It is characterized by the presence on the scalp of bald areas covered with stubs of hair and scales varying in size from a penny to a silver dollar, which are surrounded by erythematous or circularly arranged fresh or dried vesicles. Herpes tonsurans vesiculosis occurs on the non-hairy parts of the body and consists of rings of vesicles which surround a red and scaly or pale central area. Herpes tonsurans maculosus occurs more commonly than the latter and is characterized by a maculopapular eruption which spreads centrifugally and which fades and desquamates at the center. The diagnosis is made certain by the discovery of the myces and the gonidei in the stubs of hair or in the epidermal scales.

Treatment.—Vigorous rubbings with alcohol, followed by the application of 5 per cent. naphtol ointment. Epilation may be necessary for herpes tonsurans capillitii.

FOLLICULITIS ABSCEDENS (ESCHERICH)

(*Pseudofurunculosis*)

The formation of multiple abscesses in the skin occurs not rarely in badly nourished, anemic, and atrophic children, in whom the skin shows an abnormally low tone, and also in overfed children. The abscesses are due to the invasion of pyogenic staphylococci into the sweat and sebaceous glands. They occur often in large numbers (twenty, fifty, or more), especially in the scalp, back, and lower extremities. At first we note lentil- to pea-sized fairly indolent papules underneath the slightly reddened skin. If the papules develop to the size of a hazel-nut the skin which covers them is of a livid red color, thin, and allows pus to shine through it. In about one week spontaneous rupture occurs and the pus escapes, after which rapid healing sets in. (The abscesses contain pus only, and at no time necrotic tissue.) Occasionally reso-

FIG. 146.—Skin parasites. *a*. Nits attached to the shafts of the hair. *b*. Pediculus capitis. *c*. Clothes-louse. *d*. Pediculus pubis. *e*. A scabies burrow. *f*. The egg of the Acarus scabiei. *g*. Itch-mite seen from below. *h*. Itch-mite seen from above. *i*. Trichophyton tonsurans (hair with its outer root-sheath, from a case of herpes tonsurans capitis). (Mracek, *Atlas of Skin Diseases.*)

lution occurs without suppuration. The repeated occurrence of fresh crops of the eruption may prolong the

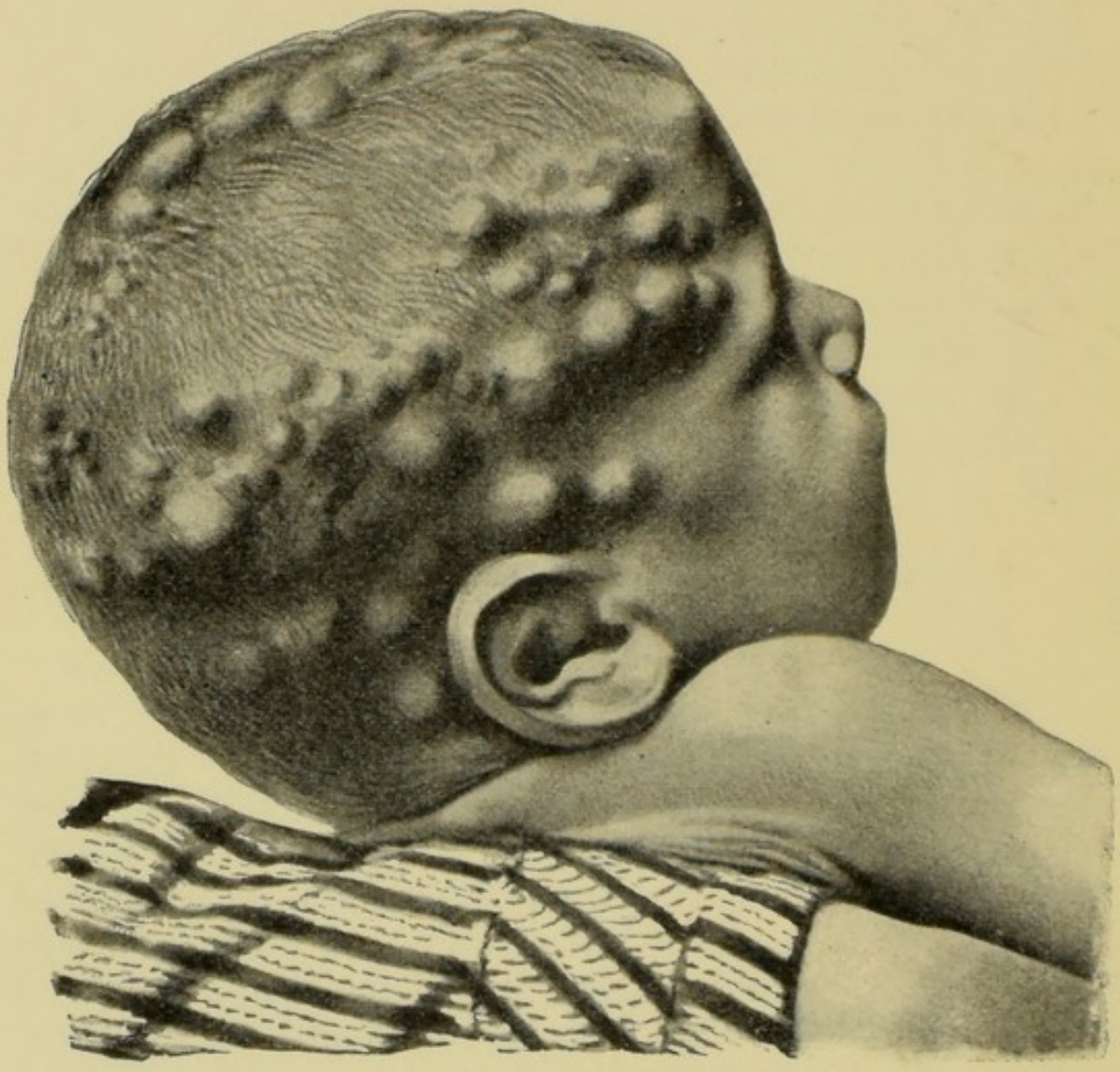


FIG. 147.—Pseudofurunculosis in an eight-months'-old poorly nourished infant who suffered from chronic disturbances of digestion.

disease considerably. The constitutional symptoms depend upon the causal disease.

Treatment.—Treat the etiologic factor. Improvement follows a reduction in diet in some cases. Many of the

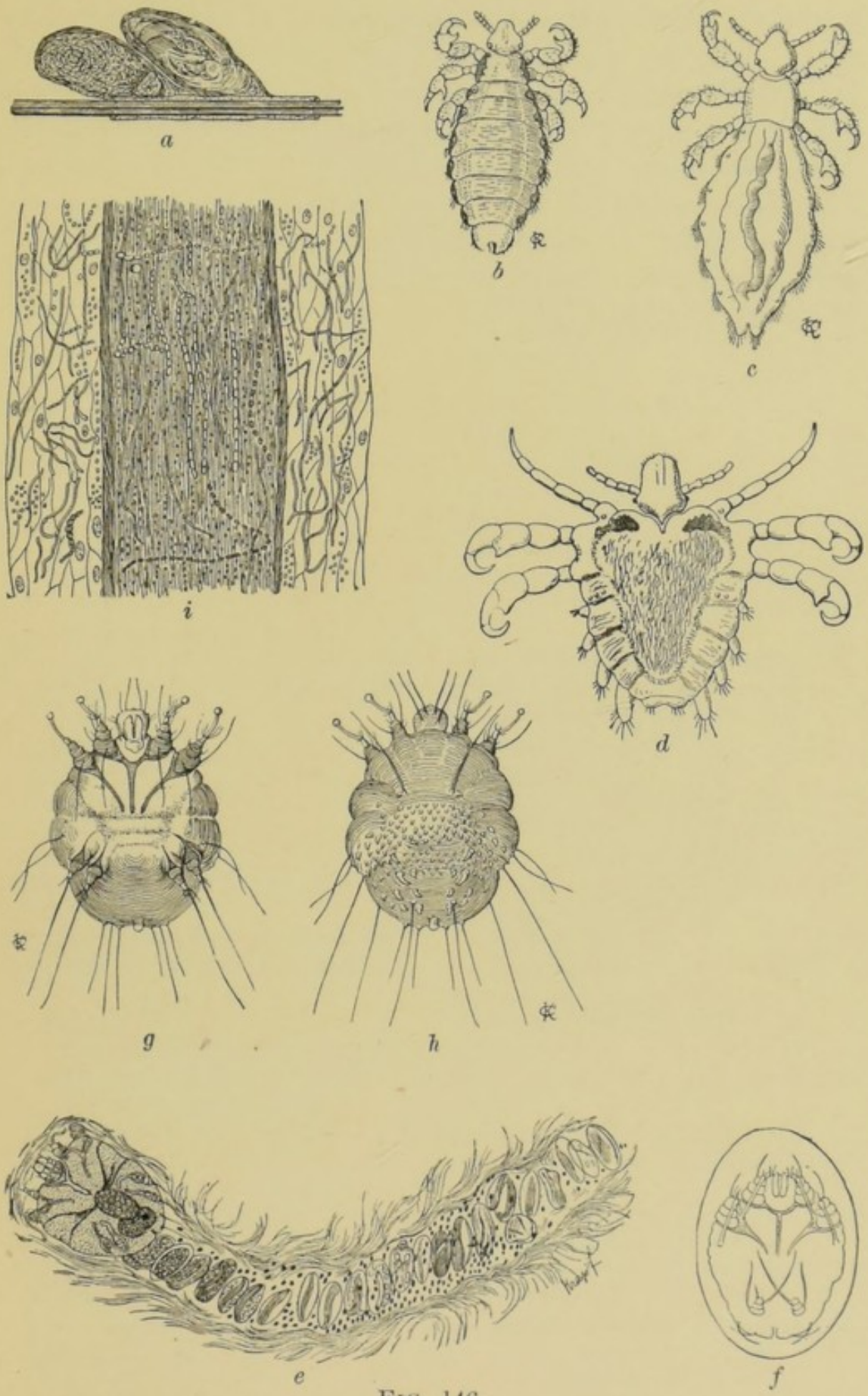


FIG. 146.

abscesses recede upon the application of aluminum acetate. The matured abscesses are pierced with a small point and carefully emptied, but not before the neighboring skin has been protected against the infection with the cocci-containing pus by anointing with a bland ointment. The abscesses which have been operated upon are covered with a moist dressing (aluminum acetate). Sublimate baths are recommended for extensive pseudofurunculosis (1 tablet to a bath).


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